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Management of Abdominal Aortic Aneurysms Clinical Practice Guidelines of the European Society for Vascular Surgery

F.L. Moll [a,*], J.T. Powell [b], G. Fraedrich [c], F. Verzini [d], S. Haulon [e], M. Waltham [f], J.A. van Herwaarden [a], P.J.E. Holt [g], J.W. van Keulen [a,h], B. Rantner [c], F.J.V. Schlösser [h], F. Setacci [i], J.-B. Ricco [j]

[a] *Department of Vascular Surgery, University Medical Center Utrecht, Utrecht, The Netherlands*
[b] *Imperial College, London, UK*
[c] *University Hospital Innsbruck, Austria*
[d] *Azienda Ospedaliera di Perugia, Italy*
[e] *Hôpital Cardiologique, CHRU de Lille, Lille, France*
[f] *St Thomas' Hospital, London, UK*
[g] *St George's Vascular Institute, London, UK*
[h] *Yale University - School of Medicine, New Haven, Connecticut, USA*
[i] *University of Siena, Siena, Italy*
[j] *University of Poitiers, Poitiers, France*

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Introduction

Purpose of these guidelines

The European Society for Vascular Surgery (ESVS) appointed the AAA Guidelines Committee to write the current clinical practice guidelines document for surgeons and physicians who are involved in the care of patients with abdominal aortic aneurysms (AAAs). Guideline development was recommended in 1990 by the Institute of Medicine to improve decision making for specific patients' circumstances and to decrease the variability in appropriate and inappropriate

* Corresponding author. Tel.: +31 887556965; fax: +31 887555017.
E-mail address: f.moll@tip.nl (F.L. Moll).

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health care between providers.[1,2] Appropriate decision-making is critical to achieving excellent outcomes.

Abdominal aortic aneurysm disease is complex and has significant clinical practice variability, although a valid evidence base is available to guide recommendations. The significant increase in the quantity of scientific literature concerning abdominal aortic aneurysmal disease published in recent years along with the number of technical and medical advances enables guideline recommendations with more certainty and supporting evidence than before. Potential increases in health care costs and risks due to industry and public-driven use of novel treatment options make the current guidelines increasingly important.[3–6]

Many clinical situations of patients with AAAs have not been the subject of randomised clinical trials. Patient care, however, needs to be delivered and decisions have to be made in these situations. Therefore, this document also provides guidance for decisions when extensive level I evidence is not available and recommendations are determined on the basis of the currently available best evidence for these situations. By providing information about the relevance and validity of the quality of evidence, the reader will be able to locate the most important and evidence-based information relevant to the individual patient.[7] To optimise the implementation of the current document, the length of the guidelines has been kept as short as possible to enable prompt access to the guideline information. This clinical guidelines document is supposed to be a guide, not a document of rules, and allows flexibility for specific patients' circumstances.

This is the resulting clinical practice guidelines document and provides recommendations for clinical care of patients with abdominal aortic aneurysms including pre-operative, perioperative and post-operative care.

Methods

Patients with AAAs are defined as male or female patients with asymptomatic, symptomatic or ruptured AAA with fusiform dilatation. This document does not cover patients with a saccular, infected or mycotic AAA or pseudoaneurysmal aortic dilatation. The AAA Guidelines Committee met in September 2009 for the first time to discuss the purpose and methods. The AAA Guidelines Committee has been constituted with incorporation of members from different European countries, from academic and private hospitals, vascular and endovascular specialists and patients to maximise the support for the final guidelines document. Since Europe encompasses a variety of health care systems and political economies, health policy makers were not included.[8]

The AAA Guidelines Committee performed a systematic literature search in MEDLINE, EMBASE and COCHRANE Library databases for each of the different topics that are discussed in this guidelines document. The Guidelines Committee used a grading schema based on levels of evidence and grades of recommendation according to the levels of evidence from the Oxford Centre For Evidence-Based Medicine.[9]

The level of evidence classification provides information about the study characteristics supporting the recommendation and expert consensus, according to the categories shown in Table 1.

The recommendation grade indicates the strength of a recommendation. Definitions of the grades of recommendation are shown in Table 2.

The AAA Guidelines Committee aimed to report as much as possible the calculated estimates of effects with their 95% confidence intervals. Every part of the guidelines document has been prepared by at least two members of the Committee and has been reviewed by the entire Committee. The initial guidelines document has been subsequently reviewed by the AAA Guidelines Review Committee. After incorporation of all comments and recommendations, the guidelines have been provided to the members of the ESVS. The final document has been approved by the ESVS.

Chapter 1 — Epidemiology

Definition of abdominal aortic aneurysms

Abdominal aortic aneurysm (AAA), which comes from the Ancient Greek word ἀνεύρυσμα, means a dilatation or widening of the abdominal aorta. The most accepted definition of an AAA is based on the diameter of the abdominal aorta: an abdominal aortic diameter of 3.0 cm or more, which usually is more than 2 standard deviations above the mean diameter for both men and women, and is considered to be aneurysmal.[10–12] Other researchers have suggested defining abdominal aortic aneurysm as the maximum infra-renal aortic diameter being at least 1.5 times larger than the expected normal infra-renal aortic diameter to compensate for individual variation in the diameter of the adjacent aorta.[13–15]

AAA can be defined as an abdominal aortic diameter of 3.0 cm or more in either anterior-posterior or transverse planes. Level 2c, Grade B.

Epidemiology

Prevalence and risk factors

Population screening studies offer the best evidence regarding the prevalence of AAA. Several of these have been conducted as randomised trials to assess the benefits of screening (MASS, Western Australia, Viborg and Chichester, the latter being the only one to include women).[16–19] Other evidence comes from the Rotterdam, Tromsø and other large epidemiological screening studies.[20,21] Prevalence rates vary according to age, gender and geographical location (Table 3). Level 1a.

In keeping with ethnic and environmental risk factors, a screening study of US veterans (between 50 and 79 years old, $n = 73{,}451$) showed the highest prevalence of AAA $\geq$3.0 cm was 5.9% and was found in white male smokers between 50 and 79 years.[22] All the aneurysm population screening data (Table 3) are now dated and there is little contemporary information for 21st century prevalence, although there are some indications, at least in the USA, that the admission rate for aneurysm repair is declining.[23]

Important risk factors for AAA are advanced age, male gender and smoking.[20–31] A positive family history for AAA especially in male first-degree relatives, is also associated

Table 1 Level of evidence classification.

Level	Therapy/Prevention, Aetiology/Harm	Prognosis	Diagnosis
1a	SR (with homogeneity) of RCTs	SR (with homogeneity) of inception cohort studies; CDR validated in different populations	SR (with homogeneity) of Level 1 diagnostic studies; CDR with 1b studies from different clinical centres
1b	Individual RCT (with narrow Confidence Interval)	Individual inception cohort study with > 80% follow-up; CDR validated in a single population	Validating cohort study with good reference standards; or CDR tested within one clinical centre
1c	All or none	All or none case-series	Absolute SpPins and SnNouts
2a	SR (with homogeneity) of cohort studies	SR (with homogeneity) of either retrospective cohort studies or untreated control groups in RCTs	SR (with homogeneity) of Level >2 diagnostic studies
2b	Individual cohort study (including low quality RCT; e.g., <80% follow-up)	Retrospective cohort study or follow-up of untreated control patients in an RCT; Derivation of CDR or validated on split-sample only	Exploratory cohort study with good reference standards; CDR after derivation, or validated only on split-sample or databases
2c	"Outcomes" Research; Ecological studies	"Outcomes" Research	
3a	SR (with homogeneity) of case-control studies		SR (with homogeneity) of 3b and better studies
3b	Individual Case-Control Study		Non-consecutive study; or without consistently applied reference standards
4	Case-series (and poor quality cohort and case-control studies)	Case-series (and poor quality prognostic cohort studies)	Case-control study, poor or non-independent reference standard
5	Expert opinion without explicit critical appraisal, or based on physiology, bench research or "first principles"	Expert opinion without explicit critical appraisal, or based on physiology, bench research or "first principles"	Expert opinion without explicit critical appraisal, or based on physiology, bench research or "first principles"

SR, systematic review; RCT, randomised controlled trial; CDR, clinical decision rule; SpPin, Specificity is so high that a Positive result rules-in the diagnosis; SnNout, Sensitivity is so high that a Negative result rules-out the diagnosis.

with increased risk for AAA.[29–31] Smoking is a strong risk factor (odds ratio >3.0 in all studies), the associated risk being much higher than for either coronary artery disease or stroke.[20–22,24,28] Level 2a.

Additionally, the following factors have been associated with AAA development: history of other vascular aneurysms,[32–35] greater height,[22] coronary artery disease,[22,33] cerebrovascular disease,[34] atherosclerosis,[22] hypercholesterolemia,[20,22] and hypertension,[21,22,35,36] although the data for some of these factors are inconsistent and studies may not have been subject to multivariate adjustment, so that spurious associations may have been reported. More recently, genome-wide association studies have demonstrated the association with variants on chromosome 9p21. The presence of rs7025486[A] in the DAB21P gene is associated with a 20% increased risk of developing AAA, odds ratio 1.21 [95%CI 1.14–1.28].[37] Black or Asian race and diabetes mellitus are negatively associated with AAA development.[22,38] Level 2a-3b.

The evidence for other risk factors including homocysteinemia, high levels of lipoprotein (a) and plasminogen activator inhibitor-1 is very weak.[39] Level 4b.

Table 2 Grades of recommendation

A	Consistent level 1 studies
B	Consistent level 2 or 3 studies *or* extrapolations from Level 1 studies
C	Level 4 studies *or* extrapolations from level 2 or 3 studies
D	Level 5 evidence *or* troublingly inconsistent or inconclusive studies of any level

"Extrapolations" are where data are used in a situation that has potentially clinically important differences than the original study situation.

Natural history

AAA growth rates

The reported average growth rate of AAAs between 30 and 55 mm ranges from 0.2 to 0.3 cm per year. Larger AAA

Table 3 The prevalence of AAA detected by population screening.

Study location	Chichester, UK[16]	Viborg, Denmark[17]	Western Australia[18]	MASS UK[19]	Rotterdam, Netherlands[20]	Tromsø, Norway[21]
n	15,775	12,628	41,000	67,800	5419	6386
Gender	Men & women	Men	Men	Men	Men & women	Men & women
Age (years)	65–80	65–73	65–79	65–74	>55	55–74
Sampling dates	1988–90	1994–8	1996–8	1997–9	1994–5	1994–5
Date published	1995	2002	2004	2002	1995	2001
Aneurysm prevalence	4.0% (7.6% in men, 1.3% in women)	4.0%	7.2%	4.9%	4.1% men, 0.7% women	8.9% men, 2.2% women

diameters are associated with higher AAA growth rates. A wide variation between patients has been reported consistently.[40–49] Level 1b-2b.

Several cohort studies have implicated that statins are associated with lower AAA growth rates.[42,50,51] However, the largest and most carefully conducted study has not demonstrated any association between statins and AAA growth.[52] Smoking has been associated with aneurysm expansion.[40,46,47,53–57] Smoking cessation may be recommended to reduce the risk of AAA growth. Level 2b, Grade B.

Data on the predictive value of hypertension,[42,55,58–60] age,[41,42,47,54,59,61] gender,[41–43,61] betablocker usage,[46,49,62–68] and diabetes mellitus[41,42,54,55] are inconsistent. However the majority of studies report a negative association between diabetes and aneurysm growth. Level 2b.

Factors that are consistently not associated with AAA growth across several studies include chronic obstructive pulmonary disease,[43,54,69] lipids,[42,55,60] and body weight.[42,47,56,59] Other less studied factors include alcohol abuse, genetics, *Chlamydia pneumoniae*, usage of some drugs apart from statins (including NSAIDs, angiotensin-converting enzyme inhibitors, angiotensin II receptor blockers, doxycycline, roxithromycin, steroids, chemotherapeutic drugs), ankle-brachial index, past medical history of peripheral vascular disease, cardiac disease and other cardiovascular diseases, organ transplantation, body length, several laboratory values, the extent of thrombus in the aneurysm sac and physical activity.[40,51,70–76]

AAA rupture

Larger initial aneurysm diameter is a significant and independent risk factor for AAA rupture.[77–85] Level 2a.

The association between AAA diameter and 12-month AAA rupture risk is depicted Table 4.

Table 4 12-month AAA rupture risk by diameter.[8,82–84]

AAA Diameter	Rupture Risk (%)
30–39	0
40–49	1
50–59	1.0–11
60–69	10–22
>70	30–33

AAA, abdominal aortic aneurysm.

Other factors that have been associated with an increased risk of AAA rupture across several studies include: female gender,[78–81] smoking,[76] hypertension,[78–80] AAA expansion rate[39,79,85–88] and peak AAA wall stress.[89–93] Level 2b-3b.

Individual studies have suggested an increased risk of AAA rupture for patients with rapid increase of intraluminal thrombus,[94] increased AAA wall stiffness,[95] increased AAA wall tension,[96] a low forced expiratory volume in 1 second (FEV_1) and for transplant patients.[71] The use of novel imaging and analysis technologies to estimate both wall stress and areas of increased metabolic activity is an area of current interest although no strong evidence has accumulated to date and no clinical recommendations can be made. Level 4, Grade D.

Chapter 2 – Screening

These guidelines refer to screening using ultrasonography for infra-renal AAA. Ultrasonography is used for screening because it is non-invasive, cheap, can be performed in a community setting and has a high sensitivity and specificity for the detection of AAA.[97,98] The technological advances in instrumentation have led to small, portable ultrasound machines with facilities for storing images. The ultrasound probe can be angled to ensure that measurements are made perpendicular to the longitudinal axis of the aorta. AAA is defined usually as an external aortic diameter ≥3 cm, although an internal aortic diameter of ≥3 cm has been used in some circumstances (see below).

The advantages and limitations of ultrasonography as a screening method

In addition to the advantages of ultrasonography, listed above, it is possible to train any health care worker to perform aortic diameter measurement using ultrasonography and this avoids the necessity of using trained ultrasonographers for population screening. Both the sensitivity and specificity of ultrasonography have been reported as being close to 100%.[97,98] There is evidence to support the used of anterior–posterior rather than transverse measurements, since the latter has worse repeatability.[99] Both the external diameter and the internal diameter may be measured. The evidence for upper threshold for AAA surveillance (5.5 cm diameter) was based on the measurement of external aortic diameter[100] (see Chapter

3: Decision-making). In contrast, the MASS trial, the largest of the population-based aneurysm screening trials, was based on the measurement of internal aortic diameter[101] (http://aaa.screening.nhs.uk/Implementation_Guidance). The Viborg aneurysm screening trial[17] and most other screening programmes have reported using external aortic diameter. Since internal diameters are 2–5 mm smaller than external diameters, there are two important issues to be resolved.

Are the threshold aortic diameters of 3 cm and 5.5 cm based on the internal aortic diameter safe? The MASS trial reports an increase of aneurysm ruptures in screened patients after 8 years of follow-up,[102] so were the smallest aneurysms overlooked?

Which diameter, internal or external, is most reproducibly measured in community screening programmes? This is important since, at best, the reproducibility of measurement of external aortic diameters is ±2 mm.[98–100]

The evidence in favour of population screening for AAA in men

The four randomised trials of population screening are the Chichester trial in the UK,[16] the Viborg trial in Denmark,[17] the Western Australia trial[18] and the MASS trial in the UK.[19] In each trial, populations were randomised to either an offer of aneurysm screening or to no offer of screening, and in each trial screening, was shown to reduce aneurysm-related mortality for men. These results, to 5 years, have been summarised in a Cochrane Review[103] and the odds ratio in favour of screening for men was 0.60 [95%CI 0.47–0.78]. A systematic review for the US Preventive Task Force reported a similar benefit for screening men, odds ratio 0.53 [95%CI 0.42–0.68].[104] The individual characteristics of the trials are summarised in Table 5. This table also serves to illuminate some of the differences between the trials. In the Western Australia trial randomisation occurred several months ahead of the invitation for screening being issued, so that about 2296 men had died before their invitations were issued; the uptake of screening was 63% if estimated from the time of randomisation and 70% if estimated from the time of invitation. There also is one broad similarity between the trials, not listed in Table 3, in that all trials were conducted in relatively advanced socioeconomic areas where a semi-rural hinterland is dotted with medium or small size towns inhabited predominantly by persons of Caucasian origin. None of the screening trials were conducted, except small part, in very deprived large city districts.

The longer-term follow-up of subjects in the MASS trial has provided additional results. After 7 years of follow-up the MASS trial reported an all-cause mortality benefit in favour of screening at the limits of statistical significance, hazard ratio 0.96 [95%CI 0.93–1.00];[105] no all-cause mortality benefit was observed in the Western Australia trial after 5 years of follow-up. Very recently the MASS trial published 10-year results.[101] These showed that aneurysm-related deaths were halved in the group invited for screening at a cost of £100 for every man screened, although there is a suggestion from a report from the USA that costs might be less than this.[106] Overall there were 552 elective aneurysm repairs in the screened group (with an operative mortality of 4%) versus 226 in the control group (with an operative mortality of 6%). However, after 8 years there was a noticeable increase in ruptures in the screened group. Although studies have reported that a single screen at age 65 years is sufficient, this may require re-evaluation, particularly as the population lives longer.[107]

Population screening of older men for AAA, in regions where the population prevalence is 4% or more, reduces aneurysm-related mortality by almost half within 4 years of screening, principally by reducing the incidence of aneurysm rupture. Level 1a, Recommendation A.

The evidence for screening in women

The population prevalence of AAA is three times higher in men than in women. Therefore, not surprisingly, there is no good evidence to support aneurysm screening in older women. The only screening trial conducted in women was in Chichester, UK,[108] and is reported as part of the Chichester trial in Table 3. There was no reduction in the incidence of aneurysm rupture after either 5 or 10 years of follow-up. Given the previous low prevalence of aneurysms detected in women, this trial may not have had sufficient power to detect any benefit from screening. However smoking, the principal risk factor for AAA, has been increasing in women and the future incidence of AAA in female smokers is unknown.

Population screening of older women for AAA does not reduce the incidence of aneurysm rupture. Level 1b, Recommendation B.

Population screening of older female smokers for AAA may require further investigation. Level 3c, Recommendation B.

Screening in other subgroups

Consideration has been given to the merits of screening by different subgroups, including those relating to smoking, ethnicity, other cardiovascular disease and those having or having had relatives with AAA.

The US Preventive Services Task Force has recommended aneurysm screening for men aged 65–75 years who have ever smoked, based on the strength of the association between smoking and AAA.[109] There is no good evidence to support this proposal, although it seems reasonable.

Ever-smoking increases the risk of developing AAA 4- to 5-fold. Screening only smokers might improve the cost-effectiveness of aneurysm screening. Level 5, Recommendation D.

The Society of Vascular Surgery recommends screening men aged 65 years with a family history of AAA.[110] This is based on reports from several countries of an increased incidence of AAA amongst first-degree relatives of AAA patients. The best data for this comes from a Swedish population study, when a family history of AAA increased the risk of AAA, odds ratio 1.9 [95%CI 1.6–2.2].[31] The benefits of screening for AAA in the presence of a family history of aneurysm has not been assessed formally.

A family history of AAA increases the risk of AAA about 2-fold. Screening of older men and women having a family history of AAA might be recommended. Level 3a, Recommendation C.

Table 5 Summary of the population-based randomised screening trials.

Trial characteristics	Chichester, UK[16]	Viborg, Denmark[17]	MASS UK[101,c]	Western Australia[18]
Number randomised	15,775	12,628	67,800	41,000
Gender	Men & women	Men	Men	Men
Age (years)	65–80	65–73	65–74	65–79
Dates recruited	1988–90	1994–8	1997–9	1996–8
Date published	1995	2002	2002	2004
% accepting screening	68%	76%	80%	70%[d]
Aneurysms found	4% (7.6% in men)	4%	4.9%	7.2%
Place of screening	Hospital	Hospital	Community	Community
Intervention policy	At 6 cm	At 5 cm	At 5.5 cm measured as internal diameter	None
Mean follow-up (months)	30.5	61	49	43
AAA mortality odds ratio screened vs not (95%CI)[a]	0.59 men only (0.27–1.29)	0.31 (0.13–0.79)	0.58 (0.42–0.78)	0.72 (0.39–1.32)
All-cause mortality odds ratio Screened vs not (95%CI)[b]	Men only 1.07 (0.93–1.22)		0.97 (0.93–1.02)	0.98 (0.91–1.04)
Other outcomes reported	No aneurysm-related mortality benefits in women	Hospital deaths Costs Quality of life	Quality of life Costs Workload	
Extended follow-up available	Yes		Yes	

[a] Pooled odds ratio overall 4 trials strongly in favour of screening, OR 0.57 (0.45–0.74), together with a halving of the incidence of aneurysm rupture in screened populations.
[b] Pooled odds ratio trend in favour of screening, OR 0.98 (0.95–1.02).
[c] The MASS trial recently has published 10-year follow-up, demonstrating the cost-effectiveness of screening and a significant all-cause mortality benefit but a rising incidence of AAA rupture in the screened group.
[d] As percentage of those alive when invitation for screening was sent: randomisation predated this invitation by several months in a large sector of subjects.

Screening those with a known family history of AAA should be evaluated and include both men and women above 50 years of age.

Two studies, both from the UK, have reported a very low incidence of aneurysms in subjects of Asian ethnic origin.[38,111] In particular in the Leicester screening programme among men aged 65 years of Asian origin the prevalence of AAA (0.45%) was significantly lower than among the Caucasian population (4.69%). Screening Asian men for AAA may not be cost-effective. Level 2b, Recommendation B.

There is no good evidence about the prevalence of AAA among other ethnic groups represented in Europe or elsewhere.

There is evidence to suggest that the incidence of AAA is high (7–10%) among those with other forms of peripheral arterial disease.[112,113]

Opportunistic screening of patients with peripheral arterial disease should be considered. Level 2a, Recommendation B.

There is some evidence to suggest that screening of patients with hypertension is not very productive.

Can screening cause harm?

There are three potential harms that may be caused by screening.

First there is the anxiety and subsequent effects on quality of life associated with being told that you have something, potentially fatal, wrong with you. Both the MASS and Viborg trials report that subjects found to have an aneurysm on screening experienced anxiety and a decreased quality of life for a short period after screening. Such effects were most marked in those with poor quality of life at baseline but the effects resolved within a few months of screening.[101,114]

Second, and perhaps more importantly, there is the mortality risk associated with intervention. If screening is to be conducted safely, the vascular surgical referral centres for patients must have an audited low mortality for both open and endovascular aneurysm repair (EVAR):[115] for elective open repair the operative mortality must be less than 5% (as practised in the Chichester, Viborg and MASS trials), and for EVAR less than 2%. The early advantage of EVAR, together with its increasing usage, is unlikely to result in a greater survival advantage of population screening because there is a "catch-up" in mortality after EVAR, so that after 2–3 years the overall mortality after open and endovascular repair is closely similar.[116–119] Recent work clearly shows that most patients have a preference for aneurysm repair by EVAR rather than by open repair. The recent results showing the risk of late endograft rupture (0.7% per 100 person-years) were unknown at the time of patient preference studies and may dampen some of the preferences for EVAR.[119] However some patients still prefer open repair since it avoids the need for long-term post-repair surveillance.[120,121] However some patients will not be anatomically suitable for routine endografting. Therefore, to allow for both patient preferences and diverse patient anatomy there is a continuing need for

Table 6 Surveillance frequency of screen-detected aneurysms.

UKSAT modelling study[122]	Surveillance interval (months)	Chichester[16]	Viborg[17]	MASS[101]	Western Australia[18]
3.0–3.9 cm	24	Annual scans	Annual scans	Annual scans	No surveillance policy
4.0–4.5 cm	12	3.0–4.4 cm	3.0–5.0 cm	3.0–4.4 cm	
4.5–5.0	6	then 3 monthly		then 3 monthly	
>5.0	3	scans to 6.0 cm		scans to 5.5 cm	

centres to provide elective AAA repair using both open surgery and EVAR with low mortality.

Third, screening may cause an unacceptable burden on local vascular surgical services. The MASS and other trials have shown that the rate of elective repairs doubles with the advent of screening, although the burden of out-of-hours ruptures is reduced.[101–103]

Screen detection of an AAA causes a small but temporary reduction in quality of life. Aneurysm screening should only be conducted if the audited mortality from aneurysm repair at the referral hospital is low. Level 2a, Recommendation B.

Referral hospital facilities to cope with an increased number of elective AAA repairs, both open and endovascular, must be in place before aneurysm screening starts. Level 5, Recommendation D.

Referral hospitals should offer both open and endovascular repair. Level 2c, Recommendation B.

Potential health benefits associated with screening

Detection of an aneurysm should be accompanied by referral for cardiovascular risk assessment and lifestyle advice. The benefits of stopping smoking, good control of blood pressure and other relevant lifestyle and therapeutic changes, including statins, are discussed in Chapter 3 below.

An effective treatment to reduce or stop the growth of small AAA has not yet been identified clearly. Systematic review of the evidence to hand suggests that statins may reduce aneurysm growth rates by about 50%, although a large recent study found no such benefit associated with statin therapy.[122,123] Smoking cessation appears to reduce growth rate by 20–30%.[41]

All subjects with a screen-detected aneurysm should be referred for cardiovascular risk assessment with concomitant advice and treatment, including statins and smoking cessation therapy. Level 2c, Recommendation B.

The management of patients with screen-detected aneurysm

The management of patients with AAA detected on screening depends principally on the aneurysm diameter and these issues are discussed in Chapter 3. Most people with aneurysms in the diameter range 3–5.5 cm will be kept under review in surveillance programmes.

The frequency of resurveillance for those with small aneurysms

A modelling exercise using data from the UK Small Aneurysm Trial and Study has been the most scientific approach to date of optimal resurveillance intervals.[41] These intervals are compared with the intervals used in the screening trials in Table 6. There is consensus that the rescreening interval is inversely related to the aneurysm diameter, but optimal rescreening intervals remain to be established. The National Institute of Health Research in the UK has commissioned such research, which is in progress.

Rescreening intervals should shorten as the aneurysm enlarges. Level 2a, Recommendation B.

Evidence to support safe, cost-effective rescreening intervals is awaited.

To prevent interval rupture, it is recommended that a vascular surgeon review patients within 2 weeks of the aneurysm reaching 5.5 cm or more in diameter. Level 5, Recommendation D.

Where should screening take place – hospital or local centre?

Screening can take place either in hospitals[16,101] or community care by visiting sonographers with portable ultrasound equipment,[18,100] or by a combination. The success of either model may depend on distribution of the screened population (urban or rural) and the presence of a suitable community network or general practitioners or community medical facilities. There are no studies directly comparing these approaches.

The screening model chosen should be flexible for the local population characteristics. Level 4, Recommendation D.

When to screen

Age is an important risk factor for AAA and all of the randomised trials screened at 65 years and older. This has been chosen as an age when the prevalence of AAA is high enough for there to be a benefit for screening whilst balancing risk of rupture at an earlier age against the cost of repeat screening when older. A significant number of ruptures occur in those younger than 65 years, although the proportion reported varies from 5 to 18%.[124,125] Data from national statistics could be used to determine the age of screening in individual countries.

No trial has assessed the optimum age at which there is greatest benefit in terms of lives saved and cost-benefit. In a simulation cohort model screening at 60 instead of 65 was equally cost-effective with the advantage of more life years gained.[126] There may be an argument for earlier screening and repeat screening for those at higher risk for aneurysm although in the model the benefit of treating higher risk groups was eliminated by their lower life expectancy.[127]

The incidence of new AAA after a single normal scan at 65 years is rare, and when present rarely reaches

a significant size, although the MASS trial has reported an increase in late rupture (after 8 years) in those with a normal screen at 65 years.[128] A negative result on a single scan at 65 years greatly reduces the risk of future AAA rupture.[107,124,129–131]

Men should be screened with a single scan at 65 years old. Level 1a, Recommendation A.

Screening should be considered at an earlier age for those at higher risk for AAA. Level 4, Recommendation C.

Repeat screening should be considered only in those initially screened at a younger age or at higher risk for AAA. Level 2b, Recommendation C.

When should patients be referred to a vascular surgeon?

Size, symptoms and growth rates.

The size criteria for referral for patients have been set between 5.5 cm and 6.0 cm diameter. These were based on earlier evidence that suggested that the annual rupture rate in patients with aneurysm 6.0 cm in diameter was lower than the mortality rate for elective surgery in most centres.[125,132] The safety of surveillance for aneurysms less than 5.5 cm has since been confirmed in trials.[100,133] Data from MASS trial suggests that size alone is the best indicator of risk with symptoms and rapid expansion being poor indicators.[134]

Men should be considered for surgery when the maximum aortic diameter reaches 5.5 cm or more. Level 1b, Recommendation A.

Increased risk groups.

Female gender, smoking, hypertension and chronic airway disease are associated with an increased risk of small aneurysm rupture in some studies.[78,135,136] Women have a 3- to 4- fold increased risk of rupture when under surveillance[100] and average aortic size at rupture is 5 mm smaller in women than men,[137] although operative outcomes tend to be worse for women than men.[138]

Patients with a higher risk of rupture should be considered for surgery when the maximum aortic diameter reaches 5.0 cm. Level 3, Recommendation C.

How to optimise uptake of screening?

Optimising uptake will reduce average cost per person of screening, although when modelled the attendance rate had little effect on the cost-effectiveness ratio.[129] Factors that may affect attendance include public awareness, the demographics of the screened population, the location from where invitations are sent, the use of written and telephone invitations, the site of screening, scheduling of appointment times, removal of financial barriers to attend and re-invitation strategies for non-attendees.[139] It is possible that invitations to screening coming from the family or general practitioner will be received more favourably than those coming from a hospital or screening programme. However, there are no studies evaluating the effectiveness of these or other factors in AAA screening programmes.

Screening programmes should be tailored to the local population to maximise attendance. Invitation to screening from the general or family practitioner might be received favourably. Level 4, Recommendation D.

Patients reviewing these guidelines felt strongly that uptake would be optimised by a better advertising campaign for screening, general practitioner invitations and community screening.

Screening programmes should be well advertised. Level 4, Recommendation B.

Problems with ultrasonography

Ultrasound has high sensitivity and specificity if performed with adequate quality assurance and false positives or negatives must be minimised to ensure a benefit of screening. Ultrasound can reliably image the aorta in 99% of subjects,[98] but difficulty visualising the aorta may occur in some cases and this must be recognised (1.2% in the MASS trial).[101] The subject should be rescanned in a hospital setting by an experienced sonographer.

The incidence of false-positive scans is uncertain but is small and of little clinical consequence as they are likely to be detected on surveillance rescanning or confirmatory CT.

If screening programmes use relatively inexperienced screening staff and portable ultrasound devices, programmes should be audited for quality control. Level 5, Recommendation D.

Detection of incidental pathology

The incidence of incidental discovery of other pathologies in screening programmes for AAA appears to be low (none reported in MASS). In the MASS study iliac aneurysms were referred if over 3 cm[140] but there are no reported data on the incidence detected.

Incidental pathology should be referred to the family practitioner. Level 5, Recommendation D.

Summary

Although the evidence that screening programmes reduce the incidence of aneurysm rupture and are likely to be cost-effective is very strong, there are still many practical aspects relating to screening programmes which require better evidence. These include techniques to optimise the uptake of screening, whether internal or external diameter should be measured, cost-effective surveillance intervals, and the management of patients with small aneurysms to reduce anxiety and cardiovascular risk. Merely mimicking the practice of the successful screening trials is not enough and there is an urgent need for further evidence around the practicalities of screening programmes.

Chapter 3 — Decision-making for Elective AAA Repair

These guidelines refer to the management of elective infra-renal AAA only— for cases that are amenable to treatment by a standard, commercially available endograft, or by open repair utilising an infra-renal aortic clamp placement. Cases that will require the use of branched/fenestrated endografts, a suprarenal aortic clamp, suprarenal aneurysms and thoraco-abdominal aneurysms should be referred to units specialising in the treatment of these more complex, higher-risk cases.

The threshold for repair of asymptomatic AAA

The management of AAA depends on the size or diameter of the aneurysm and is a balance between the risk of aneurysm rupture and the operative mortality for aneurysm repair (Fig.1 and Fig. 2). A commonly used definition of AAA is when the maximum aortic diameter is ≥3.0 cm.

There is consensus that for very small aneurysms, 3.0—3.9 cm, the risk of rupture is negligible. Therefore, these aneurysms do not require surgical intervention and should be kept under ultrasound surveillance at regular intervals (see Chapter 2 Screening).

The management of aneurysms 4.0—5.5 cm in diameter has been effectively determined by two large multi-centred randomised controlled trials of early open elective surgery versus surveillance, the UK Small Aneurysm Trial (UKSAT) and the American Aneurysm Detection And Management study (ADAM)[100,133] and a smaller trial of endovascular repair versus surveillance (CAESAR).[141] Another trial of early endovascular repair versus surveillance, PIVOTAL, focused only on the 4.0—5.0 cm diameter range.[142] All the trials had clearly defined intervention policies for the surveillance groups in addition to reaching the threshold diameter: these included rapid growth (>1 cm/year and the development of symptoms referable to the aneurysm). Neither trial of endovascular repair versus surveillance enrolled many women.

In the UKSAT, 1090 men and women, 60—76 years old, with asymptomatic small aneurysms (4.0—5.5 cm in diameter) were randomised either to early open surgery or to an aneurysm surveillance protocol. Mid-term results reported at the end of the trial showed no significant difference in all-cause mortality at 5 years between the two groups, and results were similar after 12 years of follow-up.[100,143] The aneurysm rupture rate was 1% per year in the surveillance group and the elective mortality rate for open surgery in the immediate repair cohort was 5.6%. Most patients in the surveillance group eventually underwent surgery because of aneurysm enlargement. Cost-effectiveness analyses suggested that surveillance was less costly than early surgery.[137]

The ADAM study recruited 1136 patients, nearly all male, with small aneurysms from Veterans' Affairs hospitals in the USA who were aged between 50 and 79 years old and were considered to be fit for open AAA repair. In this population, both the rupture rate in the surveillance group (0.6% per year) and the perioperative mortality rate in the surgery group (2.7%) were lower than in the UKSAT. As with UKSAT, the majority (60%) of the surveillance group underwent operative AAA repair by the end of the study period because of aneurysm enlargement. The findings of these two trials, summarised in a recent Cochrane review (at 6 years HR 1.11 [95%CI 0.91—1.34]), show the safety and hence benefits of a policy of surveillance for aneurysms 4.0—5.5 cm in diameter.[144]

Early surgery with EVAR?

In the UKSAT, the elective operative mortality rate was 5.6%, in ADAM 2.7%. At the time of these trials, opinion was

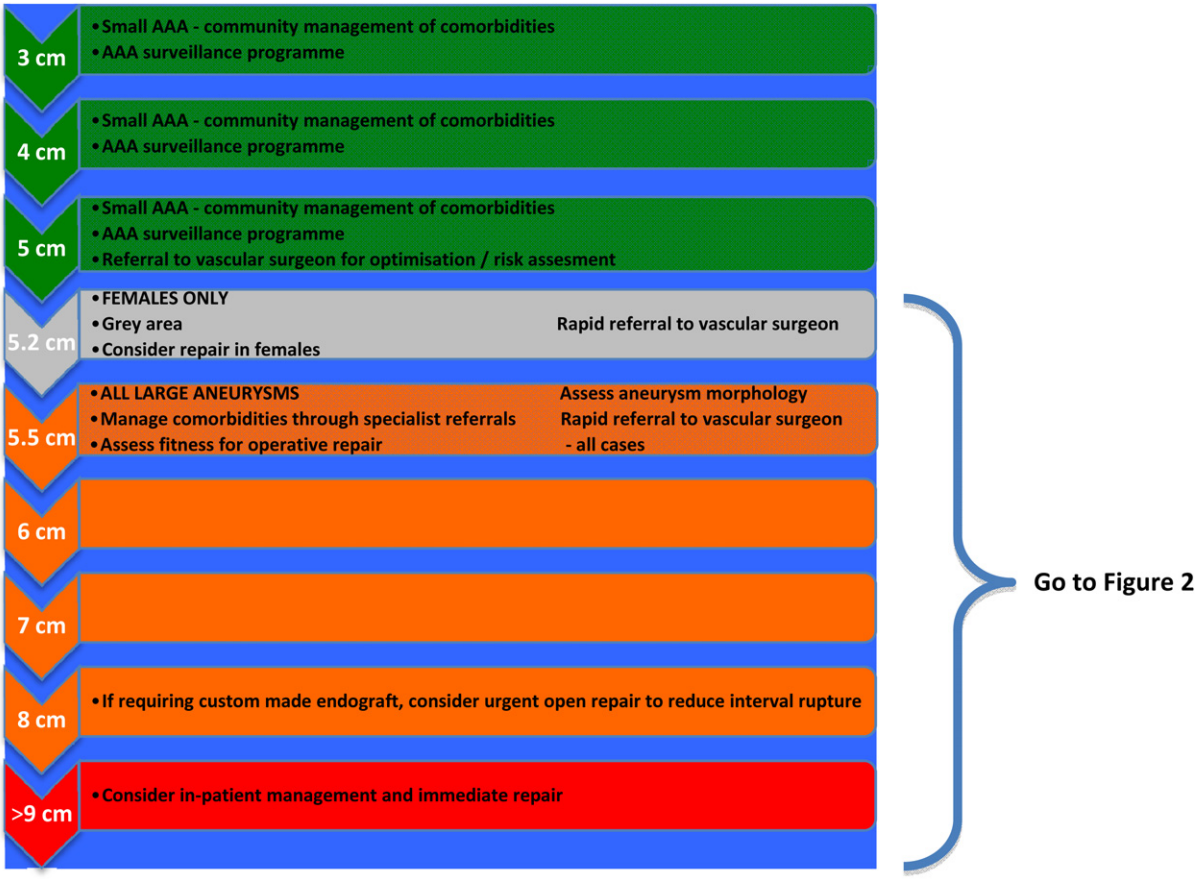

Figure 1 Management of AAA depending on size of aneurysm (continued in Figure 2).

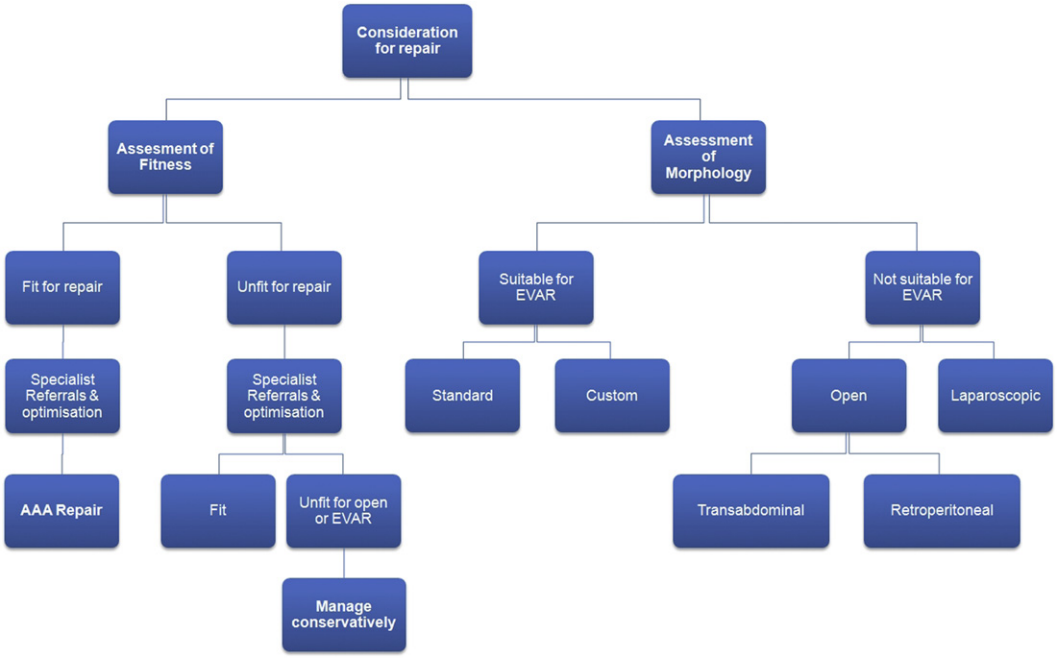

Figure 2 Management of AAA for large aneurysms (continued from Figure 1).

divided as to whether patients undergoing surgery in units with lower mortality would derive a greater long-term benefit from repair, potentially pushing the results in favour of early surgery. Subsequent analyses have demonstrated population-based perioperative mortality rates higher even than those reported in the trials and this supports a policy of small aneurysm surveillance in the general population.[145–147]

The advent of endovascular aneurysm repair (EVAR), associated with an elective mortality rate of approximately one-third that of open repair (1–2%)[117,118,147–149] has enlivened debate about the relevance of these historical results in modern surgical practice. Would early endovascular intervention be associated with improved longer-term survival when compared with a surveillance group?

Two multicentre randomised controlled trials of early EVAR versus surveillance for small aneurysms have been conducted.[141,143] These trials again have shown the very low rupture rate of small aneurysms with the early EVAR groups showing no mortality benefits at 3 years of follow-up, although the PIVOTAL trial used time to aneurysm rupture or aneurysm-related mortality as the primary endpoint, rather than all-cause mortality, as well as focussing only on AAA of 4.0–5.0 cm. PIVOTAL reported the primary endpoint as an unadjusted hazard ratio of 0.99 [0.14–7.06; $p = 0.99$].[150] There was no difference in overall mortality with a hazard ratio of 1.01 [0.49–2.07; $p = 0.98$]. In CAESAR, three years after randomisation, survival was similar in the two groups: 96.4% in the early EVAR arm vs 92.4% in the surveillance arm ($p = 0.6$). There were no significant differences in aneurysm-related mortality (0.6% vs 0.6%; $p = 1$), 30-day mortality (1% vs 0%; $p = 1$), aneurysm rupture (0% vs 0.2% $p = 0.2$) and secondary procedure rates (9.3% vs 5.3%; $p = 0.4$).

Therefore, these trials have not altered the current recommendations of surveillance as the preferred policy for aneurysms 4.0–5.5 cm in diameter. These findings also are supported by the Chichester screening trial, where surveillance of men to an aneurysm diameter of 6 cm was used safely and effectively and MASS where a 5.5 cm internal diameter was used.[16,101]

A policy of ultrasonographic surveillance of small aneurysms (4.0–5.5 cm) is safe and advised for asymptomatic aneurysms. Level 1a, Recommendation A.

When the threshold diameter (5.5 cm, measured by ultrasonography, in males) is reached or symptoms develop or rapid aneurysm growth is observed (>1 cm/year), immediate referral to a vascular surgeon is recommended. Level 3a, Recommendation B.

To prevent interval rupture, it is recommended that a vascular surgeon review patients within 2 weeks of the aneurysm reaching 5.5 cm or more in diameter. Level 5, Recommendation D. In some centres an earlier referral, at between 5.0 and 5.5 cm is an acceptable alternative practice.

There remains some uncertainty about the management of small aneurysms in defined subgroups (e.g. young patients, females, and those with limited life expectancy), see below.

Younger patients and women with AAAs

None of the randomised trials were powered to detect differences in all-cause mortality between subgroups by age or gender. No individual patient data meta-analyses have been conducted to detect these possible differences. The randomised trials have recruited very few women, the exception being UKSAT. Again, no individual patient data meta-analysis has been conducted. However females with small aneurysms are three or four times more likely to rupture whilst under surveillance than males, are less likely to be offered emergency treatment and have worse outcomes from subsequent interventions (AAA repair).[100,151] Furthermore, females appear more likely to

suffer AAA rupture at smaller aortic diameters than males. While there remains a paucity of data to definitively support earlier intervention in females, that which does exist would point towards a policy of surgery at a maximum aortic diameter, measured by ultrasonography, of closer to 5.2 cm, rather than the 5.5 cm threshold used for men.[152]

Females should be referred to vascular surgeons for assessment at a maximum aortic diameter of 5.0 cm as measured by ultrasonography.

Aneurysm repair should be considered at a maximum aneurysm diameter of 5.2 cm in females. Level 3b, Recommendation C.

Patients with limited life expectancy

The benefit of intervention in patients with limited life expectancy, or considered unfit for intervention remains uncertain. There is no early benefit (up to 3 years) of endovascular repair with respect to either aneurysm-related or all-cause mortality.[152] For the frail patient with life expectancy of more than 3 years, endovascular repair reduces aneurysm-related mortality and may attenuate all-cause mortality.[119,152]

Surveillance scan frequency

The optimum frequency for ultrasonographic surveillance scans of aneurysms 3.0—5.5 cm in diameter has not been determined by randomised trials and is discussed further in the chapter on screening and management of the patient with small screen-detected AAA. A few centres use CT scanning for surveillance and on average CT reports higher diameters than ultrasonography.

Concomitant aneurysms

Iliac, femoral and popliteal aneurysms may safely be monitored at 6-monthly intervals. Referral to a vascular surgeon to discuss intervention can be recommended at the following maximum diameters: Iliac 3 cm; femoral and popliteal 2.5 cm. It should be noted that 85% of patients with a femoral artery aneurysm, and 62% of those with a popliteal artery aneurysm, will have a concomitant AAA. These guidelines will not expand further on the evidence, or techniques, behind popliteal aneurysm repair.

Patients with an infra-renal AAA should have formal imaging through CT scanning of the iliac and common femoral arteries. Level 5, Recommendation D.

Medical optimisation of patients with AAA

AAA is a disease of the ageing population and often presents in patients with several comorbidities. Cardiac, respiratory and renal comorbidities all have a significant effect on the outcome of subsequent AAA repair.[153] Therefore, several pre-operative care strategies may improve early post-intervention morbidity and mortality.

Where patients have large aneurysms, medical optimisation should be initiated by vascular surgeons who must develop robust referrals pathways with other hospital specialists. For small aneurysms, there is more time to introduce beneficial lifestyle modifications and treatment options. Community health services must be made aware of the necessity for this and referrals made to specialists from within the community. All patients with a diagnosed aneurysm should be medically managed to best current evidence. As the evidence for therapeutic interventions in medical optimisation is continually evolving, specialists managing patients with AAA must remain conversant with the current evidence in the field.

Several interventions have been tested in randomised trials of surgical patients, often those undergoing open vascular surgical procedures, but none are uniquely based on aneurysm patients.

Optimising respiratory function

Smoking cessation

Smoking cessation can provide for short-term improvements by reducing lung secretions and lung function can be improved by physiotherapy or exercise programmes.[154] Intensive smoking cessation therapy introduced 4—6 weeks before surgery can reduce post-operative cardiac complications and length of hospital stay.[155—157]

Longer-term, chronic respiratory disease has been shown to be associated with increased aneurysm expansion rates and higher rates of AAA rupture.[158] The forced expiratory volume in 1 second in particular is correlated with surgical outcome.[154,159,160] In tandem with smoking cessation programmes, the optimisation of pulmonary function should be a priority in the pre-optimisation of patients with AAA.

Smoking cessation and physiotherapy can reduce post-operative complications. Level 2a, Recommendation A.

All patients undergoing AAA repair should have an assessment of their respiratory function (with referral to a respiratory physician to optimise respiratory disease prior to surgery if considered appropriate). Level 5, Recommendation D.

Pharmacotherapy for AAA patients

Statins

Two randomised trials and a number of cohort studies have demonstrated the effect of a short pre-operative course of statins to improve cardiac morbidity and mortality within 30 days of vascular surgery.[161—165] The recent trial of fluvastatin (80 mg daily for 30 days before surgery and continued until at least 30 days after surgery) showed that treatment with fluvastatin significantly halved both the primary 30-day outcome of post-operative myocardial ischemia and the secondary outcome of non-fatal myocardial infarction and cardiovascular death.[162] Almost half of the patients in this trial underwent surgery for abdominal aortic aneurysm, spread evenly between open and endovascular repairs. These findings have been supported by a number of other trials.[166—168]

Statins should be started one month before intervention to reduce cardiovascular morbidity. Level 1a, Recommendation A.

Statins should be continued in the perioperative period, for an indefinite duration. Level 3b, Recommendation C.

β-blockade

The DECREASE research group previously conducted a clinical trial showing similar benefits for the use of pre-operative bisoprolol, started about 1 month before surgery, in

vascular surgical patients of the highest cardiovascular risk.[169] Recently they suggested that there also may be a reduction in cardiovascular morbidity when bisoprolol is started well before surgery in intermediate-risk patients.[170] There is no evidence that β-blockade reduces either aneurysm expansion rate or rupture risk.[64,65,67,68,154]

For many patients surgery cannot be delayed for 1 month or more. Large clinical trials where β-blockade was started a few days before surgery, such as POBBLE, POISE and MaVS, have indicated either no benefit or even harm for perioperative beta-blockade.[171–173] These trials all used short duration (perioperative) treatment with metoprolol in a number of different patient groups. These included vascular surgical candidates and specifically AAA repair patients. Both MaVS and POBBLE demonstrated that patients treated with metoprolol prior to surgery did not have a lower rate of cardiac events or death in the perioperative period (POBBLE adjusted risk ratio 0.87; 95% confidence interval, 0.48–1.55; MaVS relative risk reduction 15.3%, 95% CI -38.3% to 48.2%).

These findings would suggest that short course β-blockade is not without significant complications and should be avoided. These negative effects are linked with perioperative bradycardia or hypotensive episodes and might be related to inadequate perioperative monitoring.[174] Longer-term β-blockade, when patients can be assessed for adequacy of effect preoperatively (aiming for a heart rate of 60–70 bpm) is a safer treatment strategy.

Only use β-blockade in the patients of highest cardiac risk and if β-blockade can be started one month before intervention. Level 1b, Recommendation A.

β-blockers are recommended in patients with ischaemic heart disease or who have myocardial ischemia on stress testing. Level 2a, Recommendation B.

Anti-platelet therapy

The evidence for anti-platelet therapy is, in part, based on a meta-analysis of primary and secondary prevention randomised trials.[175] None of the trials investigated AAA patients specifically although those on secondary prevention did consider patients with proven vascular disease. The results suggested that, in terms of secondary prevention, the use of low-dose aspirin was associated with a reduction in major coronary events (RR 0·80 [0·73–0·88], $p < 0·00001$) including non-fatal myocardial infraction (0·69 [0·60–0·80]) and coronary heart disease-related mortality (0·87 [0·78–0·98]). In terms of stroke, a significant reduction in all stroke (0·81 [0·71–0·92]) and ischaemic stroke (0·78 [0·61–0·99]) was seen, but at a non-significantly increased risk of haemorrhagic stroke (1·67 [0·97–2·90]). A trend level significance for a reduction in all vascular deaths was demonstrated (RR 0·91 [0·82–1·00], $p = 0·06$) with no significant effect on non-vascular mortality (RR 0·85 [0·66–1·08], $p = 0·2$), yielding a 10% reduction in total mortality (RR 0·90 [0·82–0·99], $p = 0·02$).

Specific evidence in regard to the prevention of perioperative cardiac events remains limited. It is a pragmatic recommendation that all patients with AAA should be started on aspirin therapy at the time of AAA diagnosis and this should be continued through the perioperative period as the risk of significant haemorrhage appears low.[176,177] Patients on warfarin therapy should stop this 5 to 7 days prior to AAA repair to prevent haemorrhage and be placed on to low-molecular weight heparin unless there is a contraindication to their use (e.g. renal failure), in which case un-fractionated heparin should be used.

Patients with vascular disease should be started on low-dose aspirin therapy, unless specific contraindications exist. Level 1a, Recommendation A.

Patients with AAA should be on low-dose aspirin and this should be continued through the perioperative period. Level 3b, Recommendation C.

Hypertension

Blood pressure control should be achieved from the time of diagnosis of AAA. The full guidelines for the management of hypertension are outside the scope of these vascular surgical guidelines, but are published by national bodies.[178,179] All vascular specialists managing aortic aneurysms should have robust referral patterns established with specialists in the management of hypertension, including complex or refractory cases.

Blood pressure control should be initiated for secondary prevention to reduce cardiovascular morbidity. Level 2a, Recommendation B.

Vascular surgeons should be familiar with their current national guidelines for the management of hypertension. Recommendation A.

Treatment for patients with small aneurysms should be initiated by community physicians with a target blood pressure of less than 140/90 mmHg.

Robust referral pathways should exist for refractory hypertension.

Pre-operative cardiac evaluation

Patients undergoing AAA repair have a high cardiac risk, which carries an associated mortality. Ischaemic cardiac events are a major cause of perioperative morbidity and mortality in non-cardiac surgery with 10–40% of perioperative deaths being due to myocardial infarction. This can be effectively reduced through detailed pre-operative cardiac assessment of patients to identify those at the highest risk (for medical therapy see section 2 above).

All patients undergoing AAA repair should be assessed for cardiac risk. A thorough medical history, resting ECG and assessment of cardiac symptoms is the starting point, eliciting details of previous myocardial infarction, angina pectoris (stable or unstable), congestive heart failure, diabetes mellitus, renal failure, and a history of transient ischaemic attack (TIA) or cerebrovascular accident (CVA), all of which affect outcome.

Based on the planned operation (EVAR, laparoscopic or open repair) and the patient's symptoms, the cardiac risk assessment and initiation of cardio-protective medications should follow the publication of the recent European Society of Cardiology guidelines for perioperative cardiac care.[180,181] These have been recently summarised in reference to vascular surgery.[182] Urgent referral to a cardiologist to consider optimisation of cardiac function before aneurysm repair should be considered for all patients of medium to high cardiac risk.

Two trials have assessed the role of prophylactic coronary revascularisation in vascular surgical patients — CARP[183] and DECREASE-V.[184] The latter investigated a higher risk group of patients than CARP, with a large number of patients having left mainstem, or three-vessel disease and left ventricular ejection fractions below 35%. Both studies demonstrated that there was no difference in the primary outcome measures of mortality or myocardial infarction in patients who had undergone revascularisation (either CABG or PCI) or not prior to vascular surgical intervention.

All patients undergoing AAA repair should have a formal assessment of their cardiac risk. This includes a pre-operative ECG in all cases. Level 1c, Recommendation A.

Patients undergoing open or laparoscopic AAA repair, in the presence of cardiac risk factors, or a positive cardiac history, should undergo a pharmacological stress echo or myocardial perfusion scan prior to surgery. Level 2b, Recommendation B.

Patients undergoing EVAR, in the presence of cardiac risk factors, or a positive cardiac history should have a transthoracic echocardiogram and consideration of a pharmacological stress test or myocardial perfusion scan prior to AAA repair. Level 2c, Recommendation B.

Coronary revascularisation should be considered prior to AAA repair for patients who have ischaemic coronary symptomatic or left main coronary artery disease. Level 1b, Recommendation B.

The role of ECG-gated coronary CT as a diagnostic adjunct should be actively evaluated by clinicians in vascular surgical practice. No evidence-based recommendation can be made at present as to which patients will benefit most from this technique.

Renal investigation and optimisation

Pre-operative renal function is a major determinant of outcome from AAA repair, whether by open or endovascular repair.[153,159,160,185–187] All patients should have their serum creatinine measured and renal creatinine clearance (eGFR) estimated pre-operatively. If these lie significantly outside the normal range, a review by a renal physician for the optimisation of medications prior to aneurysm repair must be undertaken. All patients should be adequately hydrated prior to AAA repair, especially where intravenous contrast is to be employed.

All patients must have serum creatinine measured and eGFR estimated preoperatively. Level 2c, Recommendation C.

Referral to a renal physician is advised where these are outside the normal range.

All patients should be adequately hydrated prior to AAA repair.

AAA repair should only be undertaken in hospitals where there are the facilities for haemofiltration on-site 24 hours a day. Level 5, Recommendation D.

Anaesthesia

The outcomes of AAA repair might be improved when the anaesthetic is performed by specialists in vascular anaesthesia. Consequently, a pre-operative assessment by an anaesthetist familiar with the current literature on the management of patients with AAA is desirable in all patients. The intra-operative management of AAA repair by a specialist vascular anaesthetist also is desirable. There remains debate about the best type of anaesthetic in EVAR; general or locoregional. This is expanded in the operative repair chapter. These issues are considered in more detail in Chapter 5.

All medium and high risk patients being considered for an AAA repair should be reviewed by a specialist vascular anaesthetist prior to admission for surgery. Level 5, Recommendation D.

Risk indices

A number of mathematical models have been generated to aid surgeons in selecting patients for AAA repair. No system has been shown to be entirely reliable especially on external validation using different patient populations and many of the models require recalibration. Specific tools for endovascular repair are becoming available, quantifying the risks of endoleak and mortality based on both morphological and anatomical criteria.

Where debate exists about a patient's fitness, risk stratification based on physiological, and morphological for EVAR, parameters should be undertaken. Level 2c, Recommendation D.

The management of large aneurysms

Large aneurysms (those with a maximum aortic diameter of greater than 5.5 cm) carry a significant rupture risk but the data derive from studies of patients considered unfit for or refusing intervention. One study reported annual rupture risks of 10—20% at 6—7 cm; 20—40% at 7—8 cm; and 30—50% at greater than 8 cm.[85] Meta-analysis has indicated that the rupture risk of AAA >6 cm in diameter is 27 per 100 person-years.[86] Large aneurysms detected at screening, or through imaging investigating another pathology, should be referred *immediately* to a vascular surgeon directly for appropriate imaging and aneurysm repair, since the risk of interval rupture is very high.

All aneurysms over 5.5 cm, or 5.2 cm in females, should be referred for an urgent surgical opinion for imaging and to plan intervention before aneurysm rupture. Level 3a, Recommendation C.

In-patient management might be considered for aneurysms over 9 cm in diameter. Level 5, Recommendation D.

The management of iliac aneurysms

Coexisting iliac aneurysms should be treated concurrently with AAA, and aortoiliac aneurysms comprise up to 43% of a specialist vascular surgeon's workload.[188] Isolated iliac aneurysms may be treated by either open or, preferentially, endovascular techniques. Intervention should be considered when the iliac diameter exceeds 3 cm. Further details can be found in Chapter 5.

Iliac aneurysms should be repaired once the diameter exceeds 3 cm. Level 3a, Recommendation C.

Endovascular treatment options should be considered in all patients and in defined subgroups this will include the consideration for iliac branch graft placement. Level 3a, Recommendation C.

Imaging

Concurrent with vascular surgical referral, formal vascular imaging is warranted to determine aneurysm, extent,

morphology and suitability for EVAR. This should normally be through contrast-enhanced computed tomography (CT) unless a contraindication exists. In these circumstances contrast-enhanced magnetic resonance imaging (MRI) is the most appropriate imaging modality. In exceptional circumstance (e.g. severe contrast allergy), non-contrast CT may be employed. Imaging should be of the whole thoracic and abdominal aorta, as 15% of patients with an AAA will have a co-existing thoracic aneurysm requiring management. Inferiorly, imaging should continue to the femoral bifurcation to allow for the complete assessment of access vessels for EVAR. Superiorly, the supra-aortic trunks should be included. When possible, ECG-gated scanning techniques should be employed along with contrast-bolus tracking when appropriate. These techniques increase the reliability of the information derived from cross-sectional imaging modalities.

For patients with multiple comorbidities and poor fitness scores but who are anatomically suitable for EVAR, optimising the management of comorbidities (including coronary angioplasty, prescription of statins, physiotherapy to improve lung function etc) should be prioritised ahead of aneurysm repair: the case for compassionate aneurysm repair remains unproven. Such patients with short life expectancies gain little in the first 3 years after EVAR,[119] partly because anatomical suitability for EVAR appears to be associated with a reduced rupture risk.[120,189]

Infra-renal AAA operative repair

Modality of repair, including patient preferences:

For all patients whether fit or unfit for open repair EVAR should be taken into consideration. As endovascular experience increases in tandem with the advent of lower profile endografts more able to comply with adverse anatomy and with superior fixation, then the large majority of patients will be suitable for EVAR. These issues are discussed further in Chapter 5.

Where patients are fit and profess an informed preference for open repair, or are anatomically unsuitable for EVAR (with standard endografts), or are unlikely to attend for post-operative surveillance, then open repair should be offered as an alternative to EVAR. For patients with aneurysms of 5.5—7.5 cm in diameter where effective repair can only be performed by using a custom-made endograft, or fenestrated stent, the risk of rupture whilst awaiting a custom graft is approximately 18 per 100 patients-years.[86] The risks and benefits of waiting must be discussed with the patient. Intervention type may be left to patient preference. For patients with the largest aneurysms (those at highest risk of interval rupture) immediate open surgical repair should be considered. For patients with urgent or symptomatic aneurysm, or even contained rupture, there is no evidence as to whether there are significant risks of rupture in awaiting either a specific or custom endograft. Many of these patients therefore may be offered immediate open repair.

Increasing importance is being given to the role of the patient in the decision-making process and patient-clinician agreement about treatment pathways. Three studies, all from the UK, formally evaluated the preferences for any future intervention of patients in small-aneurysm surveillance programmes.[120,121,190] The smallest study was based on telephone interviews with 100 hospital patients and showed that 84% would prefer EVAR if possible, principally because of the lower early mortality risk associated with this procedure. The largest study assessed the views of subjects in community screening programmes, without any exposure to the hospital environment, with a postal information pack and survey; 46% had some preference for EVAR, 18% had some preference for open repair with the remainder undecided or willing to let the clinician decide.[121] An important reason associated with preference for open repair was the absence of long-term surveillance[121] although this was not supported in another study of screened patients.[190] In this latter study, in excess of 90% of patients attending a regional AAA screening service showed a strong preference for EVAR and for treatment at high volume, low mortality centres. The long-term results of the EVAR 1 trial, which highlights the erosion of the early survival benefits of EVAR by late endograft ruptures may swing patient preferences away from EVAR.[119]

The patient's preference for type of aneurysm repair should be considered. Level 2a, Recommendation B.

Patients with large aneurysms who require a custom-made endograft should be offered open aneurysm repair. Level 5, Recommendation D.

Laparoscopic aneurysm repair

Laparoscopic AAA repair offers patients a third option for AAA repair that provides the durability of an 'open' sutured graft with a rapid recovery and reduced length of hospital stay similar to EVAR. Laparoscopic repair may be offered if patients are morphologically unsuitable for stent graft placement in centres without a fenestrated endograft practice or if the patients are concerned about lifelong EVAR surveillance.[191] It may be particularly suitable for younger, screen-detected patients who do not wish to undergo EVAR but want the advantages of minimally invasive surgery. A variety of different techniques are encompassed in the term laparoscopic aneurysm repair including total-laparoscopic repair, hand-assisted laparoscopic repair and robotic-assisted laparoscopic repair. These are considered together for the purposes of these guidelines.

Currently, the role remains limited and should be confined to centres with a specific expertise in laparoscopic aneurysm repair. This is in part due to the requirements for advanced laparoscopic practice, and also due to the steep learning curve for this procedure.[192—194] The 30-day in-hospital mortality outcomes for laparoscopic AAA repair (2—6%) fall between those for EVAR (1—5%) and open surgery outcomes in the UK (6.7—7.9%).[193,195,196] Laparoscopic AAA repair in the obese patient is feasible and negates some of the access difficulties and potential complications experienced with open repair.[197]

It should be noted that the cardiac risk of laparoscopic procedures should be considered to be the same as for open repair.[180]

Laparoscopic aneurysm repair should only be attempted in centres with an advanced laparoscopic practice and where suitable mentoring is available.

Procedures should initially only be carried out under supervision from someone experienced in laparoscopic aneurysm repair. Facilities to deal with emergency surgical conversion should be available at all times. Its role remains limited, but in selected patients it might represent a third option for AAA repair. Level 4, Recommendation C.

Hospital-volume, surgeon-volume and co-dependency of other specialties

AAA repair should be undertaken in centres with sufficient experience of elective AAA repair. Current evidence would suggest that, as relationships exist for both open repair and EVAR between annual workload (volume of AAA repairs) and outcome, this means a minimum of 50 elective infra-renal AAA repairs per annum.[145,147,198,199] Similar relationships between volume and outcome have been reported for non-elective aneurysm repair.[200–202] The best results are achieved in hospitals performing high volumes of elective and emergency aneurysm surgery by high-volume specialist vascular surgeons.

AAA repair should only be performed in hospitals performing at least 50 elective cases per annum, whether by open repair or EVAR. Level 2c, Recommendation B.

Surgeon experience and specialisation

Sufficient evidence exists to suggest that elective AAA repair should only be performed by vascular specialists who undertake a high annual volume of AAA repairs.[203] This is true for both open repair and EVAR. This means that general surgeons with a vascular interest should consider the long-term feasibility of continuing to perform any arterial surgery and aortic surgery in particular. It is also possible that many vascular surgeons will no longer have the experience or support services to safely undertake aneurysm repair, particularly open repair, and should consider referral to aneurysm specialists in an appropriate unit.

Symptomatic AAA

Symptomatic aneurysms may present with abdominal, back pain or embolic events. These aneurysms are thought to have a higher rupture risk than asymptomatic aneurysms. The management of these cases is through urgent surgical repair on the next available elective operating list. Repair should preferentially be with EVAR, where anatomically suitable.[200–202]

Symptomatic aneurysms should be repaired on the next available elective operating list as they have a higher risk of rupture. Level 5, Recommendation D.

Where morphologically suitable, patients should be offered EVAR, which has a lower operative mortality for symptomatic cases than open repair. Level 2c, Recommendation B.

Evidence needed and evidence in progress

A great number of questions remain unanswered in the management of AAA. Whilst optimising the outcome of operative repair and screening to prevent AAA rupture represent effective management at the far end of the spectrum of aneurysmal disease, the ultimate goal is to put effective primary care strategies and pharmacological management in place to prevent expansion much earlier in the disease process.

The role of ACE inhibition remains poorly defined in the management of AAA. The influence of ACE inhibitors on aneurysm expansion and rupture rates and any affect on the outcome of subsequent repair is poorly understood, with conflicting evidence being available. An RCT is currently underway to clarify these questions. Experimental studies are providing the basis for the evaluation of other drugs, including thiazoledinediones in the management of AAA.

Chapter 4 — Pre- and Perioperative Imaging

Pre-operative imaging

Several imaging modalities can be used in the preprocedural care of patients with an AAA, such as digital subtraction angiography (DSA), (duplex) ultrasound, intravascular ultrasound (IVUS), computed tomography angiography (CTA), and magnetic resonance angiography (MRA). These specific imaging techniques, all with their own indications, advantages and limitations, will be discussed here.

Duplex ultrasound

Preoperatively, ultrasound is the modality of choice for the detection and surveillance of an AAA in an asymptomatic patient.[204–206] Ultrasound is relatively cheap, non-invasive, widely available, and reliable. The specificity and sensitivity of ultrasound for the detection of AAAs in asymptomatic patients is almost 100%.[204,207,208] A disadvantage of ultrasound is that the aorta can not be visualised properly due to obesity or bowel gas in a minority of AAA patients. Moreover, the determination of aortic diameters by ultrasound is subject to operator variability.[209]

It is therefore advisable to perform imaging, additional to ultrasonography, if an AAA is approaching a size requiring intervention, or if rapid growth is suspected. Level 2, Recommendation A.

Investigation of the supra- and infra-renal borders of an AAA, the presence of periaortic disease, and of additional iliac aneurysms is not reliable on ultrasound.[208,210,211] Ultrasound is not suitable for the pre-operative work-up of an AAA patient and other imaging modalities will therefore have to be used.

Contrast-enhanced ultrasound has no proven additional value in the pre-operative work-up of AAA patients. Level 5, Recommendation D.

Digital substraction angiography (DSA)

DSA was commonly used as a pre-operative work-up modality in the past. Advantages of DSA are the visualisation of the true lumen of the aortoiliac arteries and its sidebranches. Direct intervention prior to aneurysm repair for significant problems, as renal or iliac artery stenosis, is possible while performing DSA. DSA, however,

has some major drawbacks: it images the true lumen of vessels, and the actual size of vessels and aneurysms can be underestimated due to the presence of thrombus. Moreover, DSA is invasive and exposes patients to iodinated contrast. It is for those reasons that DSA has lost its importance as a primary pre-operative work-up modality for AAAs.

DSA is not recommended as a routine pre-operative imaging modality. Level 5, Recommendation D.

Intravascular ultrasound (IVUS)

IVUS, another invasive method, can also be used pre-operatively. An advantage of IVUS is that no contrast is used and IVUS can measure aortic diameters and lengths accurately.[212,213] Moreover, post-processing of IVUS images currently is possible. Besides being an invasive technique, there are several other disadvantages of IVUS: it is not widely available, and requires significant skill and experience in both the performance and interpretation.

Computed tomography angiography (CTA)

The use of sequential computed tomography (CT) provides more information about an AAA and the surrounding structures including venous anomalies, retroaortic left renal vein and renal anomalies such as a horseshoe kidney. CT is also adequate to identify inflammatory aortic aneurysms, but CT is not optimal to provide detailed information about the arterial anatomy and its sidebranches.[214]

On the contrary, CTA is both a powerful tool for planning EVAR and open surgical repair.[215] Multidetector CT systems with, for example, 16-, 32-, 64-, 128- and even 256-detector rows are currently available. An advantage of CT systems with 128- or 256-detector rows over systems with 16- or less detector rows is the decreased scanning time, making the use of less contrast agency possible. The aortic borders are very clear on images acquired with 128- or 256-detector row CT systems, but represent the aorta in the systole, diastole, or somewhere in between.[216] Aortic images acquired with a 16- or less detector CT scanner result in a less clear border of the aorta, but this more or less represents the mean size of the aorta during the cardiac cycle.

CTA is a fast, and reproducible modality, and provides all necessary detailed anatomical information for operation planning.[217] CTA is able to visualise the entire relevant arterial anatomy including the surrounding anatomy.[218,219] CTA can provide 3D information and dynamic images, which has become more valuable since the introduction of endovascular aneurysm repair (EVAR).[216] CTA therefore currently is the primary pre-operative imaging modality in most centres. CTA currently is the primary pre-operative imaging modality. Level B, Recommendation 2c.

In addition, several CTA findings have been reported to be predictive of rupture of the aneurysm such as aortic blebs and discontinuous aortic calcifications.[220] The disadvantages of CTA include the radiation burden and the use nephrotoxic contrast agents.

Magnetic resonance angiography (MRA)

CTA is most often used, but MR and MRA can also be of use in the pre-operative assessment.[221] The use of contrast in MR imaging of the aorta is not necessary, but enhances the image quality. Most MR contrast agents in clinical use are chelates of gadolinium. Benefits of MRA are the high soft-tissue contrast, the possibility of arterial wall movement and flow quantification, and the ability to evaluate both the vascular lumen and its wall.[208,216] MRA is comparable to CTA for measurement of aneurysm diameter and accurate in determining the extent of aortic involvement in inflammatory aneurysms.[222] An MRA is obtained without the use of iodinated contrast material or radiation burden, which is important as MRA usually requires more than one sequence to generate a complete overview of the entire imaged area.[223] Since MR techniques have no ionizing radiation, MRA can be used to demonstrate multiple phases of vascular contrast, including arterial, venous and delayed phases. Drawbacks of MRA are its susceptibility to artefacts, and the acquisition of images can be time-consuming. The costs of MRAs are generally higher compared to CTA, and MRA can be contraindicated in patients with claustrophobia or implantations.

Post-processing of CTA and MRA images has become more important with the introduction and the increasing use of EVAR. Multiplanar, centre lumen line (CLL), and 3D reconstructions provide valuable information in the pre-operative planning of AAA patients.[224] These reconstructions are valuable to investigate whether a patient is suitable for EVAR. A CLL is required for appropriate stent graft sizing prior to EVAR. It allows for diameter measurements perpendicular to the aorta, and correct length measurements alongside the aorta.[224] Aortic 3D reconstruction and a CLL are necessary for the pre-operative investigation of aortic angulations.[225] Furthermore, reconstructed images can be used to preoperatively determine the most optimal C-arm position during EVAR as well.[224]

Perioperative imaging

Several imaging modalities can be used during both open and endovascular exclusion of an aortic aneurysm. Duplex ultrasound and DSA can be used to investigate the proximal and distal anastomosis and the patency of possibly inserted visceral vessels after the open or laparascopic exclusion of an AAA.

Digital substraction angiography (DSA)

The main use of angiography is during and after EVAR. The performance of this procedure on a radiolucent operating table is therefore strongly recommended. A perioperative DSA will be obtained prior to proximal stent graft deployment, with the C-arm in the most optimal position. The C-arm needs to be angulated perpendicular to the aneurysm neck and perpendicular to the armpit of the most distal renal artery. Furthermore, to avoid parallax, it is important to keep the area of interest centred in the screen when a non-flat detector is used. Possible additional DSAs may be acquired prior to distal stent graft deployment. A completion

angiography will have to be obtained after the placement of a stent graft, to investigate the stent graft position, the patency of sidebranches and the presence of endoleaks.

Iodinated contrast agents are the medium of choice for angiography, but they carry the risk of nephrotoxicity or anaphylaxis.[226] Low-osmolality iodinated contrast agents are generally preferred since they reduce the incidence of such adverse events, when compared to high-osmolality agents. Carbon dioxide arteriography is a non-nephrotoxic alternative, but the obtained images are frequently inadequate.[227] Gadolinium is another non-nephrotoxic contrast medium, and is considered to be an alternative for iodinated contrast agents in patients with renal insufficiency.[226]

An alternative for perprocedural angiography is IVUS, allowing for perioperative real-time diameter and length measurements. Perioperatively, IVUS can be a useful tool in patients without, or with an indecisive, pre-operative CTA or MRA. IVUS can help in reducing the amount of peri-operative contrast used. However, as discussed earlier, this technique is not widely available, difficult to perform, and adds time to the procedure.

Quite recently, a newer imaging technique, the on-table angiographic CT modality has been introduced. This imaging technique acquires CT-like images and might help in the detection of complications which are possibly missed by unipolar angiography. Currently, the field of view of these techniques is still limited and the acquired images have a lower resolution, compared to CTA. Nevertheless, the on-table angiographic CT is still evolving and is a promising technique for the near future.[228,229]

Chapter 5 — Management of Non-ruptured AAA

Open repair of non-ruptured AAA

Perioperative management

Antibiotics

A single shot prophylactic treatment with systemic antibiotics is recommended in any arterial reconstructive surgery. Thus wound infection and early graft infection are prohibited in almost three-quarters of patients. Medication should be administered within 30 min prior to skin incision. There is no clear evidence for an advantage of first- or second-generation cephalosporins, penicillin ± β-lactamase inhibitor, or aminoglycosides.[230,231]

A single shot antibiotic prophylaxis in patients with abdominal aneurysm repair is recommended to avoid early graft infection and wound infection. Level 2c, Recommendation B.

Body temperature

Hypothermia (<36 °C) is a risk factor for perioperative complications. Elmore *et al*. saw that patients who were hypothermic had lower cardiac output and platelet counts, higher prothrombin times and APACHE II scores, and greater incidences of sinus tachycardia and ventricular arrythmias.[232] Therefore maintenance of body temperature either by air-warming blankets or warmed inhaled gasses and fluids during aneurysm repair is beneficial.[233,234]

Body temperature should be kept at a physiological level (>36 °C) during AAA repair to avoid perioperative complications. Level 3b, Recommendation B.

Intraoperative fluid resuscitation and blood conservation

Loss of fluid during aortic surgery is on one hand due to blood loss, and on the other hand extracellular loss, due to the development of tissue edema, typically 1 L per hour during surgery and continuing into the immediate post-operative period. Especially before 'declamping' an adequate volume regimen is important to avoid the declamping shock with the blood release into a vasodilated ischaemic periphery. Although there are 38 randomised trials following the question of the best fluid management during aortic surgery, there is not enough evidence on the benefits of any particular individual or combination fluid therapy. Crystalloid solutions and colloids are commonly used with few differences in important outcomes, such as the need for allogenic blood transfusion, complications of organ failure, and length of post-operative hospital stay.[235]

Intraoperative blood salvage during aortic aneurysm repair either with red-blood-cell processors or haemofiltration devices is widely used. Although the centrifugation product of the cell processors is pure and efficient, platelets and clotting factors are lost. A review of the available literature shows that cell salvage techniques are not able to reduce the need of transfusion and do not help to reduce costs[236] in AAA repair. The use of cell salvage and ultrafiltration devices might nevertheless be recommended if large blood loss is likely, and if the risk of transfusion-related complications or disease transmission from banked blood is considered high. Transfusions of red blood cells should be considered if blood loss is ongoing and if the haematocrit is lower than 30%.[237]

No specific fluid-replacement strategy has been shown to be superior to another in the use of abdominal aortic surgery. A combination therapy from crystalloid and colloid solutions is most commonly used. Level 1a, Recommendation B.

In case of an expected large blood loss and if the risk of transfusion-related disease transmission is considered high, the use of cell salvage and ultrafiltration devices might be recommended. Red blood cells should be transfused if blood loss is ongoing and if the haematocrit is lower than 30%. Level 2b, Recommendation B.

Fast-track surgery

What was initally introduced in colorectal surgery has now become more common in patients with abdominal aortic surgery. The fast-tracking multidisciplinary programme aims to reduce periprocedural ischaemic complications and to facilitate early rehabilitation. In elective open aortic aneurysm repair severe complications such as myocardial infarction, pneumonia and acute renal failure can be observed in about 60% of patients.[161,238] Following the conventional concept of perioperative management, the median duration of ventilator use is 1.3 days and the median length of stay on intensive care unit is 3.2 days.[239] Meanwhile some high volume centres follow a fast-track regimen, consisting of:

- Patient education and instruction preoperatively
- Shortening of the pre-operative fasting to 2 h before the surgery

- No bowel washout
- Increased temperature of the operation room to 22 °C
- Pain control by pre-operatively inserted epidural catheter
- Enteral feeding and ambulation on the evening of the surgery
- Restriction of intravenous fluid application to 1L/24 h

Brustia *et al*. applied this concept among 323 unselected patients for open abdominal aortic surgery and found that they could significantly improve perioperative outcome (no need of stay on a intermediate care unit, restoration of the ambulation on the evening of the surgery) with a median post-operative discharge home after 3 days.[243] Muehling *et al*.[244] also report on the fast track programme, in a randomised trial with 82 patients they found that the traditional group had a significantly higher need for assisted post-operative ventilation (33.3% vs 5.4%). In addition the median length of stay on an intermediate care unit could be shortened and the rate of post-operative medical complications was significantly lower in the fast-track group (16.2% vs 35.7%).[242,245]

Both authors conclude, that with the fast track program post-operative morbidity after aortic aneurysm repair can be optimised.[243, 245]

Fast-track surgery can positively influence perioperative outcome after AAA repair. Appropriate outpatient pre-operative work-up with admission close to the time coupled with judicious fluid management and early mobilisation can lead to improved outcomes and reduced ICU/total lengths of stay. Level 2b, Recommendation B.

Type of incision for open repair

Access for open repair is either through a trans-abdominal or retroperitoneal approach; for the former there are options of transverse or vertical incisions and the length of incision may be important to patient recovery. Although development of incisional hernia is uncommon, a small trial has indicated that transverse incisions has been reported to reduce the incidence of incisional hernias.[246] Comparison of long vertical trans-abdominal incisions versus retroperitoneal incisions have been made in three small underpowered randomised trials, conducted 15—20 years ago.[247—249] The first two trials, conducted in the USA, found that retroperitoneal incisions may be associated with an improved post-operative course and shorter length of hospital stay, whereas the third, an Australian trial found no differences. No meta-analysis has been conducted. More recent work indicates that shorter trans-abdominal incisions are safe and may be associated with an improved post-operative course and shorter length of hospital stay.[250]

There has been little recent good quality research to improve the outcomes of open repair and with the increasing use of EVAR, the opportunity for a large trial of mini-laparotomy may have been missed. The increasing use of EVAR also means that the technical challenges associated with open repair may be heightened.

In the absence of convincing evidence favouring any one type of incision, the incision for open repair should be tailored to the patient needs and local expertise. For instance, the presence of a hostile abdomen provides an indication for the retroperitoneal approach, as does juxtarenal extension of the aneurysm, inflammatory aneurysms or horseshoe kidney. Level 2b, Recommendation C.

Graft configuration

There are several prosthetic grafts available for aortic replacement: knitted or woven Dacron, impregnated with collagen, albumin or gelatine if needed, and polytetrafluoroethylene (ePTFE). All materials show excellent patency and long-term results, so that the surgeon's preference and the costs determine the aortic graft choice.[251—253] The literature following the question of the optimal prosthesis for elective aortic replacement is controversial. Prager *et al*. found a comparable long-term patency for PTFE and Dacron, but PTFE had a higher incidence of early graft failure and graft infection.[254]

Because of the convincing handling characteristics, knitted Dacron is the material most commonly chosen. The need for preclotting can be avoided by using impregnated Dacron grafts, which makes these materials first choice grafts in the case of a ruptured aortic aneurysm.

Aneurysm size and extent determine the configuration of the graft. Because operative time is shorter, tube grafts are preferred to bifurcated grafts. A further advantage of the tube graft is the opportunity of a reduced dissection with less risk of injury to adjacent structures such as the ureter, iliac veins, or parasympathetic nerves. In the case of additional iliac artery aneurysms or a concomitant arterial occlusive disease, indication for a bifurcated graft is given, if necessary all the way to the groins. In such cases, a higher incidence of wound infection, graft limb thrombosis, and anastomotic aneurysm has been reported.[255]

Available prosthetic graft materials for abdominal aortic aneurysm repair are comparable concerning patency and long-term results. Level 3b, Recommendation B.

If the iliac arteries are unaffected (aneurysm formation or arterial occlusive disease) tube grafts should be used because of the shorter operative time and the reduced risk of adjacent injuries of the neighbouring structures. Level 2b, Recommendation A.

Pelvic circulation

The status of pelvic blood supply should, if ever possible, be investigated pre-operatively to avoid post-operative problems such as buttock claudication and colonic ischaemia.

In patients with AAA, the inferior mesenteric artery is patent in more than half of cases.[256] The ligation of a patent inferior mesenteric artery is among the most reported risk factors for the development of a colonic ischaemia.[257] The decision towards ligation is controversial. The artery can be ligated if:

- It has good backflow on release
- Pulsations of the mesenteric arcade branches are satisfactory
- At least one hypogastric artery is patent.

In the case of impaired sigmoid colon perfusion, particularly if the hypogastric arteries are diseased or excluded from the circulation, the inferior mesenteric artery needs

to be reimplanted. In questionable cases, Doppler signals from the bowel can assess bowel viability.[258,259]

To grant sufficient perfusion of the pelvic organs, at least one hypogastric artery should be preserved during aneurysm repair. Failure to accomplish this might cause a variety of problems such as erectile dysfunction, symptomatic hip and buttock claudication, in rare occasions colon ischaemia, buttock necrosis, or spinal cord (cauda equina) ischaemia. With increasing endovascular techniques in aortic aneurysm repair, the hypogastric artery is frequently embolised prior to aneurysm repair. Literature reports indicate that the incidence of buttock claudication is about 30% (178 of 634 patients in one study) after hypogastric artery embolisation: 31% of unilateral embolisations (99 of 322) and 35% of bilateral embolisations.[260]

In the presence of impaired pelvic and sigmoid colonic perfusion, the inferior mesenteric artery needs to be reimplanted during aortic aneurysm repair. The perfusion of one hypogastric artery or the inferior mesenteric artery is mandatory to avoid post-operative complications. Level 3, Recommendation B.

Table 7 Perioperative complications following open aneurysm repair[242]

Cardiac complications in 5.4% of patients:
arrythmia 3%
myocardial infarction 1.4%
congestive heart failure 1%
Pulmonary complications in 4.2% of patients
pneumonia 3%
adult respiratory distress syndrome 1%
pulmonary embolism 0.2%
Renal complications with renal insufficiency in 1.7% of patients
Sepsis in 0.7% of patients
Stroke in 0.4% of patients
Local complications were observed as:
Wound complications in 3.3%
Intestinal obstruction and ischemia in 2%
Retroperitoneal bleeding in 0.4%
Amputation in 0.1%[274]

Perioperative mortality and morbidity

Depending on the study design and patient selection the perioperative 30-day mortality rate after open aortic aneurysm repair differs widely and ranges between 1% and 8%, with selected centres of excellence reporting a 1% mortality rate. In multiple population-based series and state- or nation-wide databases perioperative mortality rates reaches 8%.[240–242,255,261–271]

Peri-operative ICU care

Patients undergoing open aneurysm repair should be managed in critical care areas that are experienced in post-operative fluid optimisation. The optimisation of cardiac output and non-invasive or invasive moitoring has been shown to reduce the post-operative complication rate and mortality in surgical patients, including AAA repair. ITU length of stay and total length of stay are also reduced. Recent evidence has shown that these benefits extend to 15 years post-surgery.[272] Furthermore, the early identification and proactive management of post-surgical complications has been shown to significantly reduce early surgical mortality.[273]

Outcome after open aortic aneurysm repair

Many authors focused on risk factors for post-operative death following elective surgical repair. Brady *et al.* investigated in the UK Small Aneurysm Trial that an impaired pre-operative lung (assessed by FEV1) and renal function (assessed by creatinine level) were strongly associated with post-operative death. The cut-off levels for an increased perioperative mortality rate for FEV1 ranked at 2.2 L, for creatinine at 104 μmol/L, respectively. Age did not matter in the fully adjusted model.[238]

Hertzer and colleagues presented data of the open aneurysm repair in the Cleveland Clinic between 1989 and 1998. The overall 30-day mortality rate was 1.2%. One hundred and fifty (13%) of the 1135 patients experienced perioperative complications (Table 7).

These data are much better than early results from Johnston in 1989 from 666 patients who underwent surgery for non-ruptured abdominal aortic aneurysm. Johnston reports a much higher percentage of cardiac (15.1%) and also pulmonary complications (8.4%). Renal damage was reported in 5.4% of patients. In addition the authors report one case of paraplegia. The percentage of ischaemic colitis reached 0.6% and 11% of patients suffered from a prolonged post-operative ileus.[255]

Schlösser *et al.* performed a study on the relationship between gender and age and the mortality risk after elective abdominal aneurysm repair. They saw that the mortality risks after elective AAA repair was strongly age related: 28-day mortality ranged from 3.3% to 27.1% in men and 3.8%–54.3% in women, 5-year mortality from 12.9% to 78.1% in men and 24.3%–91.3% in women. Female gender, increased age and prior hospitalisation for congestive heart failure were independently and significantly associated with higher 28-day and 1-year mortality in patients with elective AAA repair. Higher age, diabetes mellitus and previous hospital admission for congestive heart failure or cerebrovascular accident were associated with higher 5-year mortality. The authors assume from their findings that a general threshold of 55 mm for surgery might not be justified for all patients.[275]

Similarly, in a study by Hertzer *et al.*, the long-term mortality rate was influenced by age of more than 75 years, or previous history of congestive heart failure, chronic pulmonary disease, or renal insufficiency. A worse outcome was observed in men than women, in patients with a history of congestive heart failure, chronic pulmonary disease, or renal insufficiency. Kaplan–Meier method survival rate estimates were 75% at 5 years and 49% at 10 years. In the long-term follow-up only 0.4% of patients experienced complications that were related to their aortic replacement graft (graft infection, graft limb occlusion, pseudoaneurysm).[242]

Conrad *et al.* performed a more detailed analysis on the long-term durability of the grafts after open elective

aneurysm repair. Among their 540 patients they saw a operative mortality of 3% and post-operative complications in 13% of patients. A history of myocardial infarction, and renal insufficiency served as negative predictors for the perioperative outcome. There were 13 graft-related complications (2%), consisting of seven anastomotic pseudoaneurysms, four graft limb occlusions and two graft infections after a medain follow-up of 7.2 years.[265]

Biancari *et al*. did a retrospective study of 208 patients after aortic aneurysm repair (elective and ruptured) to assess the number of late graft-related complications in a follow-up of 15 years. The total number of complications observed was 15.4% with pseudoaneurysms being the most frequent complications: 2.9% proximal, 8.7% distal and 3.4% bilateral pseudoaneurysms, a limb occlusion occurred in 5.3% of the patients.[266]

To assess the efficacy of elective aneurysm repair, Beck *et al*. performed a study to develop a risk prediction model for the mortality during the first year after elective abdominal aortic aneurysm repair. The analysis of the data of the Vascular Study Group of Northern New England showed that a combination of age, chronic pulmonary disease, renal insufficiency and need for suprarenal clamping had a significant impact on the 1-year mortality after open aortic aneurysm repair.[267]

A history of congestive heart failure, chronic pulmonary disease, or renal disease is associated with increased 30-day mortality and reduced long-term survival after elective AAA repair. Level 2a.

Endovascular repair of non-ruptured AAA

Pre-operative evaluation

Endovascular aneurysm repair (EVAR) is a minimally invasive surgery for the treatment of AAA based on the use of a stent graft, usually deployed inside the aneurysm through femoral access to exclude the AAA sac from the circulation. EVAR requires adequate aortic and iliac fixation sites for effective sealing and fixation. These requirements should be carefully assessed and verified prior to surgery with adequate aortoiliac imaging to select suitable patients for endografting.

Potential advantages of EVAR over open repair (OR) include reduced operative time, avoidance of general anaesthesia, less trauma and post-operative pain, reduced hospital length of stay and less need for intensive care unit (ICU), reduced blood loss and reduced immediate post-operative mortality. Potential disadvantages include the risk of incomplete AAA sealing, with the development of continuous refilling of the aneurysm sac, either because the graft does not seal completely at the extremities (Type I endoleak), between segments (Type III endoleak), or because of backfilling of the aneurysm from other small vessels in the aneurysm wall (Type II endoleak). To monitor the developments of endoleaks and sac behaviour, patients after EVAR may require repetitive imaging to check for the presence of late-occurring complications. In addition, if EVAR is unsuccessful or complications arise during the primary endovascular procedure, conversion to OR may be necessary, therefore a thorough patient evaluation should be completed prior to EVAR to assess the risk of both procedures.

Comorbid disease

Regardless of the type of surgery, coronary artery disease (CAD) is the leading cause of early and late mortality after AAA repair and a substantial proportion of patients with AAA have underlying CAD. Renal insufficiency, diabetes mellitus and chronic obstructive pulmonary disease (COPD) may also influence morbidity and mortality, and therefore their careful evaluation and treatment optimisation should be obtained prior to aortic surgery.

Pre-operative evaluation of cardiac morbidity

Randomised controlled trials, large registries and single center series comparing EVAR with OR have shown that the minimally invasive approach has lower early morbidity and mortality[116,117,148,276–279] with low incidence of primary conversion to OR after EVAR, between 0.9 and 5.9%.[280–285] The DREAM trial reported an operative mortality rate of 4.6% percent in the open repair group and 1.2% in the endovascular repair group, with a higher rate of moderate and severe systemic complications in the open surgical arm. However, cardiac complication rate in this trial resulted similarly in the two groups (5.7% for OR vs 5.3% for EVAR), underlining that even EVAR should be considered a procedure with intermediate to high risk of cardiac complications.[117]

Before the planned endovascular procedure, a detailed cardiac history should therefore be obtained, and patients should be screened for all cardiovascular risk factors. Level 2, Recommendation B.

In the presence of an active cardiac disease, represented by unstable coronary occlusive disease (unstable or severe angina, myocardial infarction within 1 month), decompensated heart failure (new onset, worsening, or New York Heart Association [NYHA] Class IV), significant arrhythmia (atrio-ventricular [AV] block, poorly controlled atrial fibrillation, new onset ventricular tachycardia), or severe valvular heart disease (symptomatic, aortic valve area <1 cm^2 or pressure gradient >40 mm Hg), elective open or endovascular aortic surgery should be deferred until optimal management of cardiac comorbidity has been reached.

Patients with severe cardiac morbidities should have aneurysm repair deferred until optimal management of these morbidities. Level 2b, Recommendation B.

In patients with a history of coronary artery disease, as those with previous myocardial infarction, previous coronary intervention, or present stable angina pectoris, or with other cardiovascular risk factors such as history of cerebrovascular accident or transient ischaemic attack, age >70 years, chronic heart failure, and chronic obstructive pulmonary disease (defined as a forced expiratory volume in 1 second $<$ 70% of age and gender predictive value, or medication use), or renal insufficiency, further testing may be advisable. Pre-operative stress testing should be done according to the number of cardiac risk factors identified at pre-operative screening. Patients without cardiac risk factors usually do not benefit from additional cardiac stress testing, as those with 1 or 2 risk factors, according to the

Dutch Echocardiographic Cardiac Risk Evaluation (DECREASE II) trial.[286] Patients with three or more risk factors should undergo additional testing and eventually invasive treatment if indications are consistent with established guidelines.[180,287]

Cardiac stress testing prior to EVAR is recommended in patients with three or more clinical factors for cardiac disease. Level 2b, Recommendation B.

In case of percutaneous cardiac revascularisation, the need for long-term dual anti-platelet therapy should be taken into consideration for the choice between OR and EVAR in anatomically suitable patients. The endovascular aneurysm treatment can be carried out without discontinuation of the anti-aggregation, given the low risk of bleeding, mostly associated with the estimated risk of conversion to OR during or immediately after the procedure.[288]

Minimal invasive AAA repair can be carried out under dual anti-platelet treatment after drug-eluting coronary stenting. Level 5, Recommendation D.

Patients at high cardiac risk after maximal therapy as well as those who require AAA treatment immediately after cardiac intervention should be better treated with EVAR, if anatomically suitable. Level 4, Recommendation C.

Pulmonary disease

Dependency on home oxygen and COPD have been identified as co-morbidities associated with poor outcome for any major surgical procedure.[289,290] In addition, this condition is known to be associated with an increased prevalence of AAA[291,292] and is an independent predictor of AAA rupture.[135] Thus, patients with severe COPD and AAA are at an increased risk of rupture and have an apparently higher risk for any type of intervention.

Between 7% and 11% of patients with COPD have an aneurysm and failure to optimise COPD management is associated with increased morbidity and mortality. If COPD is severe, formal pulmonary consultation is recommended for prediction of short- and long-term prognosis and optimisation of medical therapy. In general, smoking cessation for at least 2 weeks prior to aneurysm repair can be beneficial and administration of pulmonary bronchodilators for at least 2 weeks prior to aneurysm repair is recommended for patients with a history of symptomatic COPD or abnormal pulmonary function studies.

A recent retrospective study conducted by Jonker[293] found that patients with AAA and chronic obstructive pulmonary disease had improved outcomes after EVAR compared to those undergoing open repair. In-hospital death and major complications occurred in 30% of patients after open repair compared with 12% after EVAR.

Renal protection strategies

Pre-operative renal dysfunction is a well-known determinant of early mortality after aneurysm repair.[263,294–296] The Lifeline registry of EVAR, publishing the results on 2664 EVAR patients collected under four multicenter Investigational Device Exemption (IDE) clinical trials in United States, found that renal failure was not an independent risk factor for aneurysm-related death (HR 1.775; 95% CI = 0.524 6.013, $p = 0.3569$), while it represented an independent predictor for all-cause mortality at 5 years (HR = 1.566, 95% CI = 1.062–2.311, $p = 0.0237$).[297]

According to the U.S. National Kidney Foundation guidelines, estimates of glomerular filtration rate (GFR) are the best overall indices of renal function.[298] Recent studies have described the higher prognostic value of glomerular filtration rate (GFR) compared with serum creatinine (SC) alone in patients undergoing endovascular aortic repair.[185]

EVAR is a procedure at increased risk for the development of renal complications, mostly related to administration of contrast agents (contrast-induced nephropathy, CIN), embolic debris dislodgement with catheters and wires, and potential early and late risk of arterial occlusion mainly attributable to graft impingements on the renal ostia or coverage by suprarenal bare stents.

Risk of CIN, defined as an increase of 25% of the baseline serum creatinine or an absolute increase of at least 0.5 mg/dL (44.2 μmol/L) of serum creatinine, occurring between 24 and 72 h after contrast administration, and not imputable to other factors, is quantifiable in 0.6–2.3% in the general population. It is more frequent in patients with pre-existent renal insufficiency with an estimated glomerular filtration rate (eGFR) ≤30 mL/min, diabetes mellitus, older age, reduced left ventricle systolic function, advanced heart failure, acute myocardial infarction, and shock. Volume and type of contrast medium, concomitant use of nephrotoxic medications, hypotension, dehydration, hypoalbuminemia, anemia, and the use of intra-aortic balloon pump represent the most occurring modifiable risk factors for CIN.

Volume supplementation remains the cornerstone for the prevention of CIN. Current evidence suggests that the combination of intravenous and oral volume supplementation effectively prevents CIN in low- and moderate-risk patients. Normal isotonic (0.9%) saline should be started 12 h before (or at least in the morning of) the contrast procedure with an infusion rate of 1 ml/kg of body weight per hour and be continued for 24 h. Level 2a, Recommendation A.

In addition, patients should be encouraged to drink plenty of fluids after a successfully completed EVAR. Addition of an antioxidant drug, the N-acetyl-cysteine (NAC), at an oral dose of 600–1200 mg b.i.d., has been shown to decrease the risk of CIN at least in high risk patients, although this has not been shown in patients undergoing EVAR specifically. In the meta-analysis by Kshirsagar *et al.*,[299] 16 randomised trials with a total of 1538 patients were included. The authors concluded that the heterogeneity of the current literature limits any meaningful conclusion on the benefit of NAC for CIN. In another meta-analysis by Isenbarger *et al.*,[300] seven studies selected from 19 were included, involving 805 study subjects. The odds of developing CIN were significantly lower in the NAC group (OR = 0.37; 95% CI 0.16–0.84), with a resulting number of needed-to-treat patients of nine. The REMEDIAL trial suggested that the strategy of volume supplementation by sodium bicarbonate plus NAC seems to be superior to the combination of normal saline with NAC alone or with the addition of ascorbic acid in preventing CIN in patients at medium to high risk.[301]

Use of non-ionic, low- or iso-osmolar contrast media are usually preferred in patients with pre-existing renal

insufficiency, while no evidence is supporting their preferential use in patients with normal renal function. A meta-analysis of prospective comparison trials found a nearly twofold higher incidence of CIN with high osmolar contrast media, but it has to be underlined that these studies did not routinely include prophylactic volume expansion or other pharmacologic prophylaxis.[302]

In the meta-analysis of Kelly *et al*. published in 2008,[303] fenoldopam, as ascorbic acid, prostaglandin I, dopamine, and theofilline, did not show any beneficial effect on the incidence of CIN. N-acetyl-cysteine reduced acute nephropathy with a relative risk of 0.66 (95% CI = 0.44—0.88), while furosemide increased it with a relative risk of 3.27 (95%CI, 1.48 to 7.26).

Direct intra-arterial fenoldopam infusion with specifically designed delivery systems may have the advantage of providing a higher local effective dose with potentially greater renal effects, while limiting systemic adverse effects due to renal first-pass elimination. These effects have been found to be beneficial in a prospective registry (Be-RITe!), where a reduction of 71% on the expected CIN in high risk patients was observed.[304]

Use of non-ionic, low- or iso-osmolar contrast media are to be preferred in patients with pre-existing renal insufficiency. Level 1b, Recommendation B.

Pre- and post-operative NAC administration for 3 days may be protective for those patients at high risk of developing CIN. Level 1b, Recommendation C.

Table 8 Minimal requirements for standard commercially available endografts.

Proximal aortic neck
Neck diameter >17 mm, < 32 mm
Angle between the suprarenal aorta and the juxtarenal aorta <60°
Angle between the juxtarenal aorta and the long axis of the aneurysm sac <60°—90°
Neck length >10 mm;
Neck thrombus covering <50% of the proximal neck circumference
Neck dilated <3 mm within 10 mm of the most caudal renal artery
Focal neck enlargement <3 mm within 15 mm from the most caudal renal artery
Neck calcification <50% of the proximal neck circumference
Aortic bifurcation
Aortic bifurcation diameter >20 mm in case of a bifurcated graft
Iliac artery
Iliac luminal diameter > 7 mm
Angle between the long axis of the aneurysm and the iliac axis <60°
Iliac calcification: non extensively circumferential
Iliac neck diameter <22 mm
Iliac neck length >15 mm

Morphological criteria

The increased use of EVAR has been affected by limitations of the related technology, although the percentage of AAA deemed suitable for EVAR has been growing over the past decade, due to improvements in graft design. However, long-term durability is still being questioned especially in case of adverse anatomy, rendering the pre-operative anatomical evaluation crucial for late success of EVAR. According to the instructions for use of the commercially available standard endografts, main anatomical characteristics and indications may vary according to graft model; minimal requirements are listed in Table 8.

Graft model choice

Appropriately sized aortic endograft should be selected on the basis of patient anatomy: according to the instruction for use of abdominal endografts, generally the device should be oversized 15—20% with respect to the aortic neck diameter to guarantee optimal seal. Level 2a, Recommendation A.

Several devices are available today to treat abdominal aneurysm, differing with respect to design, modularity, metallic composition and structure of the stent, thickness, porosity, methods of attaching the fabric to the stent and the presence or absence of an active method of fixing the device to the aortic wall. The overall performance among the current generations of aortic devices is quite similar and data appear to confirm low complication rate. An ideal stent graft incorporating all the advantages and no drawbacks is unreliable. Randomised trials comparing different devices would be challenging given the different anatomical requirements specific for each device.

Non-randomised comparisons of the results of different grafts have been published. At the Cleveland Clinic the authors reviewed different devices specific outcomes from their 6-year single series including 703 EVAR finding no differences in risk for aneurysm-related death, conversion, secondary intervention, migration, freedom from rupture, and Type I or III endoleaks.[305]

The European Registry Eurostar compared the outcomes of relatively new stent grafts (AneuRx, Excluder, Talent and Zenith) versus the earlier EVT/Ancure, Stentor (MinTec, La Ciotat, France) and Vanguard in 6787 patients. All new devices carried a lower risk of migration, kinking, occlusion and secondary intervention, conversion.[306]

A direct comparison between bifurcated versus aorto-uni-iliac (AUI) stent grafts may be very unreliable because it is recognised that AUI can be used to treat a large proportion of aneurysms, and are often used in older, unfit patients with larger aneurysms or in symptomatic or rupture settings. The RETA Registry reported alarmist unfavourable outcomes for the early outcomes in 263 AUI versus 733 bifurcated/tubular endografts implanted in UK centres. All in-hospital complications, reinterventions, conversions, and technical failure were significantly more frequent in the AUI group.[307]

A more recent attempt to compare results among different EVAR devices in patients enrolled in 2 randomised controlled trials on EVAR has been recently published. Two bifurcated devices, Talent and Zenith, implanted within the EVAR 1 and 2 trials were compared. Authors failed to find any convincing device-specific differences between AAA related outcomes.[308]

Type of anaesthesia

The commonest type of anaesthesia used for the intervention is general anaesthesia, chosen in 61% of the cases, followed by regional (34%) and local anaesthesia (8%).[309] In more recent publications, however, a clear preference for local anaesthesia has been underlined. Some authors reported that epidural anaesthesia is indeed feasible in a high percentage of patients in whom it is attempted; it ensures comparable outcomes to general anaesthesia and may be associated with shorter period of hospitalisation.[310]

Verhoeven *et al*.[311] suggested that a strategy based on the preferential use of local anaesthesia for EVAR, restricting regional anaesthesia or general anaesthesia only to those with predefined contraindications, is feasible and appears to be well tolerated. A more recent literature review[312] compared the impact of the type of anaesthesia (locoregional versus general anaesthesia) on the outcomes following EVAR. This review suggested that locoregional anaesthesia can improve post-operative outcomes following EVAR by reducing hospital stay, ICU stay, mortality, and morbidity. The retrospective analysis of 91 consecutive patients who underwent EVAR under local, epidural and general anaesthesia conducted by Bettex,[313] reported that local anaesthesia is a safe anaesthetic method for the endovascular repair of infra-renal abdominal aneurysm, offering several advantages such as simplicity, stable haemodynamics, and reduced consumption of intensive care resources, and hospital beds.

The preferential use of local anaesthesia for EVAR, restricting regional anaesthesia or general anaesthesia only to those with predefined contraindications, is feasible and appears to be well tolerated. Level 3b, Recommendation B.

Percutaneous access

Technology today available allows arteriotomy repair with a percutaneous suture device even after the use of large-bore introducers.[314–317]

Torsello *et al*. reported the first large, non-randomised series in order to assess the feasibility of percutaneous access, also after using sheaths up to 27F.[318] Subsequently, a German randomised study concluded that the success rate with the percutaneous technique ranged between 71.4% and 96%, depending on patient volume and selection. In the same study, cost analysis revealed no significant differences, with higher instrumentation costs with the percutaneous approach and longer mean operation time and hospital stay with the cutdown procedure.[319]

The main risk factors for failure of the closure device are represented by obesity, calcified femoral arteries, scarred groin, and kinking of both iliac arteries and aorta.[320–322] Analysing the results of percutaneous technique in a large single centre experience, Torsello *et al*. found a primary technical success of 96.1% in 500 consecutive patients. The need for early conversion correlated with femoral artery calcification (OR 74.5, 95% CI 17.8 to 310.7; $p < 0.001$) and operator experience (OR 43.2, 95% CI 9.8 to 189.0; $p < 0.001$). The risk of late complications was significantly higher in the presence of a groin scar (OR 48.8, 95% CI 9.2 to 259.0; $p < 0.001$), while sheath size and obesity played a minor role in influencing the results.[323]

Percutaneous approach for EVAR may provide a less invasive aortic access and can facilitate shorter hospital stay in selected patients. Level 3, Recommendation D.

Management of accessory renal arteries

Accessory renal arteries are frequently encountered when patients are evaluated for endovascular abdominal aortic aneurysm repair. Approximately 15%–30% of all adult patients have renal accessory arteries.[324] From the pre-operative CT angiogram, it can be possible to size the accessory renal artery and evaluate the amount of renal parenchyma dependent from this vessel. It is usually believed that preservation of accessory renal arteries should be taken into consideration for vessels > 3 mm in diameter, or supplying circulation to more than one-third of the kidney. Recently, some authors reported that occlusion of accessory renal arteries is not associated with clinically significant signs or symptoms, even in patients with mild or moderate renal insufficiency. Sacrifice of accessory renal arteries has not been found to lead to detectable renal infarction, either clinically or radiographically. Moreover, accessory renal arteries were not found to contribute to endoleaks even without prophylactic embolisation.[325,326]

Most often the occlusion of accessory renal arteries during EVAR is not associated with clinically significant signs or symptoms of renal infarct, does not contribute to any increase in endoleak rate and should not be embolised pre-operatively. Level 4, Recommendation C.

Management of concomitant iliac aneurysms

Dilation of one or both common iliac arteries (CIAs), making them unsuitable for adequate distal sealing and therefore compromising the success of endovascular repair and the feasibility of the procedure, may be present in up to 40% of EVAR patients.[327–330] Coil embolisation of hypogastric artery, followed by endograft extension into the external iliac artery (EIA), is usually performed to prevent type 2 endoleak. The sacrifice of hypogastric artery may rarely result in severe morbidity and mortality, caused by bowel or even spinal ischemia, particularly in the presence of bilateral hypogastric occlusion and/or concomitant atherosclerotic occlusive disease, while it does not definitely reduce the risk of Type 2 endoleak. Hypogastric embolisation is usually preferred over simple coverage of its ostium by the endograft to prevent the risk of Type 2 endoleak, but coils should be placed as proximal as possible to spare collateral circulation. Usually, the procedure is carried out as a single stage together with EVAR, since it was found to increase operative time without increasing significantly the operative risk.[331] Literature data show that approximately one-third of patients with hypogastric occlusion have symptoms of pelvic ischaemia: buttock claudication is fortunately the most common, occurring in about 80% of symptomatic patients, with impotence in about 10% and colonic ischaemia in 6–9% of all the pelvic ischaemic complications.[260,332–338] Bilateral hypogastric interruption, where incidence of ischaemic complications may increase, is to be avoided at least in standard risk patients. Fortunately, life threatening pelvic or intestinal ischaemia seem to occur very rarely, while a more severe and frequent buttock claudication and erectile dysfunction

can be expected in bilateral versus unilateral hypogastric occlusion.

Repair with a bifurcated iliac endograft, the iliac side branch device (IBD) has recently emerged as an alternative, flow-preserving, endovascular technique to address this problem. The use of an IBD in maintaining antegrade flow to at least one hypogastric artery for aortoiliac aneurysm repair was shown to be feasible and safe in some preliminary clinical series.[339–342]

Preservation of flow to at least one hypogastric artery is recommended in standard risk patients. Level 2c, Recommendation B.

Hypogastric embolisation is usually preferred over simple coverage of its ostium by the endograft to prevent the risk of Type 2 endoleak, but coils should be placed as proximal as possible to spare collateral circulation. Level 4, Recommendation C.

Pararenal aneurysms: fenestrated grafts

Short or diseased proximal aortic necks represent the major cause for precluding EVAR in AAA patients in up to 40% of the cases.[329,343]

Endografts with fenestrations, openings within the fabric to accommodate visceral arteries, have been introduced and are now commercially available in Europe. Preliminary single-centre experiences show promising results, at least in units with extensive experience in aortic and visceral vessels disease endovascular treatment. In a recent review, early mortality of the procedure resulted ranging between 0% and 8.5%, with a reintervention rate of 7.9–24%.[344] In the largest published single-centre series, from the Cleveland Clinic, 119 high-risk patients have been addressed with this technique with a resulting mortality of 1/119, and a renal occlusion rate of 10/231.[345] The results of multicentre trials in the USA and France are confirming these promising results.[346,347]

In case of more proximal aneurysms or thoracoabdominal aneurysms, the technique has evolved providing today branched grafts, where fenestrations are being substituted by short internal-external sidebranches or spiral external branches to provide a better seal between the aortic graft and the stent graft used for the visceral vessel.

In case of short or diseased neck the use of endografts with fenestrations shows promising results but should be performed with appropriate training and in centers with extensive experience in EVAR. Level 3, Recommendation C.

Post-operative patient management

The management of infra-renal AAAs has changed in the last decade with the introduction of endovascular techniques. EVAR is less invasive than open repair, and some of the reported advantages of EVAR are lower perioperative morbidity and mortality, shorter hospital stay, lower blood loss, and faster recovery.[116,117,348]

Post-operative analgesic treatment consists mainly of anti-inflammatory non-steroidal analgesic and/or intravenous boli of morphin. Patients in the recovery room can be transferred as soon as possible or by the end of the day to the regular ward, and are free to drink clear fluids. Regular diet as well as free ambulating are resumed on the first post-operative day. Patients at increased risk of a cardiac event following EVAR should be considered for electrocardiogram (ECG) monitoring and measurement of post-operative troponin levels, since troponin elevation is predictive of adverse outcomes.[349,350] Otherwise, troponin measurement is only recommended for patients with post-operative ECG changes, chest pain, or other signs of cardiovascular dysfunction. The comparison of early and intermediate results in patients suitable for open and endovascular technique proposed by Garcia-Madrid *et al.*[351] showed that patients in the open surgery group had a longer length of stay in the post-operative monitoring unit (median 17 h vs. 2 h in the EVAR group) and in the ward before discharge (median 6 days vs. 2 days).

Perioperative mortality and morbidity

Perioperative mortality of EVAR has decreased notably in recent years with the widespread adoption of new technologies.

In 2004 the first level I evidence for early outcomes of EVAR was provided by the results of the UK EVAR and the Dutch DREAM trials randomising patients with aneurysm greater than 5.5 cm or 5 cm in diameter to either open surgery or EVAR.[116,276] Both trials showed a 2.5-fold reduction in surgical 30-day mortality following EVAR: 4.6% vs. 1.2% in the open vs. endovascular group, respectively ($p = 0.10$) for the DREAM trial; 4.7% vs. 1.7%, in the open vs. EVAR group, respectively ($p = 0.009$) in the EVAR 1 trial. The last published RCT on EVAR patients, the OVER trial (Open Versus Endovascular Repair) from the Veterans' Affairs Cooperative Study Group showed a lower perioperative mortality rate at 0.5% in the EVAR group.[148] A recent metanalysis[352] concluded that according to RCTs, EVAR, compared to open repair, reduces operative mortality (OR 0.35; 95% CI: 0.19–0.63).

Higher perioperative mortality rates resulted from EVAR registries focusing on old devices not more in use today and early experience of the operators. Since its start in 1996, the European RETA registry reported in 2001 a 4.0% mortality within 30 days for the 389 EVAR performed with bifurcated or tubular devices in 31 vascular units.[353] The larger EUROSTAR,[354] showed a 2.3% mortality at 30 days over 4392 EVAR mainly based on commercially available devices performed up to 2002.

Non-randomised but controlled studies suggest a definite advantage of EVAR vs. open surgery in terms of perioperative mortality, with rates <2.0% in multicentre trials in the USA. There was a 1.7% 30-day mortality for 573 patients treated with the Guidant Ancure system[355] and 2% for 416 patients treated with the Aneurx device;[356] 1.0% 30-day mortality for 235 patients treated with the Gore Excluder device,[357] for 352 patients treated with Zenith device,[358,359] and for 192 patients treated with Powerlink devices;[360] and 0.8% mortality in 240 patients with Talent device.[361]

A recent Medicare population study using administrative data from 45,000 Medicare beneficiaries undergoing elective EVAR in the USA showed a 1.2% 30-day mortality with EVAR and 4.8% with open surgery (relative risk 0.25; 95% CI 0.22 to 0.29; $p < 0.001$).[118] The absolute advantage of EVAR vs. open repair increased with increasing age: from 2.1% absolute risk reduction at 67–69 years to 8.5% at 85 years or older.

Failure rate and perioperative morbidity

Perioperative adverse outcomes of EVAR include aneurysm rupture, technical failure, local vascular, device- or procedure-related complications and medical complications (myocardial infarction, pneumonia, acute renal failure, deep vein thrombosis, pulmonary embolism, colon ischemia, etc).

Technical failure is uncommon with last generation devices and increased experience in EVAR. A meta-analysis of 28,862 EVAR procedures performed before 2003 showed that primary conversion to open surgery was required in 3.8% of patients.[362] However, rates of complications decreased significantly over time (from 1992 to 2002) according to the meta-regression analysis. Immediate failures with primary conversion are reported in 1.8% of patients in all the 3 RCTs on EVAR[116,117,148] and in 1.6% of patients in the recent analysis of 45,000, propensity-score matched Medicare beneficiaries treated by EVAR from 2001 to 2004.

EVAR has the advantage of reduced median procedure times (2.9 h vs. 3.7 h), blood loss (200 vs. 1000 mL), transfusion requirement (0 units vs. 1.0 units), duration of mechanical ventilation (3.6 h vs. 5.0 h), hospital stay (3 days vs. 7 days) and intensive care unit stay (1 day vs. 4 days) when compared to open surgery. However substantial exposure to fluoroscopy (median 23.0 min vs. 0 min) and contrast (median 132.5 mL vs. 0 mL) is required.[148] In addition, EVAR may be associated with substantial 30-day intervention rate five times more often than open repair. Thirty-day reintervention rates after EVAR were 9.8% in EVAR 1 trial and 18% in EVAR 2 trial.[189] Reinterventions are often related to the presence of immediate endoleak: a condition unique to stentgrafts (persistence of blood flow outside of the lumen of the stent graft but within the aneurysm sac). Reported incidence of endoleaks within 30 days postoperatively may reach 40% in selected experiences.[363] Type I and III endoleaks are always considered clinically significant and should be treated as soon as they are diagnosed, as spontaneous resolution over time cannot be expected. In these cases the aneurysm sac is considered at high risk of rupture due to the continuous column of pressure between the aorta and the endograft and increased pressurisation of the aneurysm sac is likely. For Type II endoleaks detected at the time of EVAR, further treatment is not immediately indicated, since spontaneous resolution is possible.[364–367] Close imaging follow-up with CT scan is mandatory.

Due to the minimally invasive approach, elective EVAR procedures reveal reduced systemic complications. In a Medicare propensity matched analysis of EVAR vs. open surgery patients, all medical complications were 2% less likely after EVAR than after open repair.[365] The combined incidence of mortality and severe complications and that of mortality and moderate/severe complications at 30 days in EVAR patients were 4.7% and 18.1%, respectively in the DREAM trial.[276]

EVAR has been associated with a lower incidence of perioperative cardiac arrhythmias, myocardial ischaemia and cardiac events[368] with respect to open repair: 3.3% vs. 7.8% in the state-wide review of Anderson *et al.* on EVAR patients treated before 2002;[369] 7% vs. 9.4% myocardial infarction according to Medicare data.[335]

Colon ischaemia has been reported as occurring in as many as 1.4% of patients after EVAR;[370] however, this rate resulted lower than that after open repair, according to large Medicare beneficiaries data.[363]

Wald *et al.* showed that acute renal failure in the post-operative period was significantly better using EVAR than open repair (OR 0.42; 95% CI 0.33–0.53).[371] Administrative data also showed a reduction in the incidence of acute renal failure (5.5% vs. 10.9%) and need for dialysis (0.4% vs. 0.5%) among patients treated with EVAR.[365] Accurate surveillance of renal function in all the patients after EVAR is recommendable.

The potential of cytokine release after aneurysm sac thrombosis may be responsible for a 'post-implantation' syndrome, a rare phenomenon lasting up to 10 days after EVAR consisting in fever, malaise, back or abdominal pain with a transient rise in C-reactive protein (CRP) levels, leucocyte concentrations, and body temperature. Surveillance and aspirin are recommended in these cases.[372]

Local vascular or device-related complications may occur in 9%–16% after EVAR and have been found in 16% of patients enrolled in the DREAM trial.[276]

Most of these complications are due to groin and wound complication due to access injuries. Some are related to inefficiency or inexperience with closure devices systems. Ischemic limb complications may occur for limb occlusion or thrombosis especially when unsupported stent grafts are used in patients with aortoiliac disease, inappropriate stent graft oversizing, or small distal aorta. Distal embolisation using a lower-profile introducer system is now rare.

Chapter 6 – Management of Ruptured AAA

Open repair of ruptured AAA

Indications for open AAA repair

The incidence of ruptured abdominal aortic aneurysms ranges between 5.6 and 17.5 per 100,000 person-years in Western countries[373–375] and seems to have declined in the last decade. The number of aneurysm ruptures dropped from 18.7/100,000 (in 1994) to 13.6/100,000 (in 2003) in the USA.[376] The overall mortality rate of patients is still extremely high with an approximately 80–90%.[373,377,378] The operative mortality of ruptured aortic aneurysm has not improved significantly in recent years, with mortality rates still ranging from 32% to 80%.[379–385]

Definition of ruptured and symptomatic abdominal aortic aneurysms

AAA rupture is defined as bleeding outside the adventitia of a dilated aortic wall. Rupture is further classified into free rupture in the peritoneal cavity and retroperitoneal rupture where the retroperitoneal tissue provides tamponade and reduces temporarily the volume of blood loss. Differentiation between symptomatic and ruptured aneurysms is critical. Symptomatic AAAs are those that have become painful but without breach of the aortic wall. The inclusion of symptomatic AAAs in data on ruptured AAAs will artificially improve the results of outcome series.

Pre-operative evaluation

Since the screening of AAA has become more and more routine, the number of emergency surgeries has decreased in recent years.[376]

If a patient with known aortic aneurysm is admitted to the hospital with signs of shock and symptoms that might be linked to an aneurysm rupture, further diagnostic does not seem mandatory and the patient should be immediately transferred to the operating room. Depending on the hospital settings, emergency ultrasound scanning can be done to confirm the suspected diagnosis.

Lloyd *et al*. performed a time-to-death study in patients with ruptured AAA who did not undergo surgery for several reasons. The authors saw that the majority of patients (87.5%) survived more than 2 h after admission to the hospital, with a median time interval of about 11 h. The conclusion from these data is that most patients with a ruptured abdominal aortic aneurysm who reach the hospital alive are sufficiently stable to undergo computed tomography for further therapy setting.[386]

The timing of surgery for patients with symptomatic but unruptured aneurysms remains more controversial. An emergent open repair under less favourable circumstances includes a higher risk of perioperative complications.[387–389] Patients that may benefit from pre-operative preparation have to be identified. An individually adapted approach within 2 days might be beneficial for selected patients.[387–390]

Immediate repair is recommended in patients with documented aneurysm rupture. Level 1c, Recommendation A.

In symptomatic but unruptured AAA an optimisation of the patient and delayed repair of less than 48 h might be discussed. Level 3b, Recommendation C.

Perioperative management

Permissive hypotension

Against the initial idea of aggressive fluid resusciation in the management of haemorrhagic shock, there is considerable evidence that vigorous fluid replacement may exacerbate bleeding.[391–399] In 1991 Crawford published his experiences with 180 patients and found a survival benefit in those with hypotensive resuscitation with a target systolic blood pressure of 50–70 mm Hg and fluid restriction to allow clot formation and avoid the development of an iatrogenic coagulopathy.[400] In Hardman's review the correlation between hypotension at admission and mortality was investigated. The infusion of more than 3.5 L of fluid prior to surgery was associated with an increased relative risk of death by factor 3.54. Since the relative risk of death associated with blood pressure (per 10 mm Hg) was 0.91 it can be speculated that the volume of infused fluid has a more significant impact on the risk of death than systolic blood pressure.[401]

Van der Vliet *et al*. published the first series of patients with ruptured AAA in which a protocol of permissive hypotension has been adopted in daily routine using nitrates when indicated. The aim was to limit prehospital intravenous fluid administration to 500 mL and to maintain systolic blood pressure at a range of 50–100 mm Hg following admission. The desired systolic blood pressure range was reached in 46% of the cases whereas in 54%, a systolic blood pressure higher than 100 mm Hg was recorded for a period longer than 60 min.[402]

Currently, there is no prospective study on the effect of hypotensive resuscitation in patients with ruptured AAA available in humans. Surgery in case of aneurysm rupture needs to be performed in general endotracheal anaesthesia. The anaesthesiologist and the surgeon need to dovetail their acts, since vasodilation on induction will often lead to sudden hypotension with the need of rapid bleeding control through the surgeon.

Hypotensive resuscitation might have a beneficial effect on the survival in case of abdominal aortic aneurysm rupture. Systolic blood pressure should range between 50 and 100 mmHg depending on the patient's condition at admission. Level 4, Recommendation C.

Perioperative mortality and morbidity

Abdominal compartment syndrome

A compartment syndrome is defined as a 'condition in which increased tissue pressure in a confined anatomic space, causes decreased blood flow leading to ischemia and dysfunction' and 'may lead to permanent impairment of function'.[403] Though the abdominal compartment syndrome lacks a uniformly accepted definition, an abdominal pressure of more than 20 mm Hg in the presence of organ dysfunction is normally used to describe this critical state. It is observed in 10–55% of patients after emergent aneurysm repair.[404–406] Measurement of the intra-abdominal pressure can either be performed by urinary bladder pressure, which is the most frequently used technique, by gastric pressure or by invasive methods like catheterisation of the pressure in the vena cava.[407] The debate regarding the timing of and criteria for decompression is ongoing. The balance between effective tamponade of bleeding and the unfavourable physiological effects of compartment syndrome is delicate. In Meldrum's series, where decompression was performed at a bladder pressure of >20 mm Hg, the survival rate was 71%.[408] In past years the concept of temporary abdominal closure with impermeable mesh or Silastic sheeting of a vacuum-assisted closure were prompted. Rasmussen *et al*. saw in their case-control study that patients who needed mesh closure had a higher mortality rate than did the patients who underwent primary closure (56% vs 9%). However, the patients who underwent mesh closure at the initial operation had a lower mortality rate (51% vs 70%) and were less likely to develop a multi-organ failure (11% vs 70%) than the patients who underwent mesh closure after a second operation in the post-operative period for abdominal compartment syndrome.[409] The authors generated a list of predictors of poor outcome that warrant initial mesh closure in the initial operation for aneurysm rupture:

- Haemoglobin of less than 10 g
- Pre-operative cardiac arrest
- Systolic blood pressure of <90 mm Hg for more than 18 min
- More than 3.5 L of fluid resuscitation per hour at the operation
- Temperature less than 33°C
- Base deficit of greater than 13

Kimball *et al*. saw in a retrospective analysis of 122 patients with ruptured abdominal aortic aneurysm that preoperative hypotension, blood loss of at least 6 L, or intraoperative resuscitation with at least 12 L predicted mortality. They saw a statistically significant survival benefit in the first 24 h after surgery for patients who were

treated with a vacuum-pack technique (0% vs 21% standard primary closure), this could however not be reconfirmed after 30 days (32% vs 40%).[410]

Among the temporary abdominal closure techniques, the vacuum-assisted device delivers best results concerning time of definite abdominal closure rate, discharge from the immediate care unit and survival.[411,412]

An increased abdominal pressure serves as a negative predictive factor for the survival after open repair of a ruptured AAA. Measurement of the intra-abdominal pressure is recommended and in case of elevated levels (>20 mm Hg) in combination with organ dysfunction decompressive surgery should immediately be performed. Temporary abdominal closure systems can positively influence outcome. Level 2c, Recommendation A.

Endovascular repair of ruptured AAA

Patients who are critically ill with ruptured AAA (rAAA) could be the most likely to benefit from a less invasive procedure. However, over a decade since the feasibility was first demonstrated, EVAR of rAAA has not been widely adopted. The broad application of EVAR in rAAA settings encounters a number of barrier issues, notably aneurysm morphology, logistics, and stent graft requirement. Currently, there is no level evidence to support the widespread adoption of EVAR in an unselected population of patients who present with rAAA. A few population based studies are now supporting EVAR for rupture, although all overstate the effect due to selection bias.[200–202]

Pre-operative management

Feasibility

The anatomical suitability for EVAR of rAAA is commonly reported at 60% (range 18–83%);[413–421] the ongoing randomised Amsterdam Acute Aneurysm Trial has recently presented data on 83 enrolled patients with proven rAAA and showed that 46% were suitable for EVAR but only 35% were treated.[414] The wide range of feasibility quoted in the literature is a result of the different stent graft systems and anatomic criteria used. Many groups accept the same anatomic criteria of rAAA as in elective EVAR cases. However, more often, since the primary goal of treatment for rAAA is to save the patient's life, more liberal morphologic criteria have also been accepted, particularly in regard to the proximal seal zone length. The hypothesis is that the morbidity/mortality associated with immediate EVAR and eventually delayed conversion to OR after EVAR failure is better than that of OR as first option in emergency settings. With the newer stentgraft systems that generally use strong fixation modes, have a wide range of sizes, and can be accommodated in sharp angulations, a greater number of rAAAs will be suitable for EVAR.

Logistics

Implementing endovascular management of patients with rAAA is a complex process. Good logistics, adequate training of physician and staff and versatile stentgrafts are prerequisites for this type of treatment program. The organisation required to cover an EVAR service 24 h per day around the year with proficiency and equipment for emergency repair is one of the major drawbacks preventing the extensive dissemination of endovascular approach to rAAA.

Multidisciplinary algorithm and protocols

Paramount to the effective endovascular treatment of a rAAA is the development of a common set of rules that facilitates the synchronous passage of the patient through the emergency department and imaging service to the endovascular suite. The decision to proceed with emergent EVAR or invasive imaging studies, placement of an aortic occlusion balloon, use of local anaesthesia and criteria for feasibility are some of the most debated topics and they depend on the comfort level of the operative team and the condition of the patient.

The set-up of standardised protocols for endovascular treatment of rAAA including a multidisciplinary approach has been demonstrated successfully and should be employed.[422] Level 2c, Recommendation A.

Dedicated equipment, angiography suite and personnel

Unlike open repair, EVAR in emergency settings requires a dedicated and readily available multidisciplinary staff with trained experience as well as dedicated specific technology. An on-call endovascular team of vascular surgeons, radiologists, radiology technicians with experience in open and endovascular repair of rAAA as well as anaesthesiologists, transport personnel and operating room nurses, must be readily available at all times.

The hospital should have a dedicated endovascular suite in which open repair can also be performed. This can be provided with a mobile imaging unit or preferably a fixed fluoroscopic imaging unit in an operating room.

Equipment for EVAR and open repair should be present all the time. A 'rupture kit' for EVAR of rAAA should be maintained, with an inventory of preferred and most usable stent grafts components with which the treating surgeon has experience. Large-diameter main-body devices with short and long limb lengths should suffice in most emergent cases.[422] Level 4, Recommendation C.

Imaging

Although ultrasonography (US) can detect an aortic aneurysm, it is not a sensitive modality for the detection of rupture and has not been validated to assess aortic morphology feasibility for EVAR. Main reasons for which patients with rAAA may need pre-operative computed tomography (CT) examination before proceeding to EVAR are as follows.

Confirm true rupture

The results of emergent EVAR for any suspected rAAA may be different when applied to patients with unstable conditions, in those with aneurysm rupture but stable hemodynamic or in those with impending rupture, or symptomatic aneurysms in whom the proof of aortic wall integrity is not demonstrated. Since EVAR does not allow direct intraoperative inspection of aortic integrity, without the proof of rupture by pre-operative CT scan the prevalence and positive results of rAAA by EVAR can be overestimated.

Assess anatomical suitability

A pre-operative CT scan is generally suggested for all conscious patients in a haemodynamically stable condition.

The management is different for unstable or unconscious patients who generally mandate immediate repair. The presence of a multi-slice CT scanner in the emergency department can greatly facilitate rapid imaging and several current studies have shown that a CT scan can now be obtained in 10—15 min. It is generally accepted that the patient should remain stable during the anatomical imaging that is necessary prior to emergent repair.

CT scanning in patients with rAAA is not a totally benign intervention, particularly in critically ill patients: even with the most advanced technology, the diagnosis-to-CT scan delay is likely to be crucial if EVAR is to affect an improvement in survival from rAAA. The delay in completing and interpreting emergency CT scan remains one of the principal threats to improving the survival from rAAA by endovascular approach. The lack of broad prompt availability and good quality pre-operative CT scans in many community settings decreases the feasibility of emergent EVAR.[418]

Delay needed for imaging may not be the only disadvantage of being treated by EVAR using CT scan in emergency settings: patients with rAAA are relatively elderly and some will be in shock and hence at increased risk of contrast nephropathy. The risk is amplified by the twofold contrast exposure required for pre-operative CT and procedural angiography.

Some investigators have eliminated today the need of routine pre-operative CT scan as a prerequisite for all endovascular treatment in rAAA patients, especially when highly unstable, using in these cases the intra-operative angiogram for device selection.[423,424]With the availability of a large inventory of devices, size matching becomes less of an issue: the degree of diameter oversizing and device length may be effectively adjustable with the type of self-expanding modular stentgrafts.

EVAR should be considered as a treatment option for ruptured AAA, provided that anatomy is suitable, and the centre is appropriately equipped and the team experienced in emergency endovascular aneurysm procedures. Level 2b, Recommendation B.

Intraoperative management

Resuscitation

Approximately 25% of patients with rAAA will arrive in an hypotensive state. Fluid resuscitation should be restricted to an amount needed to maintain patient's consciousness and systolic blood pressure of 50—100 mm Hg (permissive hypotension). Experience has shown that systolic arterial pressures of 50—70 mm Hg are well tolerated for short periods and limit internal bleeding and its associated loss of platelets and clotting factors.[402,425—428] Resuscitation efforts should be preferentially managed with the use of blood products.

Whether or not pharmacological lowering of blood pressure is beneficial remains to be conclusively shown.

Aortic occlusion balloon

The placement of an aortic occlusive balloon during EVAR for rAAA can be used to control hemodynamic instability from ongoing blood loss.[429] The use should be limited only when there is severe circulatory collapse. Aortic balloon occlusion poses risks of renal and splanchnic ischaemia, distal embolisation and do not prevent bleeding from iliofemoral arteries and can adversely impact the angiogram quality. Occlusive balloons can be placed via femoral or brachial access. Level 4, Recommendation C.

Anaesthesia

Use of local anaesthesia has been advocated to prevent circulatory collapse caused by the induction of general anaesthesia and to promote peritoneal tamponade. The loss of abdominal wall muscle tone and compensated sympathetic activation during the induction of general anaesthesia can in fact promote ongoing blood loss.[430] Whether general anaesthesia is used to eliminate motion and improve fluoroscopic imaging to permit precise graft deployment remains controversial. As an alternative, local anaesthesia supplemented by sedation can be used. Level 4, Recommendation C.

Stent graft system

Both uniliac and bi-iliac device configurations have been successfully used in EVAR for rAAA, without any evidence of significant superiority of one over the other. Aortouniliac (AUI) stent grafts have the advantages of allowing expeditious introduction and deployment, and rapidly controlling bleeding by decreasing the intra-aneurysmal pressure. These stent grafts may also offer broader applicability by requiring only favourable unilateral iliac anatomy and allowing exclusion of contralateral iliac aneurysms. However, a femoro-femoral crossover bypass graft is required with AUI stent grafts preventing the use of local anaesthesia, increasing the rate of wound infections and the risk for graft occlusion.[431] In addition, with the new available devices, if difficulty is encountered with contralateral limb deployment a bi-iliac stent graft can be easily converted to an AUI device with the placement of a converter across the flow divider. It cannot be overemphasised that the devices used for rAAAs should be systems that the operator routinely uses for elective EVAR and with which he or she has significant experience.

Pre-operative fluid administration should be restricted to a minimum to maintain hypotensive haemostasis. Level 2b, Recommendation A.

Patients who are unconscious or in whom a systolic blood pressure cannot be maintained should be immediately transferred to the operating room. The decision to proceed with emergency open repair, placement of an aortic occlusion balloon or invasive imaging studies should depend on the comfort level of the surgeon and conditions of the patient. Level 4, Recommendation C.

Perioperative mortality and morbidity

Mortality

Mortality rates lower than open repair have been observed with EVAR for rAAA ranging from 18% to 53% with several studies reporting a mortality rate of 20% or less.[432—434] Unfortunately, the studies are based on very small sample size and selected populations of patients. Patient selection may be one important reason for the variation in outcomes that have been published. Another factor might be the

differences in operative technique and experience. Also, the open surgery group for rAAA is likely to contain more complex cases, such as those with pararenal diseases and more unstable patients, unfit for imaging delay. This may be misleading when comparing outcomes of EVAR vs. OR for rAAA. To date no results from a complete RCT comparing open to EVAR for rAAA are available. The only RCT that so far has been published[435] was suspended after randomizing only 32 of the 103 admitted patients because of logistical problems therefore providing inconclusive results. The trial concluded that there was no superiority of one technique over the other and 30-day mortality was similar after OR and EVAR (53% on an intention-to-treat basis). Moderate or severe operative complications occurred in 77% in the EVAR group and in 80% in the OR group. Blood loss, ICU stay and hospital stay were significantly reduced. The Swedvasc registry[436] reported 1132 AAA repairs during 2006 from 33 hospitals, 16 of which performed EVAR. Out of 84 acute aneurysm repairs, 56 were performed with EVAR, but only 37 were true rAAA. Overall 30-day mortality was 11% among the overall 56 acute cases and was 18% in EVAR vs. 23% in OR. In patients in shock, 30-day mortality raised to 29% after EVAR and 46% after OR.

Morbidity

Although technical success rates of 96—100% can be obtained, emergent EVAR raises also the risk of a number of complications.

Abdominal compartment syndrome

Abdominal compartment syndrome (ACS) has been described in as many as 20% of patients undergoing EVAR for rAAA and is a major cause of mortality. It is advantageous to keep a high index of suspicion for this entity. Avoidance of systemic heparinisation to decrease the ongoing bleeding from collateral vessels can be useful. If one or more factors associated with development of ACS (need for an aortic balloon, presence of severe coagulopathy, massive transfusion requirements, conversion of a bifurcated stent graft to aortouniliac) an on-table laparotomy may be warranted to alleviate the hypotension, improve ventilatory compliance and oliguria.[437]

In addition to routine physiologic monitoring, patients who have undergone EVAR for rAAA should have hourly bladder pressures recorded to help in the early diagnosis of ACS.[406] Level 3, Recommendation B.

End-organ ischaemia

End-organ ischaemia (visceral, spinal cord, renal) are among the most feared complications after emergency EVAR; they are often caused by embolisation or ischaemia/reperfusion after placement of an aortic occlusion balloon. Spinal cord ischemia has been observed in as many as 11.5% of patients undergoing EVAR for rAAA,[438] hypogastric artery occlusion and prolonged functional aortic occlusion being the major causative factors. In addition, the use of contrast medium either for pre-operative CT scan or for intra-operative procedure, is associated with a risk of renal failure augmented by hypoperfusion, hypotension and embolisation.

Endoleak

The development of Type I endoleak has been observed in 5—25% of patients.[414,427,435,439,440]

This range may be the result of the different anatomic criteria in determining patient eligibility for EVAR. The higher rates of Type I endoleak support the use of more stringent anatomic criteria for EVAR in rAAA. Type I endoleaks are considered unacceptable since they do not allow the EVAR repair to prevent rupture. The development of late endoleak after EVAR for rAAA should also be investigated but data on durability are lacking.

Chapter 7 — Follow-up after AAA Repair

Follow-up after open AAA repair

The true benefit of AAA repair depends on its impact on the patient's long-term survival, but most reports have focused almost exclusively on the early post-operative period. Late survival and freedom from complications such as rupture, recurrent aneurysm formation, graft infection, aortoenteric fistula, graft migration should be considered as an index of durability and long-term success of the open or endovascular procedures. This chapter refers to the survival of patients and management of late complications occurring after AAA repair.

Long-term management after open surgery

Survival and functional outcome

Five-year survival rates after non-ruptured abdominal aortic aneurysm (AAA) repair range from 60% to 75% compared with approximately 80% in the age- and gender-matched general population.[242,441—446] Overall, survival after AAA repair is reduced compared with that of a matched population because of greater associated comorbidity in patients with AAA.[447—449] The main causes of late death after AAA repair are cardiac disease (44%), cancer (15%), rupture of another aneurysm (11%), and stroke (9%).[441,442,449] A series of 263 consecutive patients with AAA who had systematic coronary angiograms has shown that the presence of an AAA was an indicator of coronary disease.[450] But so far there have been no randomised studies to ascertain the value of prophylactic coronary artery bypass for enhancing life expectancy after AAA repair.

All patients treated for an AAA should receive the best medical treatment including aspirin, statins, an ACE-inhibitor and β-blockers if tolerated. Level 2a, Recommendation B.

Stroke is another factor which is contributing to an increased mortality among patients with AAA, not only because of the coexistence of carotid disease but also because of the increased prevalence of hypertension among the patients with AAA.[441,451]

Para-anastomotic aneurysm

Para-anastomotic aneurysms after AAA repair include false aneurysms resulting from a disruption of the anastomosis and true aneurysms that develop adjacent to the anastomosis. The aetiology of para-anastomotic aneurysm is multifactorial. The break of a suture, the type of prosthetic material, degeneration of the artery and infection should be suspected in all patients with pseudoaneurysms. Szilagyi analysed a 15-year experience with open aortic repair in

which anastomoses in the femoral region were at highest risk (3%), followed by the iliac (1.2%) and infra-renal aorta (0.2%).[452] But this study done prior to CT imaging may have missed many of the intra-abdominal para-anastomotic aneurysms. In another study, Edwards *et al*.[453] have systematically followed patients after aortic surgery with serial duplex-scan and have reported an incidence of para-anastomotic aortic aneurysm of 10% at 10-year follow-up. Ylonen *et al*.[454] confirmed also that after 10 years, 20% of patients may have an anastomotic femoral pseudoaneurysm. There are no studies on the natural history of para-anastomotic aneurysms but because of the risk of rupture,[453] elective repair should be carried out on large para-anastomotic aneurysms. Redo surgery using a transperitoneal or a retroperitoneal approach can be challenging, and stent grafting when anatomically possible is the preferred approach.[455,456] Redo femoral surgery is done using an interposition graft.

As para-anastomotic aortic aneurysm is not accessible to clinical examination, Post-operative surveillance protocols, including use of colour duplex ultrasound or CT imaging is recommended at regular intervals after open AAA repair (at 5 years, 10 years, 15 years). Level 3b, Recommendation B

Natural history of common iliac artery after open AAA repair and tube graft insertion

Aortoaortic grafts have long been advocated instead of aortobiiliac grafts for surgical repair of AAA. Yet preferential use of tube grafts or bifurcated grafts remains controversial. Proponents of bifurcated grafts point out that these grafts prevent subsequent aneurysmal change of the common iliac arteries (CIA). In a retrospective study of 438 patients, Huang *et al*.[457] have shown that the expansion rate of an iliac artery aneurysm was 0.29 cm/year. These results along with the fact that no iliac aneurysm under 3.8 cm ruptured after a mean follow-up of 3.7 years provide some useful information. In a prospective multicenter study, Hassen-Khodja *et al*.[458] have shown that with reasonably long follow-up (4.8 years), no patient with a CIA less than 25 mm in diameter at the time of initial surgery will require repeat procedure for subsequent aneurysmal dilatation below an aortic tube graft. Indeed, most CIA do not expand much after tube graft insertion. This was confirmed by Ballota *et al*.[459] who showed no rupture or significant progression of CIA >25 mm in a prospective study of 201 patients receiving a tube graft and followed 7.1 years.

Tube graft placement during AAA surgery is justified even for moderate common iliac artery dilatation <25 mm. Common iliac arteries with a pre-operative diameter $\geq$ 25 mm warrant insertion of a bifurcated graft during AAA repair. Level 2b, Recommendation B

Graft infection

The reported incidence of prosthetic graft infection varies between 0.3% and 6%.[460–463] A frequency influenced by the anatomical location of the involved prosthesis but all grafts are at risk of infection either at implantation or later by haematogenous seeding during endoscopic procedures with biopsy and dental procedures. Intra-abdominal aortoaortic or aortoiliac bypass grafts mostly used to treat an aortoiliac aneurysm develop infection in less than 1% of cases.[464,465] In contrast the presence of prosthetic material in the groin increases the rate of infection to 2–4%[465] Other predisposing factors include surgical revision and emergency surgery. The diagnosis of vascular graft infection can be challenging for intracavitary grafts and for infections caused by low-virulence organisms. Presentations can be quite diverse including generalised sepsis, groin purulence, pseudoaneurysm formation.[466] Staphylococcal organisms are the most frequent bacterial isolates, with *S. epidermidis* emerging as the most common organism recovered from infected prosthetic grafts followed by *S. aureus* and *E. coli*.[467]

Potential risk of late infection by hematogenous seeding makes antibiotic prophylaxis recommended for patients with a prosthetic graft prior to endoscopy with biopsy and dental procedures. Level 4, Recommendation C.

Infections associated with prosthetic-enteric fistula (PEF) should be considered differently because they represent an initial mechanical problem followed by contamination of the exposed prosthesis. PEF are rare, less than 1%.[466–468] In a systematic review of the literature, Berqvist *et al*.,[469] identified 1135 cases from papers on complications having PEF. Although the duodenum was most frequently affected, all parts of small and large bowel have been implicated.[466–470] The development of PEF can occur at any time after primary surgery. Bleeding is the dominant symptom with herald bleeding in half of the patients and generalised sepsis in about 25% of those. Bleeding is more common when the anastomosis erodes into the GI tract, while sepsis and abscess formation may be more common with paraprosthetic fistula involving the body of the graft. Diagnostic delay is typical. The diagnosis of PEF is one of exclusion and is occasionally confirmed by endoscopy or CT scanning.[471,472] Normal findings on endoscopy do not exclude AEF and a sensitivity of 50% has been reported.[472]

Any gastrointestinal bleeding in a patient having an aortic graft should prompt the evaluation of a prosthetic-enteric fistula. Level 1c, Recommendation B.

Computed tomography (CT) usually provides the most information about the nature of the problem, extent of infection, and other associated abnormalities. CT has a sensitivy and a specificity of 90–100% when done for advanced graft infection.[473–475] But with low grade graft infection, CT sensitivity and specificity drop to 65%.[476] Magnetic resonance imaging (MRI) can differentiate fluid and inflammation from haematoma, which CT scanning cannot. Recent studies have suggested the interest of the combination of fluorodeoxyglucose-positron emission tomography (FDG-PET) and CT scanning.[477]

Critical issue: There is a need to search for functional tests that could provide assessment of graft infection.

Treatment traditionally includes excision of all infected graft material with extra-anatomic reconstruction, particularly in the presence of extensive contamination, but several recent advances prompted a reassessment of these principles. First, in many cases, the causative organism in vascular graft infection shifted from the high-virulence *S. aureus* to the low-virulence *S. epidermidis*. Second, reports emerged of the successful treatment of infection without complete graft removal. Third, cryopreservation techniques allowed the use of *in situ* preserved aortic tissue, and *in situ* autogenous venous

conduits are being used in this setting. Fourth, prostheses impregnated with antimicrobial agents became available.

With these remarks in mind, the following observations should be made. Extra-anatomical bypass followed by infected graft removal remains the procedure with the largest experience. But mortality (11–44%), graft reinfection (3–37%) and aortic stump disruption (3–24%) remain high. Only a few papers with in-situ prosthetic reconstruction using antibiotic or silver bonded grafts have been published, all with low mortality and low amputation rates but with a high risk of new graft reinfection.[478–480] Considering series of *in situ* aortic reconstruction with an aortic allograft, mortality rates are ranging from 9% to 56% with a low amputation rate but with a significant risk of late stenosis or aneurismal dilatation of the allograft and a risk of disruption of the aortic anastomosis appearing in almost every report.[481–487] Finally, *in situ* aortic reconstruction with autogenous superficial femoral vein, first described by Clagett *et al.*,[488] and Nevelsteen *et al.*, [489,490] is the more recent technique used for aortic graft infection with a mortality between 7% and 32%, a low rate of recurrent infection and variable rates of venous morbidity. But most recent series restrict the use of this technique to stable patients with less virulent organisms and without enteric fistula[490,491] making comparison difficult. On the other hand, recent reports have shown that unstable patients with generalised sepsis and bleeding could benefit from expeditious procedure to control bleeding, including a 'temporary' stent graft as a bridge before a more definitive procedure.[492]

In a recent meta-analysis of the reported outcomes comparing these four techniques for the management of aortic graft infection, O'Connor *et al.*[493] concluded that extra-anatomical bypass followed by infected graft removal[494] had the highest rate of adverse event followed by *in situ* autogenous vein, *in situ* cryopreserved allografts and *in situ* antibiotic-bonded prosthetic grafts.[483,487,495,496] This conclusion should be interpreted with caution. First, many of these studies are retrospective with variable data reporting. Second, there is no clear outcome endpoint. Third, the authors have included series with primary aortic infection as well as infected grafts with a global outcome and not according to the pathology. In these series, the most advanced grafts infections, and those caused by the most virulent organisms are generally treated by an extraanatomical bypass followed by complete graft excision.

Unstable patients might benefit from expeditious procedures to control bleeding, including 'temporary' stent graft. Level 4, Recommendation C.

Stable patients with infection caused by high virulence organisms with enteric fistula should receive a staged procedure with extraanatomical revascularisation first, followed by graft excision, debridement of the infected field, aortic stump closure with an omental flap and closure or diversion of the gastrointestinal tract. Level 2c, Recommendation B.

In situ revascularisation using autogenous superficial femoral vein or aortoiliac allograft should be used in patients without enteric fistula. Level 2c, Recommendation C.

Antibiotic-bonded prosthetic *in situ* reconstruction should only be favoured in selected patients with limited contamination. Level 2c, Recommendation C.

Critical issue: There is a need to search for infection-resistant aortic prostheses.

Limb occlusion

One of the advantage of open AAA repair is its durability. Hallett *et al.*[497] reviewed 307 patients who underwent open repair with a cumulative 10-year incidence of 3% of graft thrombosis. Biancari[266] reported at a median follow-up of 8 years, graft limb occlusion in 5.3% of patients but with a large number of aortofemoral grafts (49%) in this series. Conrad *et al.*[265] reported from a series of 152 open AAA repairs under post-operative surveillance by CT scanning for graft limb occlusion (2.6%) at 7 years. In this series only 12% of the patients received a graft extending to the femoral artery. Stenotic limbs can be successfully treated by stenting. Treatment of an occluded limb includes thrombectomy or lytic therapy with secondary endovascular or surgical intervention.

Follow-up of patients after open AAA surgery should include, in addition to clinical examination, a colour duplex ultrasound with ABI on a regular basis. Level 2a, Recommendation B.

Impaired sexual function

Retrograde ejaculation and impotence may result after AAA repair due to injury of autonomic nerves during aortoiliac dissection.[498] In the ADAM trial, 40% of men had impotence before AAA repair[499] and less than 10% developed new impotence in the first year after AAA repair. But the proportion reporting new impotence increased over time such that by 4 years after AAA repair, more than 60% reported having impotence, which underscore the multifactorial aetiology of impotence in this age group. Careful preservation of the nerves along the left side of the aorta and cross the left common iliac artery has been shown to reduce this complication.[500] Other causes of post-operative impotence include reduction of pelvic blood flow due to internal iliac occlusion or embolisation.

In patients with AAA, aortoiliac reconstructions should be performed using a nerve-sparing technique, with preservation or improvement of pelvic blood supply. Level 2b, Recommendation C.

Long-term complications related to the incision

Like any intra-abdominal operation, open AAA repair is also associated with a risk for incisional hernia and adhesive intestinal obstruction. In an observational study of 45,660 Medicare beneficiaries comparing EVAR and open AAA surgery with propensity-score methods, Schermerhorn *et al.*[118] found that the incidence of laparotomy-related complications requiring intervention within 4 years was significantly higher after open repair with lysis of adhesions (1.5% vs. 0.5%, $p < 0.001$), and repair of abdominal incisional hernia (5.8% vs. 1.1%, $p < 0.001$), a finding that appears to be significantly more common after open treatment of AAA than aortic occlusive disease with a 2.8-fold increased risk of incisional hernia ($p < 0.001$).[501] Retroperitoneal incisions for AAA repair have also been

associated with weakened lateral abdominal wall musculature and a bulge in a significant number of patients.[502] Surgical exposure of the femoral arteries is uncommon for open AAA repair, but the incidence of post-operative seroma and femoral nerve injury are well documented in these patients when a bifurcated aortofemoral graft is needed.

Critical issue: Patients with AAA appear to have a significant of risk for both inguinal and incisional hernia compared to patients with peripheral aortic occlusive disease. A large prospective multicentre study is needed to confirm this.

Follow-up after endovascular AAA repair

Randomised trials[276,503] have shown reductions in peri-operative mortality and morbidity with endovascular repair of abdominal aortic aneurysm (EVAR) as compared with open repair (OR). Long-term survival rates however were similar for the two procedures with clinically significant complications occurring more frequently after EVAR, including certain procedure specific complications, such as endoleaks requiring lifelong careful follow-up. A significant number of new complications and subsequent reinterventions continue to be reported up to 8 years after the original EVAR procedure.[119,504]

Overall survival and long-term outcomes after EVAR

There is no controversy concerning the short-term benefit of EVAR as compared to open AAA repair, but there is concern that the long-term outcome may be less favourable. In the EVAR 1 trial,[503] a lower aneurysm-related mortality rate after EVAR did appear to be maintained at 4-year follow-up (4% in the EVAR group versus 7% in the OR group), but in terms of overall mortality this was cancelled out by excess mortality from other causes at around 28% in both groups. Comparable results were found in the DREAM trial,[276] with lower aneurysm-related deaths at 2 years in the EVAR group (2.1% vs. 5.7%) but comparable survival for OR (89.6%) and EVAR (89.7%) groups. Aneurysm-related mortality is a concept created to measure the efficacy of aneurysm repair in preventing death from aneurysm rupture from a population-based and health economy perspective. The entire 3% mortality difference in aneurysm-related deaths between OR and EVAR is generated in the first 30 post-operative days, as any death in that period is 'aneurysm-related' by definition. In the EVAR and DREAM trials, the overall survival curves appeared to converge in the second year after randomisation. But as in this patient population, the reported 5-year mortality rates are 30% or higher,[505–507] the first-years benefits can be considered as highly relevant even if not maintained in the period thereafter. In this setting, the EVAR 1 and DREAM trials showed a significant improvement in the quality of life after EVAR during the first 3 months following the procedure, but this difference disappears thereafter. Schermerhorn *et al.*[118] in a propensity analysis of Medicare beneficiaries undergoing OR and EVAR compared 22,830 matched patients in each cohort and found that late survival was similar in the two cohorts although the survival curves did not converge until after 3 years, and the survival advantage was more durable among older patients. By 4 years, rupture was also more likely to occur in the EVAR group than in the OR group (1.8% vs. 0.5%, $p < 0.001$) as was intervention related to AAA (9.0% vs. 1.7%, $p < 0.001$), including both major reinterventions (e.g., open repair with in-line or extraanatomical bypass, conversion to open repair, or repair of an infected graft), 1.6% vs. 0.6%, $p < 0.001$ and minor reinterventions (7.8% vs. 1.3%, $p < 0.001$).

All patients receiving an aortic stentgraft should be kept on the best medical treatment including statins (with aspirin, ACE-inhibitor or β-blockers if considered appropriate) for secondary prevention of cardiovascular disease. Level 2a, Recommendation B.

Endoleak

In 1997, White *et al.*[514] proposed the term 'endoleak' to describe 'persistent blood flow within the aneurysm sac but outside the stent graft'. They differentiated early or primary endoleak, observed during the first 30 days after EVAR, and late or secondary endoleak, developing later during follow-up. Schlösser *et al.*[508] have shown the role of endoleaks as the main cause of rupture in 160 of 235 patients. Endoleak Type I caused rupture in 88, endoleak Type II in 23, endoleak Type III in 26, and endotension in 9. In this analysis of AAA ruptures following EVAR collected from the MEDLINE and Embase databases, endoleak type was not specified in 14 of the patients with rupture due to endoleak.

Endoleak is frequent after EVAR and has been reported in nearly one in four patients at some time during follow-up.[509–511] It is one of the most common abnormalities identified on late imaging and used to justify lifelong follow-up of these patients. The most frequent endoleaks are Type 2 endoleaks perfused by aortic branches. Most frequently, they connect an inflow source with an outflow vessel, thus limiting the increase of sac pressure. When an outflow path does not exist, the net effect is a higher mean pressure in the sac with a potential risk for complications.[512]

Further categorisation of endoleak requires information regarding the course of the blood flow into the aneurysmal sac. Four types of endoleak (Table 9) have been described.[513,514,533]

Type I endoleak is indicative of a persistence perigraft channel of blood flow caused by inadequate seal at the proximal (Type IA) or distal (Type IB) end of the stent graft. A Type I endoleak may also refer to inadequate seal of an iliac occluder (Type IC). Incidence of Type I endoleak increases with difficult anatomical situations, such as short or angulated necks, and landing zones with calcifications. Type I endoleak is associated with significant pressure elevation in the sac and has been linked to a continued risk of rupture. Analysis of 4291 patients enrolled in the Eurostar registry in 2002, showed that Type I and Type III endoleaks with structural disintegration of stentgrafts were the most commonly documented findings at the time of rupture.[515]

The development of a proximal Type I endoleak during follow-up is evidence either of the inadequacy of fixation or dilatation of the neck of the AAA. From a subset of EVAR 1 trial patients, increase in aortic neck size was

Table 9 Classification for endoleaks and endotension.

Endoleaks (Type)	Source of perigraft flow
I	Attachment site
A	Proximal end of the stentgraft
B	Distal end of the stentgraft
C	Iliac occluder
II	Branch leaks without attachment site leaks
A	Simple: one patent branch
B	Complex: two or more patent branches
III	Stentgraft defect
A	Junctional leak or modular disconnect
B	Fabric holes
IV	Stentgraft fabric porosity <30 days after placement
Endoleaks (Time of detection)	Primary, present from time of EVAR Secondary, appearing after prior negative CTA[a]
Endotension	AAA enlargement with increased intrasac pressure after EVAR without visualised endoleak on delayed contrast CTA.

From White *et al*.[514], Chaikof *et al*.[533], Veith *et al*.[513]
[a] CTA: Computed tomographic scan with delayed imaging.

much greater after EVAR than after OR.[516] When no migration is depicted, Type I endoleaks can be treated by balloon dilatation or deployment of a palmaz stent.[517] But if migration of the stentgraft occurred, this is unlikely to be efficacious and the choice is between conversion to open repair especially in patients with large aneurysms fit for open surgery[518] or deployment of a proximal cuff or a fenestrated stent graft across the renal arteries.

On occasion, some Type I endoleak may seal spontaneously by the time of the first post-operative surveillance study. But even if sealing has occurred, Type I endoleak may have serious consequence because systemic pressure can be transmitted through clot. This explains why coil embolisation for Type I or Type II endoleaks may be ineffective to prevent rupture.[513]

Management of secondary distal Type I endoleak is generally more simple. In most cases, it is sufficient to extend the stentgraft limbs into the distal common or external iliac artery. When extending into the external iliac artery, consideration should be given to embolisation of the proximal internal iliac artery trunk to prevent back bleeding into the aneurysmal sac. If the contralateral internal iliac artery is occluded, it may be advisable to use a branched stent graft to secure blood flow at least in one internal iliac artery. Secondary distal Type I endoleak can also be in relation with the shrinkage of the aneurysm sac, creating upward forces pulling the distal iliac limb into the aneurysm sac generating sac pressurisation and potential rupture. It is recommended, to avoid this complication, that the iliac limb be extended at least 3 cm into the common iliac artery.

All Type I endoleaks should be treated. Level 2b, Recommendation B.

Type II endoleak is attributed to retrograde flow from the inferior mesenteric artery (IMA) (IIa), lumbar arteries (IIb), or other collateral vessels of the aneurysm sac. Origin and outflow sources of any Type II endoleak should be specified to avoid any confusion with Type I endoleak, but detection of a Type II endoleak may be difficult because these endoleaks are often associated with low flow. Side branch reperfusion is observed on post-operative imaging in 20% of patients.[519,520] Between 50% and 80% of such leaks resolve spontaneously within the first six months after operation and no treatment is indicated at this time,[365,367,521,522] but a minority persists or are delayed and these may cause concern. Type II endoleaks although often benign and associated with aneurysm stability or sac shrinkage, an indication of low pressure in the aneurysmal sac,[513] can also lead to increased sac diameter with intrasac pressure in the systemic range and a risk of rupture.

Treatment of these Type II endoleaks associated with sac enlargement is recommended.[520,523,524] A variety of methods have been proposed to abolish side branch reperfusion. Coil embolisation by transarterial superselective catheterisation of the branches through the superior gluteal artery or superior mesenteric artery or by translumbar routes is the less invasive option.[365] More recent techniques involve entering the aneurysm sac with a microcatheter and embolisation of both the feeding and draining vessels. Additional coils are also deployed within the sac itself to prevent recurrence.[525] Mansueto *et al*.[526] have shown promising results using transcatheter transcaval embolisation. CT scan guided translumbar approach has also been reported.[527] Should embolisation fail, laparoscopic retroperitoneal clipping of the side branches is possible but require advanced laparoscopic experience.[528] Laparotomy with ligation of the feeding side branches,

laparotomy and suturing of the side branch ostia within the aneurysmal sac, but leaving the stentgraft intact and finally conversion to open repair are other alternatives. Some endoleaks could not be detected with even optimal CT scanning, but MRI with a blood pool contrast agent can improve visualisation of Type II and Type IV endoleaks.[529,530]

CT scans with delayed arterial phase are the preferred method to detect type 2 endoleaks. Level 2a, Recommendation B.

Critical issue: Some endoleaks could not be detected with even optimal CT scanning. New techniques concerning visualisation of endoleak, including MRI with a blood pool contrast agent, should be developed.

Type II endoleaks without increased sac diameter can be observed. Level 2b, Recommendation B.

Endovascular or laparoscopic treatment is recommended for Type II endoleaks with increased sac diameter $\geq$10 mm, with conversion to open surgery in case of failure. Level 2b, Recommendation B.

Type III endoleak is caused by component disconnection (IIIa), or fabric disruption (IIIb). Modular disconnection is usually related to insufficient overlap between the stent graft components. It can be treated by endovascular deployment of a covered stent to bridge the gap between the two components that have separated. It should be noted however that disconnection often occurs as a result of migration and angulation of the stent graft. In this case, consideration should be given to conversion to OR.

Treatment is recommended for Type III endoleaks. Level 2b, Recommendation B.

Type IV endoleak is caused by blood flow through an intact but porous fabric and observed during the first 30 days after graft implantation. This definition is not applicable to fabric-related endoleaks observed after the first 30-day period (Type IIIb endoleaks).

If an endoleak is visualised in imaging studies but the precise source cannot be determined, the endoleak should be categorised as of undefined origin.

Treatment is not recommended for Type IV endoleaks. Level 2b, Recommendation B.

Endotension: The term endotension was intended to describe 'sufficient pressure to cause rupture'[531] It is recognised that an AAA can enlarge after EVAR, even in the absence of a detectable endoleak and that may lead to rupture. The problem is that we do not know how much pressure is necessary to cause rupture and if continuous pressure is less hazardous or as hazardous as pressure that varies throughout the cardiac cycle.[532] An additional consideration is the presence or absence of an endoleak. The AAA sac may be pressurised via a low flow endoleak or indirectly via a clot (virtual endoleak), this explains why some AAA enlarge even when no endoleak can be detected and why endotension may occur without an endoleak.[531] In addition, endotension may also be caused by a real endoleak which cannot be visualised with current imaging techniques.

Expansion of an aneurysm is evidence that the pressure within the sac is greater than in the surrounding tissues. Measurement of pressure within the aneurysmal sac, either by translumbar puncture of the sac or by passing a catheter between the stentgraft and the artery wall has been done, but neither technique is wholly reliable. Consequently the majority of authors favour a pragmatic approach, if there is no endoleak but if the aneurysm is continuing to expand, whatever the pressure within the aneurysm, consideration may be given to OR or implantation of a new stent graft.

An enlarging abdominal aortic aneurysm after endovascular abdominal aortic repair without evidence of an endoleak and with an increase in diameter $\geq$10 mm should usually be repaired surgically or with a new stent graft. Level 2b, Recommendation B.

Post-operative device migration

Device migration after EVAR is defined as a movement of $>$10 mm relative to anatomic landmark with the use of three-dimensional CT reconstruction using a centre-line of flow or any migration leading to symptoms or requiring intervention.[533–535] Migration has been described with all current stent grafts including unibody design, modular configurations, infra-renal and suprarenal fixation and stent grafts with a longitudinal columnar support. Most series evaluating the prevalence of device migration have reported an increase after 24 months.[535–537] It can be asymptomatic and detected on CTA scan by the presence of a Type I endoleak with repressurisation of the aneurysm sac that can lead to rupture. Multiple factors affect stent graft migration: aortic neck and AAA morphology, accuracy of deployment, post-operative neck enlargement, proximal attachment failure, and characteristics of stent grafts. All these factors contribute to migration.

The length of the proximal neck above the AAA is an important factor. Instructions for use (IFU) for most devices call for a 15 mm neck length. Tonnessen *et al.*[535] found that the preoperative neck length was shorter in patients with stent graft migration (22 ± 2.1 mm vs. 31.2 mm $\pm$ 1.2, $p = 0.02$).

Proximal neck angulation ($>45^{\circ}$) also seems to predispose to migration. The diameter of the proximal neck may also be predictive of migration. Cao *et al.*[536] identified an initial neck diameter of $>$25 mm as an increased risk for development of neck dilatation in the future. Data from Conners *et al.*[537] suggest also that $>$ 20% device oversizing was associated with late aortic neck dilatation and subsequent stent graft migration. But most studies that investigated neck dilatation are flawed by poor methodology. None of the studies described a positive relationship between the degree of oversizing and the incidence of endoleaks. Oversizing up to 25% seems to decrease the risk of proximal endoleaks, and they are conflicting data regarding the risk of graft migration when oversizing is above this limit.[538] In addition, devices with limited radial force will not tolerate as much oversizing without graft infolding. Nonparallel aortic neck (conical vs. straight) and the presence of thrombus in the aortic neck have been also associated with an increased risk of distal migration.[539]

Proximal neck dilatation as a cause of stent graft migration has been the subject of an ongoing debate. Rodway *et al.*[516] have shown from a subset of EVAR 1 trial patients that the increase in aortic neck size was much greater 2 years after EVAR versus open repair, and large aortic necks may be at higher risk for dilatation as the aortic wall is often more diseased and weaker.[540,541] Resch *et al.*[542] have shown that graft design and characteristics of the device including suprarenal fixation, presence of hooks or barbs, or radial

force alone, can also influence device migration. Malina *et al*. have shown[543] on cadaveric aorta that barbs and hooks increased the proximal fixation tenfold. Heikkinen *et al*.[544] were first to report on the potential importance of iliac fixation, and Benhardash *et al*.[545] found that positional stability of suprarenal and infra-renal stent graft devices may rely heavily on iliac fixation and recommended that the iliac limb be extended at least 3 cm into the common iliac artery and preferably down to the iliac bifurcation.

Critical issues: Excellent results of EVAR for infra-renal AAA are primarily achieved in patients with favourable anatomy. Late neck dilatation following EVAR is a major cause of concern because of the potential loss of proximal fixation and seal.

Component separation

In a modular stentgraft system, there is the potential for individual components to separate. Component separation and dislocation were more prevalent in first generation stentgrafts, but even today radiological surveillance by plain films and CTA is essential to identify junctional component separation. In addition, shrinking of the aneurysm sac, creating upward forces on the iliac limbs can also generate component separation. Such a junction separation can lead to a Type III endoleak with sac pressurisation and requires either a bridging stent graft or an aorto-uniiliac conversion.[545,546] Fractures of the bare suprarenal stent struts have been described. They can result in separation from the main body of the stentgraft that are often associated with device migration. Material fatigue was also noted more often in patients with significant aortic neck angulation.[546]

Implications for surveillance after EVAR

The modes of failure after stent grafting are therefore well documented, and it is mandatory that all patients are recruited into a programme of systematic surveillance to assure the continued efficacy of the repair and to detect complications. The principal concerns are graft-related endoleak, aneurysm enlargement and migration of the stents at the aortic and iliac landing zones, and modular disconnections. Methods for surveillance are plain radiography, duplex ultrasonography (DU), contrast-enhanced computed tomography (CTA), magnetic resonance imaging (MRI) and sac pressure measurements, but as shown by Schlösser *et al*.,[508] rupture may occur in patients in whom no endoleak was seen during follow-up.

Plain radiography

Plain radiography using a standardised protocol with anteroposterior and lateral projections is very accurate in assessing stent fractures and modular disconnections. Device migration can also be depicted, but radiographs are obviously limited for the evaluation of aneurysm diameter and endoleaks. It is therefore not a stand-alone modality during follow-up.[547]

Colour duplex ultrasonography

In a recent bivariate meta-analysis of 21 published studies comparing CTA with colour duplex ultrasonography (DU) and contrast-enhanced DU (CDU), Mirza *et al*.[548] have shown that the pooled sensitivity and specificity of DU for endoleak detection was 0.77 and 0.94 respectively. In comparison, the pooled sensitivity and specificity of CDU was 0.98 and 0.88, respectively. These results should be interpreted with caution due to the heterogeneity and small sizes of the analysed trials, but this study confirms that CDU is probably a safe and sensitive modality for endoleak detection. Detection of flow direction of endoleaks is a specific advantage of DU compared to CTA, and very useful for further endoleak management. Parent *et al*.[549] reported the relationship between the Doppler waveform and the outcome of type II endoleaks. A 'to and fro' pattern was associated with spontaneous Type II endoleak seal and a monophasic or biphasic waveform was associated with endoleak persistence. But based on the lack of information about stentgraft integrity and migration, DU is not a stand-alone follow-up modality for surveillance after EVAR.

Contrast medium-enhanced CT

CTA with delayed images is the most widely used modality for follow-up after EVAR and currently the best method for detecting endoleaks. Although some controversy exists, most authors suggest that the sensitivity of CTA is superior to that of DU for endoleak detection.[550,551] CTA is the gold standard for measurement of the AAA diameter. The sensitivity and specificity rates for endoleak detection with CTA are better than those with conventional angiography and DU.[552] But detection of endoleak is very dependent on the CT protocol. The Eurostar study[553] suggested that delayed-phase CT with 3 mm slices was probably the best technique to demonstrate collateral reperfusion. Imaging of the patient after endoleak embolisation with coils, glue or other radiopaque material is challenging with CTA, and non-enhanced CT should be performed before CTA to assist in distinguishing embolic material from endoleaks. The major concerns of the frequent use of CTA are contrast agent-induced nephrotoxicity,[554] cumulative amount of exposure to ionizing radiation with potential lifetime cancer risk,[555] and cost. CTA can almost be a stand-alone modality for lifelong follow-up after EVAR but with the potential risk of radiation and nephrotoxicity.

Magnetic resonance imaging (MRI)

MRI and MRI angiography are an alternative to CTA. Reliability of MRI for the measurement of aortic diameter and detection of endoleaks is comparable to that of CTA[556] with a better analysis of endoleaks on three-dimensional gadolinium-enhanced dynamic and delayed gradient-echo sequences. The advantages of MRI versus CTA are related to the lack of exposure to the ionizing radiation and low nephrotoxicity of MRI contrast medium. Disadvantages of MRI are its lack of wide availability, difficulty assessing device integrity, contraindication in patients with cardiac pacemakers, and artefacts from stainless steel components; thus it is contraindicated with some of the current stentgrafts. MRI is not a stand-alone modality for surveillance after EVAR.

Direct sac pressure measurement

Direct pressure measurement in the aneurysm sac after EVAR has been reported. Although invasive, it is a reliable technique for the measurement of pressure inside the AAA.[557] Non-invasive AAA sac pressure measurement with implantable wireless pressure sensing systems has been developed

and is feasible, but mural aortic thrombus can affect pressure transmission. Okhi *et al*.[558] have shown the value of this technique in evaluation of the completeness of EVAR procedures; however, sac pressure did not predict the fate of the AAA during follow-up.[559] Studies that used the Remon system (CardioMEMS, Atlanta, Georgia)[560] showed good correlation between reduction of sac pressure and shrinkage of the AAA in small series with short follow-up. In case of endotension, the sensor may assist in therapeutic management. But remote pressure sensing does not provide any information about device integrity and is therefore unlikely to be a stand-alone modality after EVAR.

Nuclear medicine and experimental studies

Nuclear medicine scans for detection of endoleaks have been studied. Technetium Tc99m sulphur colloid imaging was unable to demonstrate endoleaks with rapid or slow flow. Many series with serum markers for AAA have been published. Matrix metalloproteinase (MMP-9) activity has been shown to change after EVAR and may have a role for long-term follow-up. Lack of decreasing of MMP-9 levels after EVAR may predict aneurysm expansion and could have a role as an enzymatic marker for endoleaks.[567] P-plasmin—antiplasmin complexes have been also reported as a serum marker associated with the expansion of AAA.[567] At present, the value of serum markers for follow-up after EVAR or endoleak treatment cannot be established.

Redefining post-operative surveillance after EVAR

Surveillance protocols for EVAR that are the current standard of care were derived from early trials without long-term data available and codified in the instructions for use for the devices. They include serial CTA and plain abdominal radiographs at 1, 6 and 12 months and yearly thereafter.[568,569] As previously stated major concerns with this protocol are the potential carcinogenic effects of the cumulative radiation dose and contrast load on renal function. In addition, the cost associated with this yearly lifelong protocol represents a third of the total costs of EVAR during a 5-year period.[475] Recent prospective multicentre studies series with 5-year follow-up[359,570,571] identify a patient cohort with cumulative absence of endoleak at 12 months and significant aneurysm shrinkage with a low subsequent risk for aneurysm-related complications. But Sternbergh *et al*.[475] also demonstrated also that the absence of endoleak does not confer immunity for all aneurysm-related complication with a 10.5% risk of any aneurysm-related complication at 5-year. On the basis of these data, we propose the following surveillance protocol for patient undergoing EVAR (Fig. 3).

Recommendations for surveillance after EVAR

All patients should have a CTA and plain radiographs with anteroposterior and lateral projections at 30 days post-procedure. Level 2c, Recommendation A.

If there is any endoleak or less than one stent component or iliac overlap, CTA at 6 months and 12 months with plain radiographs should be done with adequate treatment if indicated. Level 2b, Recommendation B.

In patients with no early endoleak and good component overlap, the traditional 6-month CTA could be omitted, but a CTA and plain radiographs should be done at 12-month. Level 2b, Recommendation B.

At 12 months, if there is no endoleak and a stable or shrinking AAA, a yearly DU is recommended with plain radiographs using a standardised protocol with antero—posterior and lateral projections to assess device migration, stent fractures and modular disconnections. If the patient's body habitus preclude an adequate DU, then a non-contrast CT with plain radiographs can be substituted. Level 2b, Recommendation B.

Any increasing aneurysm diameter or new endoleak, after prior imaging studies have suggested complete aneurysm sac exclusion, should prompt complete imaging with CTA and plain radiographs. Level 2b, Recommendation B.

Follow-up with DU, non-contrast CT imaging, and plain radiographs seems reasonable for patients with renal

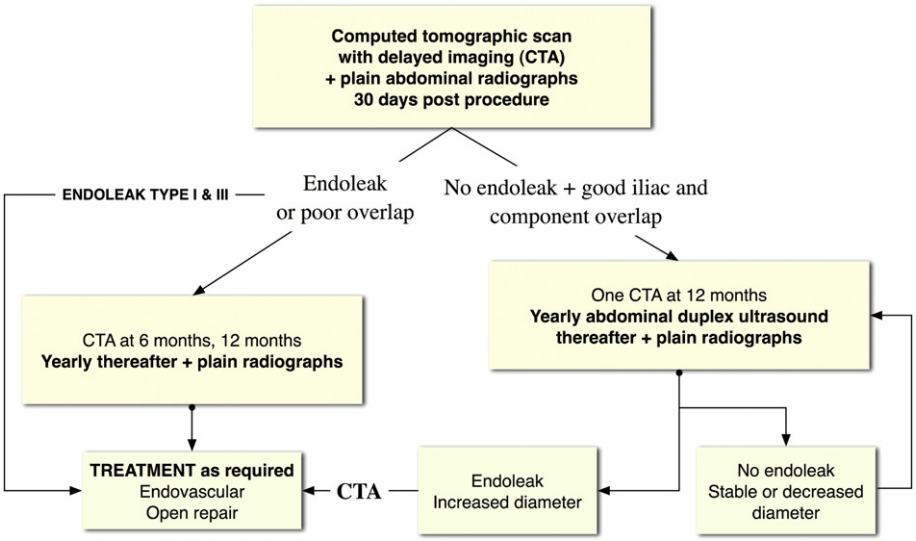

Figure 3 Simplified surveillance protocol for abdominal aortic stent grafts.

insufficiency at any time after EVAR. Level 3b, Recommendation C.

Critical issues

A set of plain radiographs with anteroposterior and lateral projections was retained in all phases of this protocol to identify potential failure in the metallic support system of different devices (stent fractures, barb separation, stent detachment) and assess adequacy of component overlap.

In some institutions, less than one stent component or iliac overlap are indications for secondary treatment before a Type I or III endoleak is observed.

New endoleaks may be identified as late as seven years following EVAR and justify lifelong follow-up of these patients. Further research with new stentgrafts is needed to confirm this lifelong follow-up.

There is a need to develop post-operative surveillance protocols, including optimal use of DU, contrast-enhanced DU, and CT imaging at various time periods after EVAR.

There is a need to study the effectiveness of pressure sensors in reduction of post-operative surveillance costs.

Limb occlusion and kinking

As observed in the EVAR 1 trial, stentgrafts are at a higher risk for limb thrombosis than prostheses placed during OR (2.3% vs. 0.2%, Odds ratio = 12.02, $p = 0.003$). Any distortion of the limbs of the stent graft used in EVAR may result in graft limb thrombosis. In a review of the Eurostar registry and over an 8-year period, post-operative stentgraft kinking was seen in 3.7% of cases and was significantly associated with Type I endoleak, Type III endoleaks (midgraft), graft thrombosis, graft migration, and conversion to open repair.[572] Patent symptomatic kinked stentgrafts can usually be treated by an additional stenting, whereas an occluded limb typically requires surgery with construction of a femoro-femoral crossover bypass. Standard mechanical balloon thrombectomy is less likely to be successful with EVAR grafts because of the angulation produced by the stents and the related risk of component or sealing zone disruption.

Follow-up of patients after endovascular AAA surgery should include a colour duplex ultrasound with ABI on a regular basis. Level 3, Recommendation B.

Stentgraft infection

The risk of graft infection after EVAR is low. The EUROSTAR registry reported only three procedures for endograft infection in 2846 patients followed up to 5 years, a rate of 0.1%.[509] The EVAR 1 trial showed a comparable incidence between OR (0.4%) and EVAR (0.2%, odds ratio = 0.48, $p = 0.49$) over a 4-year follow-up period.[116] Similarly, Schermerhorn *et al*.[118] found at 4-year in a cohort of 45,660 patients, comparable rates of graft infection among patients treated by EVAR and those who underwent OR (0.2% vs. 0.3%, $p = 0.13$). As seen in OR, stent graft infection after EVAR may present in association with a prosthetic-enteric fistula.[562] Prevention of stentgraft infection has focused on the use of antiseptic principles, including meticulous sterile technique and prophylactic antibiotics. Treatment strategies are similar to those described for graft infection after OR.

Renal failure after EVAR

EVAR attenuates the perioperative renal injury associated with OR, but in the long term, renal function actually deteriorates more quickly after EVAR[573] with a fall in the glomerular filtration rate independently associated with EVAR when compared with OR. The aetiology is probably multifactorial. Implicated factors include essentially the repeated renal contrast agent injury resulting from yearly CTA, and the potential role of suprarenal bare stent fixation with the risk of renal artery trauma, stent-induced stenosis and aortic neck thromboembolism following endovascular manipulation; however, studies have failed to demonstrate this.[563]

Quality of life

Utilities for a given health state represent the preference that individuals have for a certain health state. Utilities are usually used to estimate quality-adjusted life years. In most studies, utilities were calculated using EuroQuol-5D. Both randomised trials, EVAR 1 and DREAM,[276,503] demonstrated an initial dip in utilities due to the invasive nature of both EVAR and OR, but with OR the dip was more than that with EVAR at 4–6 weeks after the intervention. After 1 year, the utilities returned to baseline for both EVAR and OR with the exception of the DREAM trial, where utility scores for OR were better than for EVAR.[564]

Economics and cost-effectiveness

Several studies have been carried out to evaluate the cost-effectiveness of EVAR as compared to OR.[276,503,561,565,566] In most of these studies, only the hospital cost was included. Both randomised trials, EVAR 1 and DREAM,[276,503] have showed that the cost for EVAR is higher than that of OR. In addition, during follow up, the cost of EVAR is increased by a third due to the imaging requirements and more common reinterventions.

Critical issues

There is a need to develop cost-effectiveness strategies for EVAR and there is a need to develop robust, simple risk-scoring systems.

Evidence Needed

This chapter focuses on the specific areas where more evidence may improve future treatment strategies and decision-making in the care of patients with AAAs.

Aneurysm growth and rupture risk may be more accurately predicted in the future by risk scoring which includes genetic testing and measurement of mechanical and metabolic properties of the aorta. The potential slowing effect of statins and ACE inhibitors on AAA growth rates needs confirmation by randomised placebo-controlled trials. Genome-wide association studies and molecular proteomics may identify new mechanistic pathways, which can be targeted

therapeutically to effect a reduction in aneurysm growth and rupture risk.

Although the evidence that AAA screening programmes reduce the incidence of aneurysm rupture and are likely to be cost-effective is very strong, there are still many practical aspects which require better evidence. These include techniques to optimise the uptake of screening; whether internal or external diameter should be measured; cost-effective surveillance intervals; and the management of patients with small aneurysms to reduce anxiety and cardiovascular risk. Consideration has been given to the merits of screening by different subgroups. The value of population screening of older female smokers for AAA requires further investigation. Screening can take place either in hospitals or community care by visiting sonographers with portable ultrasound equipment or by a combination. Studies are needed that directly compare these approaches. Optimal safe, cost-effective rescreening intervals remain to be established.

The development of faster CT scanners and innovative post-processing algorithms today provide new possibilities for dynamic imaging. To date, the clinical relevance of dynamic imaging has not been proven, but dynamic changes of the aorta have to be taken into account in stentgraft selection and future stentgraft design. ECG-gated coronary CT as a pre-operative diagnostic adjunct should be actively evaluated by clinicians in vascular surgical practice.

The management of AAA depends on the size or diameter of the aneurysm and is a balance between the risk of aneurysm rupture and the operative mortality for aneurysm repair. There still remains some uncertainty about the management of small aneurysms in specific subgroups including young patients, females, and patients with limited life expectancy, which requires future evaluation.

There has been little recent good quality research to improve the outcomes of open AAA repair and the opportunity for a large trial of mini-laparotomy may have been missed. The timing of surgery for patients with symptomatic but unruptured aneurysms remains controversial. Patients that may benefit from surgery in an elective setting with pre-operative preparation have to be identified. After open AAA surgery, better imaging modalities are required for the diagnosis of graft infection.

The widespread adoption of EVAR in patients with ruptured AAA requires confirmation by randomised controlled trials. Branched and fenestrated endografts show promising results for the treatment of aortic disease involving visceral vessels and need to be studied more extensively to improve future endografts and treatment strategies.

On-table angiographic CT is evolving and might help in the intra-operative detection of complications which are possibly currently missed by unipolar angiography.

Endoleaks after EVAR are currently often not being diagnosed accurately, even with optimal CT scanning. New techniques should be evaluated to further increase the sensitivity of imaging modalities for diagnosing endoleaks, which may include MRI with a blood pool contrast agent and contrast-enhanced duplex ultrasonography.

Post-operative surveillance protocols can be further improved by evaluation of different follow-up imaging modalities, reintervention strategies and lengths of follow-up interval periods. Evaluation of treatment strategies to reduce late neck dilatation following EVAR is important to prevent loss of proximal fixation and seal. Better sustainability of endovascular stent-grafts is required to further reduce the risk of complications after EVAR. With increasing insight in predictors of the clinical course of patients after EVAR, it may be more and more possible to tailor treatment to each patient's unique characteristics, which will subsequently lead to an improved prognosis.

Summary and Conclusions

The Management of Abdominal Aortic Aneurysms Clinical Practice Guidelines of the European Society for Vascular Surgery provides recommendations for clinical care of patients with abdominal aortic aneurysms including pre-operative, perioperative and post-operative care.

Abdominal aortic aneurysm (AAA) can be defined as an abdominal aortic diameter of 3.0 cm in either anterior-posterior or transverse planes. Prevalence rates of AAA vary according to age, gender and geographical location. Important risk factors for AAA are advanced age, male gender, smoking and a positive family history for AAAs.

The reported average growth rate of AAAs between 3.0 and 5.5 cm ranges from 0.2 to 0.3 cm per year. Larger AAA diameters are associated with higher AAA growth rates. A wide variation between patients has been reported consistently. Smoking cessation may be recommended to reduce the rate of AAA growth.

Larger initial aneurysm diameter is a significant and independent risk factor for AAA rupture. Other factors that have been associated with an increased risk of AAA rupture include female gender, smoking and hypertension.

Population screening of older men for AAA, in regions where the population prevalence is 4% or more, reduces aneurysm-related mortality by almost half within 4 years of screening, principally by reducing the incidence of aneurysm rupture. Screening only smokers might improve the cost-effectiveness of aneurysm screening. Population screening of older women for AAA may not reduce the incidence of aneurysm rupture.

Population screening of older female smokers for AAA may require further investigation. Screening of older men and women having a family history of AAA might be recommended. Opportunistic screening of patients with peripheral arterial disease should be considered. The screening model chosen should be flexible for the local population characteristics. Men should be screened with a single scan at 65 years old. Screening should be considered at an earlier age for those at higher risk for AAA. Repeat screening should be considered only in those initially screened at a younger age or at higher risk for AAA.

Screening programmes should be well advertised and tailored to the local population to maximise attendance. Invitation to screening from the general or family practitioner might be received favourably. Incidental pathology should be referred to the family practitioner. If screening programmes use relatively inexperienced screening staff and portable ultrasound devices, programmes should be audited for quality control.

Screen detection of an AAA causes a small but temporary reduction in quality of life. Aneurysm screening should only be conducted if the audited mortality from aneurysm repair

at the referral hospital is low. Referral hospital facilities must be in place before AAA screening starts to cope with an increased number of elective AAA repairs, both open and endovascular.

All subjects with a screen-detected aneurysm should be referred for cardiovascular risk assessment with concomitant advice and treatment, including statins and smoking cessation therapy. Rescreening intervals should shorten as the aneurysm enlarges.

When the threshold diameter (5.5 cm, measured by ultrasonography, in males) is reached or symptoms develop or rapid aneurysm growth is observed (>1 cm/year), immediate referral to a vascular surgeon is recommended. To prevent interval rupture, it is recommended that a vascular surgeon review patients within 2 weeks of the aneurysm reaching 5.5 cm or more in diameter. In some centres an earlier referral, at between 5.0 and 5.5 cm is an acceptable alternative practice. In-patient management might be considered for aneurysms over 9 cm in diameter. A policy of ultrasonographic surveillance of small aneurysms (4.0–5.5 cm) is safe and advised for asymptomatic aneurysms. Patients with a higher risk of rupture should be considered for surgery when the maximum aortic diameter reaches 5.0 cm. There remains some uncertainty about the management of small aneurysms in defined subgroups (e.g. young patients, females, and those with limited life expectancy). Females should be referred to vascular surgeons for assessment at a maximum aortic diameter of 5.0 cm as measured by ultrasonography, and aneurysm repair should be considered at a maximum aneurysm diameter of 5.2 cm in females.

Several pre-operative care strategies may improve early post-intervention morbidity and mortality. Smoking cessation and physiotherapy can reduce post-operative complications. All patients undergoing AAA repair should have an assessment of their respiratory function. Statins should be started one month before intervention to reduce cardiovascular morbidity. Statins should be continued in the perioperative period, for an indefinite duration. β-blockers are recommended in patients with ischaemic heart disease or who have myocardial ischemia on stress testing and can be started 1 month before intervention. Patients with vascular disease should be started on low-dose aspirin therapy, unless specific contraindications exist, and the aspirin should be continued through the perioperative period. Blood pressure control should be initiated for secondary prevention to reduce cardiovascular morbidity. Vascular surgeons should be familiar with their current national guidelines for the management of hypertension.

All patients undergoing AAA repair should have a formal assessment of their cardiac risk. This includes a pre-operative ECG in all cases. Patients undergoing open or laparoscopic AAA repair, in the presence of cardiac risk factors, or a positive cardiac history, should undergo a pharmacological stress echo or myocardial perfusion scan prior to surgery. Patients undergoing EVAR, in the presence of cardiac risk factors, or a positive cardiac history should have a trans-thoracic echocardiogram and consideration of a pharmacological stress test or myocardial perfusion scan prior to AAA repair. The role of ECG-gated coronary CT as a diagnostic adjunct should be actively evaluated by clinicians in vascular surgical practice. Coronary revascularisation should be considered prior to AAA repair for patients who have ischaemic coronary symptomatic or left main coronary artery disease. No evidence-based recommendation can be made at present as to which patients will benefit most from this technique.

A history of congestive heart failure, chronic pulmonary disease, or renal insufficiency serve as negative predictors for the 30-day mortality and also for the long time survival after elective open AAA repair. All patients must have serum creatinine measured and GFR estimated preoperatively. Referral to a renal physician is advised where these are outside the normal range. All patients should be adequately hydrated prior to AAA repair.

All medium- and high-risk patients being considered for an AAA repair should be reviewed by a specialist vascular anaesthesiologist prior to admission for surgery. Where debate exists about a patient's fitness, risk stratification based on physiological and morphological parameters should be undertaken.

Open and endovascular treatment options should be considered in all patients with incorporation of the patient's preference and anatomical suitability. Patients with large aneurysms who require a custom-made endograft should be offered open aneurysm repair. Patients at high cardiac risk as well as those who require AAA treatment immediately after cardiac intervention should be better treated with EVAR, if anatomically suitable. Laparoscopic aneurysm repair has a limited role and should only be attempted in centres with an advanced laparoscopic practice and where suitable mentoring is available.

AAA repair should only be performed in hospitals performing at least 50 elective cases per annum, whether by open repair or EVAR. Symptomatic aneurysms should be repaired on the next available elective operating list, since they may have a higher risk of rupture. Where morphologically suitable, patients should be offered EVAR, which has a lower operative mortality for symptomatic cases than open repair.

Several imaging modalities can be used in the preprocedural care of patients with an abdominal aortic aneurysm, such as digital subtraction angiography (DSA), duplex ultrasound, intravascular ultrasound (IVUS), computed tomography angiography (CTA), and magnetic resonance angiography (MRA). DSA is not recommended as a routine pre-operative imaging modality. CTA is a fast and reproducible modality, and provides all necessary detailed anatomical information for operation planning. CTA can provide 3-D information and dynamic images, which has become more valuable since the introduction of EVAR. CTA therefore currently is the primary pre-operative imaging modality in most centres. The main use of angiography is during and after EVAR. An alternative for periprocedural angiography is IVUS, allowing for perioperative real time diameter and length measurements. IVUS can help in reducing the amount of perioperative contrast used.

Open non-ruptured AAA repair

A single shot antibiotic prophylaxis in patients with abdominal aneurysm repair is recommended to avoid early

graft infection and wound infection. Body temperature should be kept at a physiological level (<36 °C) during AAA repair to avoid perioperative complications. No specific fluid replacement strategy has been shown to be superior to another in the use of abdominal aortic surgery. A combination therapy from crystalloid and colloid solutions is most commonly used. The use of cell salvage and ultrafiltration devices might be recommended in case of an expected large blood loss and if the risk of disease transmission from transfusion is considered high.

Fast-track surgery can positively influence perioperative outcome after AAA repair. Appropriate outpatient pre-operative work-up with admission close to the time coupled with judicious fluid management and early mobilisation can lead to improved outcomes and reduced high-dependency care and total lengths of stay.

In the absence of convincing evidence favouring any one type of incision, the incision for open repair should be tailored to the patient needs and local expertise. Available prosthetic graft materials for AAA repair are comparable concerning patency and long-term results. If the iliac arteries are unaffected (aneurysm formation or arterial occlusive disease) tube grafts should be used because of the shorter operative time and the reduced risk of adjacent injuries of the neighbouring structures. Tube graft placement during AAA surgery is justified even for moderate common iliac artery dilatation <25 mm. Common iliac arteries with a pre-operative diameter $\geq$ 25 mm warrant insertion of a bifurcated graft during AAA repair.

When there is suspicion of impaired pelvic and sigmoid colonic perfusion, the inferior mesenteric artery needs to be reimplanted during aortic aneurysm repair. The perfusion of one hypogastric artery or the inferior mesenteric artery is mandatory to avoid post-operative complications.

Endovascular non-ruptured AAA repair

An appropriately sized aortic endograft should be selected on the basis of patient anatomy. Generally, the device should be oversized 15—20% with respect to the aortic neck diameter. The preferential use of local anaesthesia for EVAR is feasible and appears to be well tolerated, whilst restricting regional anaesthesia or general anaesthesia to those with predefined contraindications. Percutaneous approach for EVAR may provide a less invasive aortic access and can facilitate shorter hospital stay in selected patients.

Preservation of flow to at least one hypogastric artery is recommended in standard risk patients. Hypogastric embolisation is usually preferred over simple coverage of its ostium by the endograft to prevent the risk of Type 2 endoleak, but coils should be placed as proximal as possible to spare collateral circulation. In cases with a short or diseased neck the use of endografts with fenestrations shows promising results but should be performed with appropriate training and in centres with extensive experience in EVAR.

Open ruptured AAA repair

Immediate repair is recommended in patients with documented aneurysm rupture. Hypotensive resuscitation might have a beneficial effect on the survival in case of abdominal aortic aneurysm rupture. Systolic blood pressure should range between 50 and 100 mm Hg depending on the patient's condition on admission. An increased abdominal pressure serves as a negative predictive factor for the survival after open repair of a ruptured abdominal aortic aneurysm. Measurement of the intra-abdominal pressure is recommended and in case of elevated levels (>20 mm Hg) in combination with organ dysfunction decompressive surgery should immediately be performed. Temporary abdominal closure systems can positively influence outcome.

Endovascular ruptured AAA repair

The widespread adoption of EVAR in patients with ruptured AAA requires confirmation by randomised controlled trials. Currently, there is no level I evidence yet. A few recent population-based studies support EVAR for rupture, although the effect is likely to be overstated due to selection bias.

The set-up of standardised protocols for endovascular treatment of ruptured AAA including a multidisciplinary approach has been demonstrated successfully and should be employed. Equipment for EVAR and open repair should be present all the time.

The placement of an aortic occlusive balloon during EVAR for ruptured AAA can be used to control haemodynamic instability from ongoing blood loss, although the use should be limited to situations when there is severe circulatory collapse. Pre-operative fluid administration should be restricted to a minimum to maintain hypotensive haemostasis.

Patients who are unconscious or in whom a systolic blood pressure cannot be maintained should be immediately transferred to the operating room. The decision to proceed with emergency open repair, placement of an aortic occlusion balloon or invasive imaging studies should depend on the comfort level of the surgeon and conditions of the patient.

Follow-up after open AAA repair

All patients treated for an AAA should receive the best medical treatment including aspirin and statins. Post-operative surveillance protocols, including use of colour duplex ultrasound or CT imaging is recommended at regular intervals after open AAA repair to evaluate for para-anastomotic aortic aneurysm (at 5 years, 10 years, 15 years). Any gastrointestinal bleeding in a patient having an aortic graft should prompt the evaluation of a prosthetic-enteric fistula. Patients with AAA appear to have a relatively high risk for both inguinal and incisional hernia.

Follow-up after endovascular AAA repair

All patients receiving an aortic endograft should be kept on the best medical treatment including aspirin and statins.

CTA with delayed images is the most widely used modality for follow-up after EVAR and currently the best

method for detecting endoleaks. All patients should have a CTA and plain radiographs with anteroposterior and lateral projections at 30 days post-procedure. If there is any endoleak or less than one stent component or iliac overlap, CTA at 6 months and 12 months with plain radiographs should be done with adequate treatment if indicated. In patients with no early endoleak and good component overlap, the traditional 6-month CTA could be omitted, but a CTA and plain radiographs should be done at 12 months. At 12 months, if there is no endoleak and a stable or shrinking AAA, a yearly Doppler ultrasound (DU) is recommended with plain radiographs using a standardised protocol with anteroposterior and lateral projections to assess device migration, stent fractures and modular disconnections. If the patient's body habitus precludes an adequate DU, then a non-contrast CT with plain radiographs can be substituted. Any increasing aneurysm diameter or new endoleak, after prior imaging studies have suggested incomplete aneurysm sac exclusion, should prompt complete imaging with CTA and plain radiographs. Follow-up with DU, non-contrast CT imaging, and plain radiographs seems reasonable for patients with renal insufficiency at any time after EVAR. Follow-up of patients after endovascular AAA surgery should include also a colour duplex ultrasound with ABI on a regular basis.

Treatment of endoleaks differs per type. All Type I endoleaks should be treated during follow-up. Type II endoleaks without increased sac diameter can be observed. Endovascular or laparoscopic treatment is recommended for Type II endoleaks with sac diameter increase $\geq$10 mm. Conversion to open surgery may be required in case of failure of the reintervention. Treatment is recommended for Type III endoleaks. Treatment is not generally recommended for Type IV endoleaks. Endotension, an enlarging AAA after endovascular abdominal aortic repair without evidence of an endoleak, and with an increase in diameter $\geq$10 mm should usually be repaired surgically or with a new endograft.

Excellent results of EVAR for infra-renal AAA are primarily achieved in patients with favourable anatomy. Post-operative surveillance protocols can be further improved by evaluation of different follow-up imaging modalities, reintervention strategies and lengths of follow-up interval periods. Evaluation of treatment strategies to reduce late neck dilatation following EVAR is important to prevent loss of proximal fixation and seal. Better durability of endovascular stent-grafts is required to further reduce the risk of complications after EVAR. With increasing insight into the predictors of the clinical course of patients after EVAR, it may be more and more possible to tailor treatment to each patient's unique characteristics, which will subsequently lead to an improved prognosis.

Guidelines Committee

Authors

Frans L. Moll, MD, PhD, chairman, University Medical Center Utrecht, Utrecht, The Netherlands;

Gustav Fraedrich, MD, University Hospital Innsbruck, Austria;

Janet T. Powell, MD, PhD, Imperial College, London, United Kingdom;

Jean-Baptiste Ricco, MD, PhD, University of Poitiers, Poitiers, France;

Fabio Verzini, MD, University of Perugia, Perugia, Italy.

Co-authors

Felix J.V. Schlösser, MD, PhD
- Introduction
- Chapter 1 — Epidemiology
- Evidence needed
- Summary

Peter J.E. Holt, MD, PhD
- Chapter 2 — Screening
- Chapter 3 — Decision making for elective AAA repair

Matthew Waltham, MD, PhD
- Chapter 2 — Screening
- Chapter 3 — Decision making for elective AAA repair

Joost A. van Herwaarden, MD, PhD
- Chapter 4 — Pre- and perioperative imaging

Jasper W. van Keulen, MD, PhD
- Chapter 4 — Pre- and peroperative imaging

Barbara Rantner, MD, PhD
- Chapter 5 — Management of non-ruptured AAA Open AAA repair
- Chapter 6 — Management of ruptured AAA Open AAA repair

Francesco Setacci, MD
- Chapter 5 — Management of non-ruptured AAA Endovascular AAA repair
- Chapter 6 — Management of ruptured AAA Endovascular AAA repair

Stéphan Haulon, MD, PhD
- Chapter 7 — Follow-up after AAA repair

Reviewers

Professor Gerry Fowkes, University of Edinburgh, UK;
Anne Cheetham, patient & public panel from the Circulation Foundation;
Professor Frank Lederle, University of Minnesota, USA.

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Fabio Verzini: None declared.
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Matthew Waltham: None declared.
Barbara Rantner: None declared.
Francesco Setacci: None declared.
Stéphan Haulon is a consultant for Cook Medical and General Electric.

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