The care of patients with an abdominal aortic aneurysm: The Society for Vascular Surgery practice guidelines

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DEFINITION OF THE PROBLEM

Purpose of these guidelines

The Clinical Practice Council of the Society for Vascular Surgery charged a writing committee with the task of updating practice guidelines, initally published in 2003, for surgeons and physicians who are involved in the preoperative, operative, and postoperative care of patients with abdominal aortic aneurysms (AAA).[1] This document provides recommendations for evaluating the patient, including risk of aneurysm rupture and associated medical co-morbidities, guidelines for selecting surgical or endovascular intervention, intraoperative strategies, perioperative care, long-term follow-up, and treatment of late complications.

Decision making related to the care of patients with AAA is complex. Aneurysms present with varying risks of rupture and patient specific factors influence anticipated life expectancy, operative risk, and the need to intervene. Careful attention to the choice of operative strategy, as influenced by anatomic features of the AAA, along with optimal treatment of medical co-morbidities is critical to achieving excellent outcomes. Moreover, appropriate postoperative patient surveillance and timely intervention in the case of a late complication is necessary to minimize subsequent aneurysm-related death or morbidity. All of these clinical decisions are determined in an environment where cost-effectiveness will ultimately dictate the ability to provide optimal care to the largest possible segment of the population. Currently available clinical data sets have been reviewed in formulating these recommendations. However, an important goal of this document is to clearly identify those areas where further clinical research is necessary.

Methodology and evidence

A comprehensive review of the available clinical evidence in the literature was conducted in order to generate a concise set of recommendations. The strength of any given recommendation and the quality of evidence was scored based on the **GRADE system** (Table I).[2] When the benefits of an intervention outweighed its risks, or, alternatively, risks outweighed benefits, a **strong recommendation** was noted. However, if benefits and risks were less certain, either because of low quality evidence or because high quality evidence suggests benefits and risks are closely balanced, a **weak recommendation** was recorded. The quality of evidence that formed the basis of these recommendations was scored as high, moderate, or low. Not all randomized controlled trials are alike and limitations may compromise the quality of their evidence. In addition, if there is a large magnitude of effect, the quality of evidence derived from observational studies may be high. Thus, **quality of evidence was scored as high** when additional research is considered very unlikely to change confidence in the estimate of effect; **moderate** when further research is likely to have an important impact in the estimate of effect; or **low** when further research is very likely to change the estimate of the effect.

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Table I. Criteria for strength of a recommendation and grading quality of evidence

Strength of a recommendation
Strong
Benefits > Risks
Risks > Benefits
Weak
Benefits ~ Risks
Quality of evidence precludes accurate assessment of risks and benefits
Grading quality of evidence
High
Additional research is considered very unlikely to change confidence in the estimate of effect
Moderate
Further research is likely to have an important impact on in the estimate of effect
Low
Further research is very likely to change the estimate of the effect

Adapted from Guyatt G, Gutterman D, Baumann MH, Addrizzo-Harris D, Hylek EM, Phillips B, et al. Grading strength of recommendations and quality of evidence in clinical guidelines. Chest 2006;129:174-81.

Table II. Risk factors for aneurysm development, expansion, and rupture

Symptom	*Risk factors*
AAA development	• Tobacco use • Hypercholesterolemia • Hypertension • Male gender • Family history (male predominance)
AAA expansion	• Advanced age • Severe cardiac disease • Previous stroke • Tobacco use • Cardiac or renal transplant
AAA rupture	• Female gender • ↓ FEV_1 • Larger initial AAA diameter • Higher mean blood pressure • Current tobacco use (length of time smoking ≫ amount) • Cardiac or renal transplant • Critical wall stress – wall strength relationship

AAA, Abdominal aortic aneurysm.

GENERAL APPROACH TO THE PATIENT

History

The medical history is helpful in determining the patient's risk of developing an AAA. Even in the absence of clinical symptoms, knowledge of the risk factors for developing an AAA may facilitate early diagnosis. The Aneurysm Detection and Management Veterans Affairs Cooperative Study Group (ADAM) trial found a number of factors to be associated with increased risk for AAA: advanced age, greater height, coronary artery disease (CAD), atherosclerosis, high cholesterol levels, hypertension, and, in particular, smoking.[3] Indeed, aortic aneurysms occur almost exclusively in the elderly. In a 2001 study, the mean age of patients undergoing repair for AAA in the United States was 72 years.[4-8] Men outnumber women by a factor of 4 to 6 to 1.[4-8] Family members are also at significant risk with 12% to 19% of those undergoing aneurysm repair having a first-degree relative with an AAA.[9-11] In a recent nationwide survey conducted in Sweden, the relative risk of developing AAA for first-degree relatives was approximately double that of persons without a family history of AAA.[12] Neither the gender of the index person nor the first-degree relative influenced the risk of AAA. An AAA is over seven times more likely to develop in a smoker than a nonsmoker, with the duration of smoking, rather than total number of cigarettes smoked, being the key variable (Table II).[13] The risk for developing an AAA is lower in women, African Americans, and diabetic patients.

Risk factors for rupture have also been identified. The United Kingdom Small Aneurysm trial (UKSAT) reported 103 aneurysm ruptures in 2,257 patients over a period of seven years, with an annual rupture rate of 2.2%.[14] Factors significantly and independently associated with an increased risk of rupture included female gender, large initial aneurysm diameter, low forced expiratory volume in one second (FEV1), current smoking history, and elevated mean blood pressure.[14-16] Women are two to four times more likely to experience rupture than men.[14-17] Aneurysms in transplant patients also appear to have high expansion and rupture rates.[18]

Prior surgical history is crucial to exclude certain disease processes, such as appendicitis or cholecystitis, that may mimic the presentation of a symptomatic aneurysm. In addition, the nature and extent of previous abdominal surgery may influence operative approach. When a pulsatile abdominal mass is discovered in a patient who has previously undergone open surgical repair (OSR) of an AAA, the presence of an anastomotic pseudoaneurysm,[19] iliac artery aneurysm,[20] or suprarenal aortic aneurysm should be considered. Likewise, complaints of abdominal or back pain in a patient with a prior history of endovascular aortic aneurym repair (EVAR) requires the treating physician to exclude an endoleak with attendant aneurysm expansion or rupture.[21-24]

Physical examination

An abdominal aortic aneurysm has been defined as "a pulsating tumor that presents itself in the left hypochondriac or epigastric regions."[25] The abdominal aorta begins at the level of the diaphragm and the 12th thoracic vertebra and runs in the retroperitoneal space just anterior to and slightly to the left of the spine. At approximately the level of the umbilicus and the fourth lumbar vertebra, the aorta bifurcates into the right and left common iliac arteries.

Unfortunately, only 30% to 40% of aneurysms are noted on physical examination with detection dependant on aneurysm size.[26] Aneurysms greater than 5 cm are detected in 76% of patients, whereas aneurysms between 3

cm and 3.9 cm are identified in only 29%. As would be anticipated, detection is limited by truncal obesity.[27,28] Although most abdominal aneurysms are supraumbilical, in some patients, the aorta becomes more tortuous and elongated with age and an aneurysm may appear to be infraumbilical or to one side of the abdomen. The common iliac arteries may also become aneurysmal and palpable in one of the lower abdominal quadrants. It bears emphasis that palpation has not been reported to precipitate aortic rupture. An abdominal aneurysm may be present in up to 85% of patients with a femoral artery aneurysm and in up to 62% of those with a popliteal aneurysm.[29,30] In contrast, patients with an abdominal aneurysm have a 14% incidence of either a femoral or a popliteal artery aneurysm.[31]

Physical examination should include an assessment of femoral and popliteal arteries in all patients with a suspected abdominal aortic aneurysm.

Level of recommendation:	Strong
Quality of evidence:	High

Co-morbid disease

Coronary artery disease (CAD) is the leading cause of early and late mortality after AAA repair.[32] Chronic kidney disease, chronic obstructive pulmonary disease (COPD), and diabetes mellitus may also influence morbidity and mortality. Accordingly, further evaluation is warranted and optimization of perioperative status beneficial when any of these conditions are present.

Cardiac disease

Preoperative evaluation of cardiac morbidity. Several studies have documented a lower incidence of perioperative cardiac complications with EVAR than OSR. In review of a statewide experience between 2000 and 2002, Anderson et al reported a 3.3% incidence of cardiac complications for EVAR as compared with 7.8% for OSR in 2002, which was similar to the events rates in 2001 and 2000.[33] Likewise, Schermerhorn et al[34] identified a significantly lower incidence of myocardial infarction (MI) among patients undergoing EVAR as compared with open repair in an analysis of propensity-score-matched cohorts of Medicare beneficiaries treated between 2001 and 2004 (7% vs 9.4%, $P < .001$). Similarly, in a small retrospective review of patients with three or more cardiac risk factors undergoing EVAR or OSR, the incidence of elevated troponin levels was significantly lower among those treated by EVAR (13% vs. 47%, $P = .001$).[35] However, not all studies have documented a reduction cardiac morbidity with EVAR. Notably, the incidence of cardiac complications was similar for both EVAR (5.3%) and OSR (5.7%) in the Dutch Randomized Endovascular Aneurysm Management (DREAM) trial.[36] In summary, while elective open AAA repair can generally be considered to carry a higher risk for a perioperative cardiovascular event, EVAR should be considered a procedure that is associated with intermediate to high cardiac risk in the range of 3% to 7%.

Table III. Preoperative cardiac evaluation for patients undergoing aneurysm repair

1. Is there an active cardiac condition?	• Unstable coronary syndrome
	  • Unstable or severe angina
	  • Recent MI (<1 month)
	• Decompensated CHF
	• Significant arrhythmias
	• Severe valvular disease
	Presence cancels or delays aneurysm repair until conditions treated. Implement medical management and consider coronary angiography.

2. Does the patient have good functional capacity without symptoms?	• MET ≥4 (see Table IV) Clinical risk factors: • Mild angina pectoris • Prior MI • Compensated or prior CHF • Diabetes mellitus • Renal insufficiency May proceed with aneurysm repair. In patients with known cardiovascular disease or at least one clinical risk factor, beta blockade is appropriate.
3. Is functional capacity poor or unknown?	• MET <4 (see Table IV) Clinical risk factors: • Mild angina pectoris • Prior MI • Compensated or prior CHF • Diabetes mellitus • Renal insufficiency In patients with three or more clinical risk factors, preoperative non-invasive testing is appropriate if it will change management.

CHF, Congestive heart failure; *MET,* metabolic equivalent unit; *MI,* myocardial infarction.
Adapted from Fleisher LA, Beckman JA, Brown KA, Calkins H, Chaikof E, Fleischmann KE, et al. ACC/AHA 2007 Guidelines on perioperative cardiovascular evaluation and care for noncardiac surgery: executive summary. Circulation 2007;116:1971-96.

Regardless of the nature of repair, a substantial portion of patients with AAA have underlying CAD and postoperative MI carries with it a substantially increased risk of death, as well as a high risk for later cardiovascular events and death.[37,38] Thus, it is critical to minimize the risk of cardiac morbidity during the course of OSR or EVAR for AAA. Guidelines, endorsed by the Society for Vascular Surgery, have been recently updated for preoperative cardiac evaluation of patients undergoing noncardiac vascular surgery.[39] Past guidelines have proven safe and effective in reducing unnecessary resource utilization.[40-42]

The first step, prior to planned aneurysm repair, is to determine whether an active cardiac condition exists, such as an unstable coronary syndrome (unstable or severe an-

Table IV. Estimated energy required for various activities

Activity level	*Examples of activity level*
Poor (1-3 METs)	Eating, walking at 2-3 miles per hour, getting dressed, light housework (washing dishes)
Moderate (4-7 METs)	Climbing a flight of stairs or walking up a hill, running a short distance, heavy housework (scrubbing floors or moving furniture)
Good (7-10 METs)	Doubles tennis, calisthenics without weights, golfing without cart
Excellent (>10 METs)	Strenuous sports such as football, basketball, singles tennis, karate, jogging 10 minute mile or greater, chopping wood

MET, Metabolic equivalent unit (1 MET = 3.5 mL $*$ kg^{-1} $*$ min^{-1} oxygen uptake).
Adapted from The Duke Activity Status Index (Hlatky MA, Boineau RE, Higginbotham MB, Lee KL, Mark DB, Califf RM, et al. A brief self-administered questionnaire to determine functional capacity (the Duke Activity Status Index). Am J Cardiol 1989;64:651-4. and Nelson CL, Herndon JE, Mark DB, Pryor DB, Califf RM, Hlatky MA. Relation of clinical and angiographic factors to functional capacity as measured by the Duke Activity Status Index. Am J Cardiol 1991;68:973-5.).

gina, recent MI <one month), decompensated heart failure (new onset, worsening, or New York Heart Association [NYHA] Class IV), significant arrhythmia (atrioventricular [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) (Table III). These conditions represent major clinical risks for postoperative MI or cardiovascular related death and mandate intensive management prior to aneurysm repair. In these cases, patients may be referred for coronary angiography to assess further therapeutic options. Depending on the results of the test and the risk of delaying repair, it may be appropriate to proceed to planned surgical or endovascular treatment with maximal medical therapy.

In the absence of an active cardiac condition, further non-invasive testing is only indicated if it will change management. For an otherwise elective repair, both the patient's functional capacity and the presence of clinical risk factors will dictate the need for further testing. The functional capacity can be estimated from a patient's ability to perform various activities (Table IV). Asymptomatic patients capable of a moderate or high activity level (metabolic equivalent unit [MET] $\geq$ 4), such as climbing stairs or a short run, generally do not benefit from further testing. Those who cannot achieve these levels of activity or in whom activity level is unknown may benefit from non-invasive stress testing, if additional clinical risk factors exist. Lee et al[43] have derived and validated a Revised Cardiac Risk Index for stable patients, without an active cardiac condition. Five independent clinical risk factors were identified including heart disease (history of MI, positive treadmill test, use of nitroglycerin, angina, or electrocardiogram [ECG] with abnormal Q waves); congestive heart failure (history of heart failure, pulmonary edema, paroxysmal nocturnal dyspnea, peripheral edema, bilateral rales, S3, or chest radiograph with pulmonary vascular redistribution); cerebral vascular disease (history of transient ischemic attack or stroke); diabetes mellitus; and renal insufficiency (creatinine > 2 mg/dL). The presence of an increasing number of risk factors correlates with an increased risk of a postoperative event and patients who display three or more risk factors and have an unknown or low activity level (MET < 4) may benefit from stress testing, if it will change management. Otherwise, all patients should undergo a 12-lead ECG within one month of planned repair. Transthoracic echocardiography (TTE) may be of value in patients with dyspnea of unknown origin or worsening dyspnea and a history of heart failure, if TTE has not been performed within the past year. However, it should be noted that resting left ventricular function is not a consistent predictor of postoperative MI or death.

Patients with active cardiac conditions (eg, unstable angina, decompensated heart failure, severe valcular disease, significant arrythmia) should be evaluated and treated per American College of Cardiology (ACC)/American Heart Association (AHA) guidelines before EVAR or OSR.

Level of recommendation: Strong
Quality of evidence: Moderate

Noninvasive stress testing should be considered for patients with a history of three or more clinical risk factor factor (ie, coronary artery disease [CAD], congestive heart failure [CHF], cerebrovascular accident [CVA], diabetes mellitus [DM], chronic renal insufficiency [CRI]) and an unknown or poor functional capacity (MET<4) who are undergoing aneurysm repair, if it will change management.

Level of recommendation: Strong
Quality of evidence: Moderate

A preoperative resting 12-lead ECG is recommended for all patients undergoing endovascular or open aneurysm repair within 30 days of planned treatment.

Level of recommendation: Strong
Quality of evidence: High

Preoperative echocardiography is recommended for patients undergoing aneurysm repair with dyspnea or heart failure.

Level of recommendation: Strong
Quality of evidence: High

Routine coronary revascularization by coronary artery bypass grafting (CABG) or percutaneous transluminal coronary angioplasty (PTCA) prior to elective vascular surgery in patients with stable cardiac symptoms does not appear to significantly alter the risk of postoperative MI or death or long-term outcome. In the Coronary Artery Revascularization Prophylaxis (CARP) trial, 510 patients with significant coronary artery stenosis scheduled for vascular operations were randomized to PTCA or CABG or no coronary

revascularization before surgery.[44] Patients with left main disease, severe aortic stenosis, or an ejection fraction of less than 0.20 were excluded. Both short-term risk and long-term outcome were not changed by coronary intervention. Similar findings have been reported by other randomized studies.[45,46] Patients with stable cardiac disease and established risk factors remain at risk, whether or not coronary revascularization is performed. Nonetheless, it bears emphasis that coronary revascularization is indicated for those patients with acute ST elevation MI, unstable angina, or stable angina with left main coronary artery or three-vessel disease, as well as those patients with two-vessel disease that includes the proximal left anterior descending artery, and either ischemia on non-invasive testing or an ejection fraction of less than 0.50.

In summary, percutaneous or surgical intervention for coronary artery disease prior to aortic surgery should be consistent with established guidelines.[47] If a patient requires PTCA, it may be reasonable for patients to wait at least two weeks and, preferably, four to six weeks after PTCA before undergoing aneurysm repair.[39] If a drug eluting stent is utilized, it may be reasonable to defer surgery for 12 months if rupture risk is not high. If this is not possible, EVAR is preferred as thienopyridine therapy can be continued during the perioperative period. If OSR is required, the thienopyridine should be discontinued 10 days preoperatively, aspirin continued, and the thienopyridine restarted as soon as possible after surgery.[48] Simultaneous aneurysm repair and CABG has been evaluated, and there are some data to support its use in select symptomatic patients with critical CAD.[49,50]

Coronary revascularization is recommended prior to aneurysm repair in patients with acute ST elevation MI, unstable angina, or stable angina with left main coronary artery or three-vessel disease.

Level of recommendation:	Strong
Quality of evidence:	High

Coronary revascularization is recommended prior to aneurysm repair in patients with stable angina with two-vessel disease that includes the proximal left anterior descending artery, and either ischemia on non-invasive testing or an ejection fraction of less than 0.5.

Level of recommendation:	Strong
Quality of evidence:	High

In patients who may need AAA repair in the subsequent 12 months and in whom coronary revascularization with percutaneous coronary intervention (PCI) is appropriate, a strategy of balloon angioplasty or bare-metal stent placement followed by four to six weeks of dual-antiplatelet therapy is suggested.

Level of recommendation:	Weak
Quality of evidence:	Moderate

It is suggested to defer elective open AAA repair for four to six weeks after bare-metal coronary stent implantation or coronary artery bypass grafting or for 12 months after drug-eluting coronary stent implantation, if rupture risk is not high.

Level of recommendation:	Weak
Quality of evidence:	Low

In patients who have received drug-eluting coronary stents and who must undergo open AAA repair, it is suggested to discontinue thienopyridine therapy 10 days preoperatively, continue aspirin, and restart the thienopyridine as soon as possible after surgery.

Level of recommendation:	Weak
Quality of evidence:	Low

Perioperative medical management of coronary artery disease. Although animal studies have suggested that beta blockade protects against aneurysm expansion and rupture, the evidence in clinical trials has generally not supported this view.[51-56] However, in patients with known cardiovascular disease or at least one clinical risk factor, perioperative heart rate control with beta blockade appears appropriate. Several randomized, controlled trials have demonstrated that perioperative beta blockade in high-risk patients reduce cardiac morbidity and death.[57-59] Beta blockers should be started days to weeks before elective surgery and accumulating evidence suggests that heart rate control less than 65 beats per minute should be targeted.[45,60] Additionally, data suggest that long-acting beta blockade may be superior to short-acting beta blockade.[61]

Recent clinical data also supports the notion that statins,[62-64] alpha-2 agonists for perioperative control of hypertension,[65,66] and calcium channel blockers[67] reduce perioperative cardiac morbidity and death. Aspirin is beneficial in reducing risks associated with CAD[68] and most surgeons continue aspirin during the perioperative period.[69] Coumadin should be discontinued five to seven days prior to abdominal aortic aneurysm repair. Use of either intravenous heparin or low molecular weight heparin to bridge patients undergoing a major aortic operation has been reviewed elsewhere.[70,71]

Beta blockers should be continuedin patients undergoing aneurysm surgery who are currently receiving beta blockers to treat angina, symptomatic arrhythmias, or hypertension.

Level of recommendation:	Strong
Quality of evidence:	High

Beta blockade is recommended for patients undergoing aneurysm repair in whom preoperative assessment identifies CAD or who are at high cardiac risk due to the presence of one or more clinical risk factors (i.e. CAD, CHF, CVA, DM, CRI).

Level of recommendation:	Strong
Quality of evidence:	Moderate

Pulmonary disease. Between 7% and 11% of patients with chronic obstructive pulmonary disease (COPD) have an aneurysm.[14] In a study of 4,404 men between 65 and 73 years of age, the prevalence of aortic aneurysms was 4.2%, but increased to 7.7% among men with COPD.[72] Although an accelerated rate of aneurysm expansion has been observed in patients treated with corticosteroids, the association between aneurysm and COPD has been largely attributed to accelerated elastin degradation caused by tobacco use. Indeed, smoking remains the most significant risk factor for aneurysm formation and has been implicated in

aneurysm development,[3,13] expansion,[55,73,74] and rupture.[75] Current smokers are over seven times more likely to have an aneurysm than nonsmokers with the duration of smoking being the most important variable.[13] Each year of smoking increases the relative risk of developing an aneurysm by 4%. After cessation of smoking, there is a slow decline in the risk of aneurysm occurrence.

Several studies have reported that COPD is an independent predictor of operative mortality after AAA repair.[4,76,77] However, a study of Veterans Administration patients found no significant correlation between the presence of COPD and increased operative mortality, though morbidity was notably higher.[78] Upchurch et al[79] have also demonstrated that abnormal preoperative pulmonary function tests and arterial blood gas values were not predictive of a poor outcome after aneurysm repair. However, failure to optimize COPD management was associated with increased morbidity and mortality.

Given these considerations, room air arterial blood gases and pulmonary function tests should be considered to assess the extent of COPD. If COPD is severe, formal pulmonary consultation is recommended for prediction of short- and long-term prognosis and optimization of medical therapy. In general, smoking cessation for at least two weeks prior to aneurysm repair can be beneficial and administration of pulmonary bronchodilators for at least two weeks prior to aneurysm repair is recommended for patients with a history of symptomatic COPD or abnormal pulmonary function studies. Finally, the diagnosis of an aneurysm can be a strong motivator for smoking cessation.[80] Numerous studies and several Cochrane reviews have assessed the efficacy of smoking cessation strategies. Nicotine replacement,[81] nortriptyline and bupropion,[82] and the combination of in-patient counseling coupled with at least one month of outpatient follow-up have proven beneficial for smoking cessation.[80]

Preoperative pulmonary function studies, including room air arterial blood gases, are suggested for patients with a history of symptomatic COPD, long-standing tobacco use, or inability to climb one flight of stairs.

Level of recommendation:	Weak
Quality of evidence:	Low

Smoking cessation for at least two weeks prior to aneurysm repair can be beneficial.

Level of recommendation:	Strong
Quality of evidence:	Low

Administration of pulmonary bronchodilators for at least two weeks prior to aneurysm repair is recommended for patients with a history of symptomatic COPD or abnormal pulmonary function studies.

Level of recommendation:	Strong
Quality of evidence:	Low

Renal impairment. Preoperative renal insufficiency is known to be a risk factor for a poor outcome after aneurysm repair[76,78,79] and should be evaluated and, if possible, corrected. In a review of 8,125 intact abdominal aneurysm repairs in the state of Michigan, the presence of renal failure was associated with a 41% mortality as compared with 6% for those patients without significant renal disease.[83] Likewise, severe renal dysfunction (creatinine $>$ 2.5 mg/dL) lead to increased mortality and morbidity among 342 patients undergoing EVAR with increased length of stay, intensive care unit (ICU) admission, and congestive heart failure.[84] A recent review has also observed that while many patients with renal insufficiency will have only a transient deterioration in renal function, mortality and morbidity, including the need for more intensive postoperative organ system support, appear to be increased.[85]

Surprisingly, there is little data regarding the specifics of management in patients undergoing AAA repair with chronic renal insufficiency (CRI) and whether management should be guided by serum creatinine, creatinine clearance, or glomerular filtration rate. In the perioperative period, relative or absolute changes in blood volume can cause renal injury. Thus, preoperative hydration is recommended to ensure euvolemia, especially for those patients with renal insufficiency. However, the optimal type, route, volume, and timing of hydration are not well defined. Likewise, given the ability of angiotensin-converting enzyme inhibitors and angiotensin receptor antagonists to induce efferent arteriole vasodilatation, these medications should be held the morning of surgery and restarted only after the patient is euvolemic.[86-88] Although administration of antioxidants, such as mannitol, during OSR has been advocated as renoprotective agents, a recent meta-analysis did not identify a significant benefit with mannitol use alone.[89] However, a small, prospective randomized trial has suggested that perioperative administration of multiple antioxidants (allopurinol, vitamin E, ascorbic acid, N-acetylcysteine, and mannitol) may lead to a beneficial increase in creatinine clearance after OSR.[90] Fenoldopam mesylate, a dopamine-1 receptor agonist, has also been studied as a renoprotective agent in both surgical and critically ill patients at risk for acute renal insufficiency. A recent meta-analysis of 16 randomized studies found that fenoldopam reduced the risk of acute kidney injury, renal replacement therapy, and in-hospital death.[91] Many of the beneficial trials administered fenoldopam at ~0.1 μg/kg/min beginning at the initiation of surgery. Gilbert et al[92] and Halpenny et al[93] have noted a renoprotective effect during and after infrarenal aortic cross-clamping among patients with normal or mild renal dysfunction.

Contrast-induced nephropathy (CIN) has been defined as a 25% elevation in serum creatinine or an absolute increase of 0.5 mg/dL (44 μmol/L) occurring two to seven days after contrast administration. Patients with a history of renal disease, diabetes, CHF, ejection fraction (EF) $<$40%, hypertension, anemia, advanced age, proteinuria, renal surgery, and gout are at increased risk for CIN. Multiple investigations and meta-analyses have sought to determine if prophylactic intravenous hydration, N-acetylcysteine (Mucomyst), or other agents can reduce the risk of CIN. Pre- and post procedure hydra-

tion with normal saline or sodium bicarbonate, to alkalinize renal tubular fluid and minimize tubular damage, appears to be beneficial.[94] However, whether all patients benefit equally from this treatment, as well as the optimal type, route, volume, and timing of hydration are not well defined. Fenoldapam, dopamine, theophylline, or calcium channel blockers do not appear to be beneficial in preventing CIN. The benefit of N-acetylcysteine in reducing CIN remains uncertain[95] and in a recent trial did not reduce CIN in patients undergoing EVAR.[96] Nonetheless, its use continues to be advocated for patients at increased risk of CIN.

Contrast agents with an osmolality of greater than 780 mOsm/kg display increased nephrotoxicity. Additional nephroprotection through further reduction in radiocontrast osmolality was suggested by a study comparing iohexol (Omnipaque, a low-osmolar agent; 600-800 mOsm/kg) with iodixanol (Visipaque, an iso-osmolar agent; 290 mOsm/kg).[97] However, the rates of CIN for iopamidol (Isovue-370, 796 mOsm/kg, non-ionic) are similar to iodixanol, which suggests that other physiochemical properties, apart from omolarity, are important determinants of CIN.[98] Likewise, several randomized trials of ionic and nonionic contrast agents have demonstrated no difference in CIN.[95]

In summary, patients at increased risk for CIN should receive intravenous hydration prior to (normal saline 1 ml/kg/h for six to 12 hours or D5W/sodium bicarbonate 154 meq/L, 3 mL/kg for one hour) and after (normal saline 1 ml/kg/h for six to 12 hours or D5W/sodium bicarbonate 154 meq/L, 1 mL/kg for six hours) EVAR. Fluid overload and metabolic alkalosis should be monitored. Periprocedural N-acetylcysteine and ascorbic acid may be of benefit.[95] Minimizing the volume of low-osmolar or iso-osmolar contrast agents administered during EVAR is recommended. Use of CO2 gas, as an alternate imaging agent may be considered.[99] Gadolinium-based contrast agents carry an increased risk of nephrogenic systemic fibrosis among patients with severe renal insufficiency.

Angiotensin-converting enzyme inhibitors and angiotensin receptor antagonists should be held the morning of surgery and restarted after the patient is euvolemic.

Level of recommendation:	Strong
Quality of evidence:	Moderate

Preoperative hydration is recommended for patients with renal insufficiency prior to aneurysm repair.

Level of recommendation:	Strong
Quality of evidence:	Moderate

Intraoperative diuresis using fuorosemide or mannitol is probably not beneficial in reducing the risk of postoperative renal insufficiency after aneurysm repair.

Level of recommendation:	Strong
Quality of evidence:	High

Pre- and post procedure hydration with normal saline or 5% dextrose/sodium bicarbonate is recommended for patients at increased risk of contrast induced nephropathy.

Level of recommendation:	Strong
Quality of evidence:	Moderate

Diabetes mellitus. Whether diabetes mellitus is truly an independent risk factor for morbidity or mortality after aortic surgery is controversial. Several small studies have shown that the risk of perioperative complications, but not death, is greater among diabetic patients.[100-102] A recent report demonstrated that diabetes did increase the risk of death and cardiovascular complications in Veterans Administration patients undergoing major vascular procedures.[103] However, subgroup analysis failed to demonstrate that diabetic patients undergoing OSR were at increased risk. In contrast, Leurs et al[104] investigated the influence of diabetes on outcome after EVAR among 6,017 patients enrolled in the European Collaborators on Stent Graft Techniques for Abdominal Aortic Aneurysm Repair (EUROSTAR) registry, of whom 731 (12%) had diabetes. A significantly higher risk of device-related complications was observed in diabetic patients (8% vs 6%, $P < .049$; odds ratio [OR]: 1.35). Likewise, early mortality was significantly higher in the diabetic population (13% vs 10%, $P < .039$; OR: 1.27). Insulin-controlled type 2 diabetic patients had significantly lower rates of endoleaks and secondary interventions than diet-controlled type 2 diabetics and nondiabetic patients. Survival at 48 months was similar among diabetic and non-diabetic patients. In summary, diabetes likely identifies patients with a variety of comorbidities that increase the risk of morbidity and mortality. Nonetheless, conventional glucose control during the perioperative period, with a target of 180 mg or less per deciliter (< 10.0 mmol/L), is recommended.

Hematologic disorders. Elevated levels of homocysteine (OR: 7.8; $P < .0001$), plasminogen activator inhibitor-1 (PAI-1) (OR: 3.2; $P < .0001$), and lipoprotein (a) (OR: 2.4; $P < .0001$) have been observed among patients with aortic aneurysms when compared to age and gender matched controls.[105] The role of these factors in aneurysm formation is unknown and the benefit of treatment in reducing aneurysm expansion uncertain. Anticardiolipin antibodies (ACA), MTHFR C677T polymorphism, prothrombin gene G20210A variant, and Factor V Leiden mutation have not been found more frequently in patients presenting with aortic aneurysms.

A number of studies have documented that even in the elective setting, anemia or a low hemoglobin level is associated with increased mortality following open AAA repair.[79,106] Ho and colleagues[106] documented that a hemoglobin level of less than 10.5 g/dL was an independent determinant of blood loss. A hematocrit less than 28% has also been associated with an increased incidence of postoperative MI in patients undergoing vascular surgery.[107] Therefore, we recommend perioperative blood transfusion if the preoperative hematocrit is less than 28%.

Standard surgical dogma suggests that an operation should not be performed unless the platelet count is greater than 20,000 to 40,000 platelets/μL. While the question of what platelet count specifically is too low to perform elective AAA repair and the impact this might have on mortality has not been addressed directly, it is recognized that the presence of an AAA does impact both platelet count as well as platelet function. In a prospective study comparing 105 patients with AAAs with 32 patients with symptomatic carotid disease, Milne and coauthors documented that a combination of a low platelet count and high glycocalicin levels suggests that there is increased platelet destruction in patients with AAAs, most likely due to activation within the aneurysm sac.[108] While not specifically addressing the impact on mortality, Ho and others did document in 129 patients undergoing elective AAA repair that a platelet count of 130,000 platelets/μL or less was associated with increased risk of bleeding.[106] In addition, it is well recognized that following aortic clamping, platelet sequestration and thrombocytopenia occur in the early postoperative period. Patients subsequently develop hyperfibrinogenaemia and thrombocytosis, which may persist for several weeks.[109] Finally, Matsumura and colleagues suggested that a lower preoperative platelet count was an independent predictor of two-year mortality among patients undergoing OSR and EVAR (P = .012).[110] Thus, further hematologic assessment is recommended if the preoperative platelet count is less than 130,000 platelets/μL.

Perioperative blood transfusion is recommended if the preoperative hematocrit is <28%.

Level of recommendation:	Weak
Quality of evidence:	Low

Further hematologic assessment is recommended if the preoperative platelet count is less than 130,000 platelets/μL.

Level of recommendation:	Weak
Quality of evidence:	Low

Circulating biomarkers for the presence and progression of aortic aneurysms. The identification of circulating biomarkers that indicate the presence or enlargement of AAA remains an area of active investigation. Such markers may assist in identifying new targets for pharmacotherapy and could be used for both diagnosis and monitoring response to therapy. Among a large number of biomarkers evaluated to date, fibrinogen, D-dimer, and IL-6 have been consistently associated with the presence of AAA in multiple cross-sectional case controlled studies.[111] However, none of these biomarkers have appropriate sensitivity or specificity to be used as a diagnostic test. A reliable marker of AAA progression has yet to be identified.

Genetic markers identifying risk of aortic aneurysm. Genetic abnormalities associated with AAA, include Ehlers-Danlos type IV (COL3A1), an autosomal dominant defect in the type-III collagen synthesis.[112] Of interest, while mutations in the type I collagen, alpha I (COL1A1) gene leads to osteogenesis imperfecta there is no evidence indicating these mutations nor those of the elastin gene are responsible for AAA.[113] Marfan syndrome is autosomal dominant disorder with variable penetrance caused by mutations in the fibrillin 1. The most common vascular complication includes aneurysmal degeneration and dissection of the ascending aorta. While arteriomegaly is common, isolated AAA that is unrelated to a prior aortic dissection, is uncommonly associated with Marfan syndrome.[113] Definitive data is lacking regarding the contribution of genetic mutations of other structural components of the connective tissue or genetically related alterations in post-translational protein modification, protein metabolism, or proteases and their inhibitors to familial AAA.

In a related area, population screening for single nucleotide polymorphisms (SNPs) to identify patients at risk for AAA have generated conflicting results. Although a number of genetic variants have been identified, few, if any, of these findings have been reproduced in more than one independent research group. A recent large study has suggested that a common sequence variant on 9p21, rs10757278-G, is associated with a 31% increased risk of abdominal aortic aneurysm.[114] Genome wide screening may serve to identify additional genetic markers for disease, which may further assist in targeting early ultrasound screening for populations at greatest risk.

Aneurysm imaging

Modalities for aneurysm imaging. Among asymptomatic patients, ultrasound detects the presence of an abdominal aortic aneurysm accurately, reproducibly, and at low cost. Sensitivity and specificity approach 100%, but in 1% to 3% of patients, the aorta cannot be visualized due to bowel gas or obesity.[115,116] Ultrasound is ideal for screening,[117] but is imprecise in measuring aneurysm size,[118-120] which is an important component of prognosis[121] and in the determination of aneurysm growth rate.[122] A growth rate of > 0.7 cm per six months or 1 cm per year has been suggested as a threshold for proceeding to surgery irrespective of aneurysm size.[51,123,124] However, it should be noted that the presumption that growth rate influences rupture risk independent of aneurysm size has not been confirmed by population based studies.[125]

CT is more reproducible than ultrasound, with more than 90% of re-measurements within 2 mm of the initial reading.[126] However, aneurysms measured by standard axial CT imaging are generally more than 2 mm larger in diameter than those measured by ultrasound. This likely reflects that the cross-section of the aorta obtained by axial CT imaging is not in the transverse plane and presumably yields an overestimate of aneurysm size. Overall, the advantages of portability and decreased expense have made ultrasound the preferred diagnostic technique for both aneurysm screening and surveillance.[126-128] Nevertheless, CT imaging remains the pri-

mary modality for operative planning, given the capacity of this technique to determine the extent and morphology of the aneurysm, as well as the presence of accessory or anomalous renal arteries and coexistent occlusive disease. It bears noting that plain abdominal films and aortography have low sensitivity in the detection of AAA. In the latter instance, the amount of aortic dilatation may be obscured by the presence of thrombus.

Ultrasound is readily available in most emergency departments and used with increasing facility by the emergency physicians to exclude a variety of causes of abdominal pain including urinary retention, pancreatitis, cholecystitis, and hydronephrosis, among other disorders. Several studies have reported high sensitivity (94% to 100%) and specificity (98% to 100%) in detecting non-ruptured aneurysms by ultrasound.[129,130] However, significant portions of the aorta may not be visualized in non-fasted patients and aortic rupture is not easily detected and up to half of aneurysm ruptures may be missed.[128,131] CT imaging for detection of symptomatic AAA has been found to have a sensitivity and specificity of 90% and 91%, respectively.[132] However, in a study of 653 patients with suspected AAA rupture, CT imaging had a sensitivity of 79% and a specificity of 77%. Only 40% of CT scans were performed with contrast, which presumably contributed to the diminished sensitivity and specificity noted in this report.[133]

Image derived criteria to predict risk of AAA rupture. The maximum AAA diameter remains the most widespread criterion to predict risk of AAA rupture. The adoption of maximum diameter as a measure of rupture risk was based, in part, on a retrospective review by Darling et al[134] of 24,000 consecutive, non-specific autopsies performed over a 23-year period, which identified 473 non-resected AAA, of which 118 were ruptured. Approximately 40% of AAA greater than 5 cm in diameter ruptured. Nonetheless, this same report highlights the limitation of aneurysm diameter as a predictor of rupture risk since 40% of AAA between 7 cm and 10 cm had not ruptured, while nearly 13% of patients with aneurysms smaller than 5 cm had ruptured. Thus, a variety of alternate parameters have been proposed as more sensitve predictors of rupture risk including AAA expansion rate[15,123,135,136] increase in intraluminal thrombus thickness,[137] wall stiffness,[138] wall tension,[139] and peak AAA wall stress.[140-142]

Most recent reports have emphasized the potential significance of aortic wall stress as a predictor of AAA rupture. Hall et al[139] suggested that a critical aortic wall stress exists above which rupture is imminent and that this stress can be predicted by the Law of Laplace and maximum AAA diameter. However, others[140-145] have demonstrated that stresses are highly dependent on AAA shape and cannot be adequately described by the Law of Laplace. Using patient-specific finite element (FE) simulations, Fillinger et al[140,141] have observed that peak wall stress for ruptured or symptomatic AAA were significantly greater than those electively repaired (46.8 ± 4.5 N/cm^2 vs 38.1 ± 1.3 N/cm^2).[142] Recently, Kleinstreuer and Li[146] have proposed a patient-specific "severity parameter" to estimate the risk of AAA rupture. This time-dependent parameter incorporates features of AAA geometry including size, shape, expansion rate, and thrombus, as well as diastolic pressure, peak AAA wall stress, and stiffness change. As an enlarging AAA is accompanied both by an increase in wall stress and a decrease in wall strength, recent efforts have also been directed to accurately map the pointwise distribution of AAA wall stress and strength as a more accurate determinant of rupture risk.[147] Further validation of these tools will be required before they can be applied with confidence in clinical practice.

Table V. Prevalence of small and medium AAAs among 73,451 US military veterans between 50 to 79 years†

Race	*Gender*	*Smoking status*	*AAA ≥3cm (%)*	*AAA ≥4cm (%)*
White	Male	Smoker	5.9	1.9
		Nonsmoker	1.9	0.4
White	Female	Smoker	1.9	0.3
		Nonsmoker	0.6	0
Black	Male	Smoker	3.2	0.8
		Nonsmoker	1.4	0.1

Adapted from Table II in Lederle FA, Johnson GR, Wilson SE, Chute EP, Littooy FN, Bandyk D, et al. Prevalence and associations of abdominal aortic aneurysm detected through screening. Aneurysm Detection and Management (ADAM) Veterans Affairs Cooperative Study Group. Ann Intern Med 1997;126:441-9.

†The prevalence of AAAs among black females was not reported.

Recommendations for aneurysm screening. Aneurysm screening efforts have been motivated by a desire to reduce AAA-related mortality and to prolong life expectancy. The incidence of AAA is less than one per 1000 for those adults younger than 60 years, peaks to approximately seven per 1000 among those in their mid 60s, and then decreases and remains at approximately three per 1000. It is estimated that 5% to 10% of older adult men have an AAA, but the majority of AAAs are small.[148] The prevalence of AAA is approximately six times lower in women than in men.[149] Overall, the probability of AAA in the general population is very low but, as noted earlier, is increased when certain risk factors are present. These include increasing age, male gender, white race, smoking, family history, history of other vascular aneurysms, hypertension, atherosclerotic diseases, including coronary artery, peripheral arterial, and cerebrovascular disease, and hypercholesterolemia.[51,150-159] The largest AAA screening program in the United States was conducted in asymptomatic veterans between 50 and 79 years old.[158] The prevalence of AAA in the most important risk groups is summarized in Table V. Two-thirds of aneurysms detected by ultrasound were found to be <4.0 cm. Of note, the prevalence of AAA is much higher among patients in the health care setting than in the general population. In one study, 9% of men aged 60 to 75 years with hypertension or coronary artery disease had an AAA >3.5 cm.[28]

Four randomized clinical trials that included 127,891 men and 9,342 women between the ages of 65 and 79 years have provided evidence that ultrasound screening is effective in reducing AAA-related mortality, but not all-cause mortality.[149,160-164] A recent follow-up report of the MASS study confirms that mortality benefits are maintained at seven years and cost-effectivness is well below accepted thresholds for interventions. Significantly, a trend toward a reduction in all-cause mortality was observed.[165] Screening of AAA in women has been more controversial. Given the small number of women among these four trials, a significant decrease in mortality from AAA (OR: 1.99; 95% confidence interval [CI] 0.36 to 10.88) or incidence of rupture (OR: 1.49; 95% CI 0.25 to 8.94) in women could not be identified.[166] However, others have claimed that screening of women for AAA is cost-effective.[167] Although the prevalence of AAA in women is lower than in men, rupture rate and life expectancy are higher. Studies have demonstrated that re-screening patients for AAA yields very few positive results, which suggests that a single ultrasound scan would be sufficient to screen patients 65 years of age or older.[168,169]

Despite these data, the availability of limited health care resources has motivated efforts to identify risk factors to be used in selective screening of high-risk groups. For example, a Canadian hospital-based case-control study found that if AAA risk was based on age, smoking, blood pressure, body mass index, history of heart disease, and serum high-density lipoprotein, 80% of AAA cases could be identified by studying 17% of patients.[170] Likewise, Akkersdijk et al have suggested that use of a risk factor score could identity 94% of men with AAA by screening approximately half of the male population.[171] Others have concluded, however, that while selective screening is feasible, approximately 25% of clinically significant AAAs may be missed.[172]

We recommend one-time ultrasound screening for AAA for all men at or older than age 65, or as early as age 55 for those with a family history of AAA. Ultrasound screening should be performed for women at or older than age 65 who have smoked or have a family history. This is in contrast to the US Preventive Services Task Force recommendation for one-time screening by ultrasonography for men aged 65 to 75, if a history of smoking exists.[173,174] Currently, Medicare offers an ultrasound screening benefit to men who have smoked at least 100 cigarettes during their life, and men and women with a family history of AAA as part of their Welcome to Medicare Physical Exam. This benefit became law on February 8, 2006, as the Screening Abdominal Aortic Aneurysms Very Efficiently (SAAAVE) Act.

One-time ultrasound screening for AAA is recommended for all men at or older than 65 years. Screening men as early as 55 years is appropriate for those with a family history of AAA.

Level of recommendation:	Strong
Quality of evidence:	High

One-time ultrasound screening for AAA is recommended for all women at or older than 65 years with a family history of AAA or who have smoked.

Level of recommendation:	Strong
Quality of evidence:	Moderate

Re-screening patients for AAA is not recommended if an initial ultrasound scan performed on patients 65 years of age or older demonstrates an aortic diameter of <2.6 cm.

Level of recommendation:	Strong
Quality of evidence:	Moderate

Recommendations for aneurysm surveillance. The optimal frequency surveillance of AAA has not been defined by randomized clinical study. Some authors have suggested that there is no need to follow aneurysms less than 3 cm in diameter given their low risk of rupture.[169,175] However, McCarthy et al[176] determined in a 12 year analysis of 1121 small aneurysms in 65 year-old men that 13.8% of aortas with an initial aortic diameter of 2.6 to 2.9 cm exceeded 5.5 cm at 10 years. Among patients with an aortic diameter between 3.0 and 3.4 cm, 2.1% had reached 5.5 cm at three years and of those with a diameter between 3.5 and 3.9 cm, within two years 10.5% exceeded 5.5 cm or required surgery and 1.4% had ruptured.

Two randomized controlled trials, the UK Small Aneurysm Trial[177] and the US-based Aneurysm Detection and Management (ADAM) Trial,[3] as well as a follow-up study of patients detected in the UK Multicenter Aneurysm Screening Study (MASS)[161] demonstrated that a policy of surveillance until aneurysm diameter exceeds 5.5 cm was safe and associated with a very low rate of rupture (~1%/year). While AAA size was defined by the maximum external aortic diameter in all trials, the surveillance frequency differed among these studies. The MASS trial scanned patients with diameters between 4.5 and 5.0 cm at three-month intervals, whereas the UK Small Aneurysm Trial used six-month intervals for these patients. Patients in the ADAM study with AAA diameter of 5.0 to 5.5 cm were rescreened every six months, compared with every three months in the UK. In an analysis of expansion rates among 1743 patients over a mean interval of 1.9 years, Brady et al[178] noted that AAA growth rate increased with aneurysm size and among current smokers, was lower in those with low ankle-brachial index and diabetes, and unaffected by lipids and blood pressure. The authors estimated that for a patient with a 4.5 cm AAA, the risk of enlargement to 5.5 cm was less 1% during a 12-month interval.

In summary, we recommend follow-up surveillance imaging at 12-month intervals for patients with an AAA of 3.5 to 4.4 cm in diamater. Surveillance imaging at six-month intervals is recommended for those patients with an AAA diameter between 4.5 and 5.4 cm. For otherwise healthy patients, follow-up imaging is recommended at three years for those between 3.0 and 3.4 cm in diameter and at five years for aneurysms between 2.6 and 2.9 cm. It

bears noting that these recommendations are based upon maximum external aortic diameter.

Surveillance imaging at 12-month intervals is recommended for patients with an AAA of 3.5 to 4.4 cm in maximum diameter.

Level of recommendation:	Strong
Quality of evidence:	Low

Surveillance imaging at six-month intervals is recommended for those patients with an AAA between 4.5 and 5.4 cm in maximum diameter.

Level of recommendation:	Strong
Quality of evidence:	Low

Follow-up imaging at three years is recommended for those patients with an AAA between 3.0 and 3.4 cm in maximum diameter.

Level of recommendation:	Strong
Quality of evidence:	Low

Follow-up imaging at five-year intervals is recommended for patients whose maximum aortic diameter is between 2.6 and 2.9 cm.

Level of recommendation:	Weak
Quality of evidence:	Low

Recommendations for imaging patients with a symptomatic aneurysm. In patients with abdominal or back pain, we recommend ultrasound imaging to determine if an AAA is present and to identify other causes of abdominal pain or back pain. If an aneurysm is detected, the patient should have a CT scan to exclude rupture and be referred to a vascular surgeon. In order to avoid delay, a CT scan is the preferred initial test in patients with recent or severe symptoms or a pulsatile epigastric mass or significant risk factors for AAA. If AAA rupture is suspected and the patient is hemodynamically stable, an emergency CT scan with contrast may be considered for preoperative planning during the mobilization of the surgical team.

A CT scan is recommended to evaluate patients that present with recent onset of abdominal or back pain, particularly in the presence of a pulsatile epigastric mass or significant risk factors for AAA.

Level of recommendation:	Strong
Quality of evidence:	Moderate

TREATMENT OF THE PATIENT WITH AN AAA

The decision to treat

Patients that present with an AAA and abdominal or back pain, even of an atypical nature, are at increased risk of rupture and intervention is recommended. Should aneurysm rupture occur, more than half of patients die prior to hospitalization. Of those who reach the operating suite, the outcome is dependent on the presenting clinical condition, but typically carries a mortality of approximately 50%. For those who present with an asymptomatic AAA, management is dependant on the size of the aneurysm.

There is general agreement that small fusiform aneurysms, less than 4.0 cm maximum diameter, are at low risk of rupture and should be monitored and a fusiform aneurysm greater than 5.4 cm in maximum diameter should be repaired in a healthy patient. Elective repair is also reasonable for patients that present with a sacular aneurysm. Debate remains for patients presenting with AAAs between 4.0 cm and 5.4 cm regarding the most appropriate role for either immediate treatment or surveillance and selective repair for those aneurysms that subsequently enlarge beyond 5.4 cm. In both the United Kingdom Small Aneurysm Trial (UKSAT)[179] and Aneurysm Detection and Management (ADAM) Trial,[180] the 30-day operative mortality in the immediate surgery group (5.5% UKSAT, 2.1% ADAM) led to an early disadvantage in survival. Investigators found no statistically significant difference in long-term survival between the immediate surgery and surveillance groups.

Potential benefits of early surgery were noted in both the UKSAT and ADAM study for selected subgroups. In the UKSAT, the estimated adjusted hazard ratios were in the direction of greater benefit of early surgery for younger patients and those with larger aneurysms but statistical significance was not demonstrated.[179] Likewise, long-term results from the ADAM study suggested a similar effect.[180] Neither study was designed or powered to examine the question of whether immediate surgery might be harmful for patients with aneurysms between 4.0 cm and 4.4 cm, but beneficial for patients with aneurysms between 5.0 cm and 5.4 cm. Moreover, differential effects for older or younger cohorts, female patients, or those of exceptional physiologic fitness could not be addressed. Uncertainty regarding the potential benefit of early repair in selected patients with small AAA is further magnified by the demonstration that EVAR is associated with reduced perioperative mortality. The Comparison of surveillance vs endografting for small aneurysm repair (CAESAR)[181] and Positive impact of endovascular options for treating aneurysm early (PIVOTAL) trials compare immediate EVAR with surveillance and selective EVAR, but neither trial was designed to determine whether immediate EVAR might be beneficial or harmful for specific AAA size ranges or age subgroups.

Patients need to appreciate the therapeutic uncertainty for AAA in the range of 4.0 cm to 5.4 cm. Ultimately, all recommendations are individualized. At present, surveillance with selective repair is most appropriate for older male patients with significant co-moribidities. Young, healthy patients, and especially women, with AAA between 5.0 cm and 5.4 cm may benefit from early repair.

Repair is recommended for patients that present with an AAA and abdominal or back pain.

Level of recommendation:	Strong
Quality of evidence:	High

Elective repair is recommended for patients that present with a fusiform AAA $\geq$5.5 cm in maximum diameter, in the absence of significant co-morbidities.

Level of recommendation:	Strong
Quality of evidence:	High

Elective repair is suggested for patients that present with a saccular aneurysm.

Level of recommendation:	Weak
Quality of evidence:	Low

Surveillance is recommended for most patients with a fusiform AAA in the range of 4.0 cm to 5.4 cm in maximum diameter.

Level of recommendation:	Strong
Quality of evidence:	Moderate

Young, healthy patients, and especially women, with AAA between 5.0 cm and 5.4 cm in maximum diameter may benefit from repair.

Level of recommendation:	Weak
Quality of evidence:	Low

The benefit of repairing a small aneurysm is uncertain in patients who will require chemotherapy, radiation therapy, or solid organ transplantation.

Level of recommendation:	Weak
Quality of evidence:	Low

Medical management during the period of AAA surveillance

A number of approaches have been proposed to prevent progression of aneurysmal disease during the period of aneurysm surveillance including hemodynamic control, as well as inhibition of inflammation and protease activity.[182] Several studies have demonstrated that tobacco use is associated with an increased rate of aneurysm expansion and smoking cessation is likely the most important recommendation that can be made to a patient with AAA.[74,156,178] Evidence from two large randomized trials indicates that propranolol does not inhibit aneurysm expansion.[53,56] However, these results were compromised by low compliance, with 20% to 40% of patients discontinuing propanolol during the study period. Small observational studies suggest that 3-hydroxy-3-methylglutaryl (HMG) coenzyme A reductase inhibitors (statins) may inhibit aneurysm expansion.[183,184] Animal studies have demonstrated that angiotensin-converting enzyme (ACE) inhibitors or Losartan, an angiotensin receptor blocker decrease the rate of AAA expansion. Hackam et al[185] recently reported in an analysis of 15,326 patients in linked administrative databases that use of ACE inhibitors within the prior three to 12 months was less frequent among those patients with AAA rupture. Beta-blockers, lipid-lowering agents, and angiotensin receptor blockers showed no relationship to rupture. Further, Lederle and Taylor[186] observed an increased risk of aneurysm rupture among those patients who discontinued ACE inhibitors within the past three to 12 months. Lindholt et al[187] have suggested that serological evidence of a *C. pneumoniae* infection is associated with an increased rate of AAA expansion and small randomized trial of approximately 100 patients demonstrated that a one month course of roxithromycin decreased the rate of aneurysm expansion.[188] A number of studies have suggested that doxycycline can inhibit matrix metalloproteases in plasma and aneurysm tissue.[189,190] Morosin et al[191] randomized 32 patients with AAA to doxycycline (150 mg/d) or placebo for three months. Patients were followed for 18 months. *C. pneumoniae* titers were not affected by doxycycline treatment. A trend toward a lower AAA growth rate was observed in the doxycycline-treated group. Additional studies are needed to clarify the potential role of doxycycline, roxithromycin, and statin therapy in the progression of aneurysmal disease.

In summary, during the surveillance period, patients should be counseled to cease smoking if tobacco products are being utilized. Patients should be encouraged to seek appropriate management for hypertension, hyperlipidemia, diabetes, and other atherosclerotic risk factors. A statin and ACE inhibitor should be initiated given their broad potential benefits and acceptable safety profile. Insufficient data exists to recommend use of doxycycline or roxithromycin. Patients should be counseled that moderate physical activity does not precipitate rupture and may limit AAA growth rate.[192] Screening of family members should be recommended.

Smoking cessation is recommended to reduce the risk of AAA growth and rupture.

Level of recommendation:	Strong
Quality of evidence:	High

Statins may be considered to reduce the risk of AAA growth.

Level of recommendation:	Weak
Quality of evidence:	Low

Table VI. Transperitoneal or retroperitoneal approach for open abdominal aortic aneurysm repair

	Transperitoneal	*Retroperitoneal*
Advantages	• Most rapid, greatest flexibility • Provides widest access • Enables evaluation of intra-abdominal pathology	• Avoids hostile abdomen • Facilitates exposure and control for juxta-pararenal AAA • Potential for less ileus, other physiologic stress • Obesity • Inflammatory AAA/horseshoe kidney
Disadvantages	• Longer ileus • Potential for greater fluid losses • Difficulty with exposure and control for juxta – or pararenal AAA	• Poor access to right renal and iliac arteries • Cannot evaluate intra-abdominal pathology • Longer to open and close • More flank bulges, chronic wound pain

AAA, Abdominal aortic aneurysm.

Doxycycline, roxithromycin, ACE inhibitors, and angiotensin receptor blockers are of uncertain benefit in reducing the risk of AAA expansion and rupture.

Level of recommendation:	Weak
Quality of evidence:	Low

The use of beta blockers to reduce the risk of AAA expansion and rupture is not recommended.

Level of recommendation:	Strong
Quality of evidence:	Moderate

Screening for AAA is recommended for first degree relatives of patients presenting with an AAA.

Level of recommendation:	Strong
Quality of evidence:	High

Open surgery

Once the need for AAA repair has been determined, a choice will need to be made between conventional OSR and EVAR. The basic technique of endoaneurysmorrhaphy first popularized by Creech[193] emphasizes minimal dissection and intrasaccular ligation of lumbar artery branches, with suture attachment of the prosthetic graft to the proximal and distal aspects of the aneurysm. This, together with steady advances in graft and suture materials, surgical experience, and perioperative anesthesia and critical care, have made conventional OSR a very successful and durable correction for aortoiliac aneurysmal disease.

Operative approach. For standard open operative repair, an anterior transperitoneal (TP) or left-flank retroperitoneal (RP) approach may be employed. Each method has its own potential advantages and limitations (Table VI). In addition, the individual training and personal experience of the surgeon may also influence decision-making in this regard.

A TP approach via a generous midline abdominal incision is most commonly employed for infrarenal AAA repair, and most familiar to surgeons. This exposure can be performed rapidly, provides wide intra-abdominal access and maximal flexibility, and enables evaluation of other possible intra-abdominal pathology. In contrast to the typical midline vertical incision, a few surgeons recommend a transverse incision just above or below the umbilicus, as they believe such incisions may result in less postoperative pulmonary complications or a lower incidence of incisional hernias in long-term follow-up. However, such claims have never been clearly established and transverse incisions are infrequently utilized.

Advocates of a RP approach claim various physiologic benefits, including reductions in fluid losses, cardiac stress, postoperative pulmonary complications, and severity of ileus. All of these potential benefits are felt to lead to a reduction in ICU and hospital length-of-stay, diminished costs, and quicker recovery. However, several randomized prospective studies examining these possible advantages of a RP approach have reached differing conclusions. Cambria and colleagues[194] did not detect any of the physiologic benefits observed by Sicard and co-workers[195] other than a shorter duration of postoperative ileus and slightly earlier resumption of oral intake. A more recent randomized comparison of TP and RP approaches for infrarenal aortic surgery by Sieunarne et al[196] also failed to document any significant physiologic or other perioperative outcome differences, but did note a higher evidence of long-term wound problems, including incisional pain, bulges, and hernias in the RP group.[196] Such conflicting results suggest that the choice of surgical approach is best individualized in each patient, based upon individual anatomic and clinical circumstances. A RP approach may be preferable for patients with a "hostile abdomen" secondary to multiple prior intra-abdominal operations, a history of irradiation, presence of abdominal hernias, stomas, or marked obesity. Similarly, most vascular surgeons agree that a RP incision is preferred for repair of inflammatory aneurysms or AAA associated with a horseshoe kidney.[197,198]

In current practice, perhaps the clearest indication for a RP approach is extension of aneurysmal disease to the juxtarenal or visceral aortic segment. Exposure and control of the aorta in this region, as well as the left renal and visceral branches, are facilitated by a left lateral RP approach and opening of the left diaphragmatic crura. Use of an extended posterolateral RP approach with the left kidney mobilized anteriorly has the additional advantage of transposing the left renal vein anteriorly, thereby avoiding the possible need to divide the left renal vein to gain adequate exposure of the juxtarenal or suprarenal aorta which may be required on occasion with an anterior TP approach. While some authorities feel the left renal vein may be divided without consequence as long as collateral branches are preserved, several other reports document some adverse impact on renal function.[77,199] Use of an RP approach avoids this dilemma. The ease and flexibility of exposure and clamping of the suprarenal or supravisceral aorta via this approach has even led some investigators to advocate use of a high extended posterolateral approach for emergency repair of ruptured aneurysms.[200] Good results have been achieved, without the potential consequences of entering the area of retroperitoneal hematoma and loss of the tamponade effect which has long been a concern expressed by many surgeons for such an approach.

Because many infrarenal AAA with favorable neck anatomy are currently repaired with endovascular stent grafts, in contemporary practice, all vascular surgeons recognize that the technical complexity and challenges of OSR have increased since only aneurysms with adverse neck anatomy not felt suitable for EVAR undergo standard OSR.[201-203] It is clear, therefore, that a vascular surgeon should be familiar and experienced with both a TP and RP approach, utilizing each as determined by patient anatomy and clinical need.

A retroperitoneal approach should be considered for patients in which aneurysmal disease extends to the juxtarenal and/or visceral aortic segment, or in the presence of an inflammatory aneurysm, horseshoe kidney, or hostile abdomen.

Level of recommendation:	Strong
Quality of evidence:	Moderate

Use of a retroperitoneal exposure or a transperitoneal approach with a transverse abdominal incision may be considered for patients with significant pulmonary disease requiring OSR.

Level of recommendation:	Weak
Quality of evidence:	Low

Division of the left renal vein may be considered to gain suprarenal aortic exposure.

Level of recommendation:	Strong
Quality of evidence:	High

Aortic clamping. For standard infrarenal AAA, proximal aortic clamp placement is below the renal arteries, but should be as close to the renal vessels as feasible in order to perform the proximal graft anastomosis as high as possible. This is emphasized in order to minimize the incidence of late development of recurrent aneurysmal degeneration above the graft, which may occur if the anastomosis is performed at a low level on the aorta.

If an anterior TP approach has been employed, it may be necessary to mobilize the crossing left renal vein and retract it superiorly to allow high proximal clamp application. This may require division of its gonadal and posterior lumbar branches to avoid tearing and injury to the vein. Complete division of the vein is best avoided in view of its possible adverse impact on postoperative renal dysfunction. If the renal vein is to be divided, the decision should be made prior to division of the gonadal, adrenal, and lumbar branches, which provide collateral flow from the left kidney. Alternatively, end-to-end anastomosis of the divided renal vein may be considered.

The proximal extent of aneurysmal disease and quality of the aorta at the anticipated clamp site are best determined by careful examination of a high-quality fine-cut abdominal CT scan, both with and without contrast to allow accurate identification of aortic wall calcification and the extent of atheromatous debris and the length and diameter of the aneurysm neck. While there are essentially no anatomic constraints to OSR as exist for EVAR, careful preoperative evaluation of aortic and aneurysmal anatomy are nonetheless important aspects of preoperative planning prior to operation.

If extensive calcification or intraluminal atheromatous disease is noted, or the aneurysm extends very close to the renal arteries, a decision to clamp at a higher level becomes advisable to minimize the risk of atheromatous embolization into the renal arteries or clamp injury to the aortic wall. Whether the clamp is placed suprarenal but below the visceral arteries, between the superior mesenteric (SMA) artery and celiac axis, or at a supraceliac level is dependent upon anatomy and aortic quality at these varying levels. Often there is little room between the renal arteries and SMA, or between the two visceral branches, to allow safe dissection and clamp application. If pararenal atheromatous disease or significant calcification is observed on the CT scan, it will likely be preferable to go to a supraceliac level to minimize the risk of renal embolization or flow obstruction, despite the possibility of increased hemodynamic and cardiac stress.[204,205] Although suprarenal clamping is associated with an increased risk of postoperative decrease in renal function and overall adverse events, 30-day mortality is comparable to those patients repaired with infrarenal crossclamping.[206,207]

It is usually recommended that the proximal clamp be applied first, in order to minimize the occurrence of atheromatous embolization. This has never been truly studied or established, however, it would seem reasonable to clamp the least diseased arterial segment first, whether proximal or distal, in order to accomplish this objective. Distal clamping is almost always at the iliac level, since aneurysmal disease usually extends close to the aortic bifurcation even in patients suitable for a straight tube graft reconstruction. As with determination of the best site for proximal clamping, the surgeon should evaluate the preoperative CT scan, as well as palpate the iliac arteries at surgery, to determine the best site and method of distal clamping. If severely calcified, intraluminal control of the iliac arteries with balloon catheters will be a safer and more effective method of control. Use of soft-jawed clamps may also be considered. Because iliac plaque and disease is most often posterior, clamping in a transverse plane may also be safer than the typical anteroposterior (AP) application.

Irrespective of clamp location and method, systemic heparinization (75-100U/kg) is utilized by almost all vascular surgeons for elective AAA repair. In the circumstances of a ruptured aneurysm or other unusual situations, heparin may be omitted, with vigorous flushing of the graft prior to restoring blood flow, or limited amounts of heparinized saline may be instilled directly into distal vessels after placement of the proximal clamp. For patients with a history of heparin-induced thrombocytopenia, a thrombin inhibitor (eg, Bivalirudin, Argatroban) is recommended at the time of aortic clamping.

A high-quality preoperative CT scan is recommended to determine the optimal site of proximal aortic clamping based upon the extent of aneurysmal disease and quality of the aorta.

Level of recommendation:	Strong
Quality of evidence:	High

A transbrachial or transfemoral balloon for aortic control may be considered prior to anesthetic induction for patients with a ruptured aortic aneurysm.

Level of recommendation:	Weak
Quality of evidence:	Low

A thrombin inhibitor (eg, Bivalirudin, Argatroban) is recommended at the time of aortic clamping for patients with a history of heparin-induced thrombocytopenia.

Level of recommendation:	Strong
Quality of evidence:	Moderate

Type and configuration of the graft. Excellent patency and long-term results have been achieved with a wide variety of prosthetic grafts utilized for open AAA repair. Numerous modifications of graft material, such as polyester or polytetrafluoroethylene (PTFE), methods of fabrication, including knitted or woven, external or double velour, high

or low porosity, have been devised in the hope of improving performance and other characteristics such as ease-of-handling, durability, healing, resistance to infection, or reduced dilatation over time. One may conclude, however, that patency and other late outcomes appear to be equivalent for grafts placed in the aortic position. Hence surgeon preference and cost are usually the dominant determinants in aortic graft choice.[208-210] Currently, "zero porosity" polyester grafts, rendered impermeable by various biologic coatings, such as collagen, gelatin, or albumin, are the most popular and widely employed. Although more expensive, their expediency, intraoperative time savings, and reduced blood loss attributable to the lack of preclotting requirements more than offset such costs.

Aorto-aortic "straight tube" grafts are generally regarded as preferable to bifurcated prostheses due to a shortened operative time, reduced blood loss, and less need for dissection with attendant risk of injury to adjacent structures such as the ureter, iliac veins, or autonomic nerve networks. In most series of elective open aortic graft repair, tube grafts are utilized in 40% to 50% of cases, although the proportion of straight tube or bifurcated configuration varies in the literature range between 0% and 85%.[211] In the Canadian Aneurysm Trial, for example, 38.5% of AAA grafts were tube grafts, aortobiiliac in 30.7%, iliac-femoral distal anastomoses in 6.5%, and aortobifemoral in 24.3%.[212] Certainly bifurcated grafts are advisable if clinically significant concomitant iliac aneurysms (>2.0 cm to 2.4 cm) are present. Prior reports have suggested that iliac aneurysms may be present in 20% to 30% of patients with infrarenal AAA.[212,213] If coexistent symptomatic aortoiliac occlusive disease exists with limiting claudication, both aneurysmal and obliterative disease can be corrected with an aortobifemoral graft. However, a somewhat higher incidence of wound infection, graft limb thrombosis, and anastomotic aneurysm has been reported if the graft is extended to the groin.[212]

If significant iliac aneurysmal or occlusive disease is not present at the time of repair, many series have documented a low incidence of late disease progression necessitating reintervention.[211,214] Thus, the surgeon can be confident in placement of a tube graft at the time of initial repair. In addition, current increased endovascular options for stent grafting and/or balloon angioplasty/stenting offers assurance that any late problems developing in the iliac arteries that may require reintervention can likely be managed by catheter-based therapies.

Benefit from the prophylactic use of rifampicin impregnation of prosthetic graft material in preventing early or late graft infection has not been identified in three multicenter studies including 3,379 patients. In all cases, gelatin-coated Dacron grafts were soaked in 1 mg/mL of rifampicin for 15 minutes before insertion. The Joint Vascular Research Group trial[215,216] included patients undergoing extra-anatomical grafts (axillofemoral, femorofemoral, and iliofemoral crossover grafts) whereas the Italian Investigators Group[217] included patients undergoing mono-, bifemoral, or iliofemoral grafts.

Straight tube grafts are recommended for OSR of AAA in the absence of significant disease of the iliac arteries.

Level of recommendation:	Strong
Quality of evidence:	High

The proximal aortic anastomosis should be performed as close to the renal arteries as possible.

Level of recommendation:	Strong
Quality of evidence:	High

It is recommended that all portions of an aortic graft should be excluded from direct contact with the intestinal contents of the peritoneal cavity.

Level of recommendation:	Strong
Quality of evidence:	High

Maintenance of pelvic circulation. Blood supply to the left colon and pelvic organs is comprised of a complex network, with contributions from the SMA to the inferior mesenteric artery (IMA) via the meandering mesenteric artery and marginal artery of Drummond, the internal iliac or hypogastric arteries, and collateral vessels from circumflex branches of the common and deep femoral arteries. Failure to properly maintain pelvic blood flow during aneurysm repair may lead to a variety of problems, including postoperative sexual dysfunction, troublesome hip and buttock claudication, or, more significantly, colon ischemia or infarction and possible spinal cord ischemia or infarction with paraparesis or paraplegia. Thus, the status of pelvic blood supply must be assessed preoperatively, and steps taken during surgery to maintain adequate colonic and pelvic circulation.

Although multifactorial in origin, ligation of a patent IMA is the most commonly noted risk factor for development of colon ischemia in many series.[218] While the IMA may already be occluded at the time of surgery in 40% to 50% of patients secondary to concomitant occlusive disease or intrasaccular mural thrombus, the IMA is patent in over one-half of patients.[219] Whether or not to reimplant a patent IMA into the aortic graft remains controversial. Some authors have recommended frequent or even routine reimplantation of a patent IMA,[219,220] but its value has not been clearly established.[221,222] A prospective randomized trial examining this question found no statistically significant reduction of perioperative colon ischemia with reattachment and preservation, although the data did suggest that older patients or those with increased intraoperative blood loss might benefit from reimplantation.[223]

It seems reasonable to conclude that IMA reimplantation should be considered in the presence of associated celiac or SMA occlusive disease, an enlarged meandering mesenteric artery, a history of prior colon resection, inability to preserve hypogastric perfusion, poor IMA backbleeding, poor Doppler flow in colonic vessels, or should the colon appear ischemic. Prediction and prevention of colon ischemia remain of paramount importance, which carries up to a 50% mortality if transmural colonic infarction occurs. The surgeon should first ascertain the status of the

IMA by careful examination of the preoperative CTA. At the time of operation, the vessel can be readily interrogated with a sterile hand-held Doppler when the aneurysm is first exposed. If a patent IMA is present, it should not be ligated or divided, but rather controlled with a bulldog clamp or silastic loop. If the vessel is large, particularly if co-existent celiac or SMA occlusive disease is present or an enlarged meandering mesenteric collateral network is noted, preservation and reimplantation is warranted. A history of prior colon resection may be presumed to have compromised SMA to IMA territory collateral blood flow, and may also suggest the advisability of replanting a patent IMA. Inability to restore preexistent hypogastric blood flow may be another impetus to reimplant a patent IMA. Finally, poor back bleeding from a patent vessel once graft reconstruction has been completed and distal blood flow restored suggests inadequate collateral flow and the likely need to reimplant the IMA. This may be associated with an ischemic appearance of the colon and abnormal or absent Doppler signals in the colonic arcade or anti-mesenteric bowel wall. If clamp time has been prolonged, blood loss substantial, or periods of significant intraoperative hypotension have been present, as in the case of a repair of a ruptured aneurysm, the threshold to proceed with reimplantation should be lower. In the Canadian Aneurysm Study, the IMA was reimplanted in 4.8% of cases, but was associated with an increased incidence of postoperative bleeding.[77] If IMA reimplantation is elected, most surgeons accomplish this by preservation of a small island of aneurysm wall tissue around the orifice of the vessel and suturing of this "button" of tissue into a small opening in the graft as a Carrel patch.

It has long been accepted as a basic principle of aortic reconstruction that blood flow to at least one internal iliac artery should be maintained. Failure to achieve this has usually been felt to result in problems with erectile dysfunction, symptomatic hip and buttock claudication, or occasionally colon ischemia, buttock necrosis, or spinal cord (cauda equina) ischemia. For example, in the Canadian Aneurysm Study, the incidence of clinically apparent colon ischemia was 0.3% when hypogastric flow was preserved to at least one side, while it was 2.6% when flow was lost bilaterally. The one case of paraplegia in this report occurred in a patient in whom pelvic blood flow was not maintained.[212]

Recently, the necessity to preserve perfusion of a least one hypogastric artery has been questioned as endovascular stent graft aneurysm repair has become more widely utilized. Mehta and associates reported no mortality or significant morbidity in a series of 48 patients requiring interruption of flow to both hypogastric arteries as part of endovascular (n = 32) or open surgical (n = 16) repair of aortoiliac aneurysms;[224] however, they noted buttock claudication symptoms in 42% and new onset of erectile dysfunction in 14%.

While firm conclusions on this topic cannot be reached with certainty, it seems prudent to make every effort to preserve hypogastric perfusion on at least one side. During OSR, this can usually be achieved, even in patients with fairly extensive iliac aneurysmal disease, by distal anastomosis at the iliac bifurcation, or by end-to-side anastomosis to the external iliac artery and preservation of retrograde perfusion to the internal iliac. On rare occasions, a separate short "side arm" graft can be constructed directly to the internal iliac to maintain flow. Similarly, when preservation of at least one hypogastric artery during EVAR is difficult, some investigators recommend concomitant surgical bypass from the external to internal iliac prior to or at the time of endovascular repair to maintain pelvic circulation.[225,226]

Reimplantation of a patent inferior mesenteric artery (IMA) should be considered under circumstances that suggest an increased risk of colonic ischemia.

Level of recommendation:	Strong
Quality of evidence:	High

It is recommended that blood flow be preserved to at least one hypogastric artery in the course of OSR or EVAR.

Level of recommendation:	Strong
Quality of evidence:	High

Management of associated intra-abdominal non-vascular disease. With increasing use of preoperative CT scans and other imaging modalities for preoperative evaluation of aortic aneurysms, associated intra-abdominal pathology may be detected. Conversely, it is quite common for an unsuspected aneurysm to be initially detected during radiologic studies to evaluate other intra-abdominal conditions. Unsuspected intra-abdominal pathologies may be found at the time of abdominal exploration during OSR. In all of these scenarios, there is considerable uncertainty about the timing of AAA repair by OSR or EVAR and management of other intra-abdominal pathology. In many instances of combined disease, EVAR is particularly attractive, allowing quicker recovery from aneurysm repair and facilitating earlier correction of the associated condition.

General guidelines for decision making in these circumstances emphasize treatment of the most life-threatening or symptomatic problem first; simultaneous combined repair of both lesions is generally avoided, particularly in circumstances where bacterial contamination (eg, gastrointestinal, genitourinary) of the prosthetic aortic graft might occur with potentially devastating consequences.

In regard to colonic malignancies, resection of the tumor or diverting colostomy should be performed only for instances of impending obstruction, significant bleeding, or perforation. Otherwise, isolated aneurysm repair takes precedence, with subsequent colonic operation in four to six weeks. Initial colonic resection prior to AAA repair is generally unwise in elective circumstances, as any septic complications after bowel resection may delay aneurysm repair for many months.[227] In contrast, simultaneous repair of "clean" lesions, such as renal or ovarian tumors, may be

considered and successful experiences have been reported.[228] Although gallstones may be found in 5% to 15% of patients undergoing AAA repair, post-operative cholecystitis following OSR or EVAR is rare.[229] Thus, the motivation for concomitant cholecystectomy is limited, especially given the potential for direct contamination of the graft should bile leakage or spillage occur.

Concomitant surgical repair of an AAA and co-existent intra-abdominal pathology may be considered in highly selective circumstances.

Level of recommendation:	Strong
Quality of evidence:	High

Unusual aneurysms

Juxtarenal aneurysms. Aneurysms that extend close to the origins of the renal arteries and thereby require suprarenal aortic clamping to allow repair are termed juxtarenal aneurysms. Because endovascular stent graft repair is performed in 40% to 70% of patients with standard infrarenal AAA in current practice, such juxtarenal aneurysms represent an increasing percentage of open surgical repairs.[202] Due to the technical challenges of adequate surgical exposure and aortic control, as well as a period of obligatory renal and/or visceral ischemia and increased cardiac stress as a consequence of suprarenal or supraceliac clamping, repair of juxtarenal AAA is more complex and associated with increased perioperative morbidity and mortality risk.[202,230,231] Thus, because of the increased risk of repair, many vascular surgeons feel that operation may not be indicated until the aneurysm reaches a greater maximum diameter than the threshold size for a standard infrarenal AAA.

When extension of aneurysmal disease to the juxtarenal level is recognized from the preoperative CT scan, modifications of operative strategies regarding approach and clamp placement must be considered. Most surgeons prefer a left flank extended retroperitoneal approach, but others recommend an anterior approach with transcrural supraceliac clamping or medial visceral rotation.[202,230,231] The exact site of proximal aortic clamp placement is determined by anatomy with principal emphasis on extent of aneurysmal and associated aortic atherosclerotic disease. The proximal graft anastomosis is then performed just below the renal artery origins. By definition, renal artery bypass grafting or reimplantation is not required in juxtarenal AAA repair unless mandated by co-existent renal artery disease.

Inflammatory aneurysms. Inflammatory AAAs are characterized by a markedly thickened aortic wall and dense perianeurysmal fibrosis. As a consequence of this fibrotic process, neighboring structures such as the duodenum, left renal vein, and ureters are often densely adherent to the aneurysm sac, making open surgical repair of such aneurysms more challenging and possibly associated with a high incidence of perioperative injuries or complications.[232] Recognition of an inflammatory aneurysm is usually possible from the preoperative scan, which reveals the markedly thickened aneurysmal wall with a "halo" of soft tissue around the aneurysm, which enhances with administration of intravenous contrast.

If an inflammatory aneurysm is suspected, EVAR is a preferred approach. Otherwise, several principles should be considered if an inflammatory AAA is treated by OSR. Should the perianeurysmal process obstruct the ureters, preoperative insertion of ureteral stents may be helpful in alleviating hydronephrosis, as well as in facilitating identification of the ureters, thereby minimizing the danger of ureteral injury during operative repair. In most cases, repair of the aneurysm leads to regression of the perianeurysmal fibrosis and relief of ureteral obstruction. Most authors urge caution in attempting ureterolysis at the time of surgery in order to relieve obstruction due to the significant risk of ureteral injury and leakage. Many vascular surgeons favor a retroperitoneal approach for repair of inflammatory aneurysms as the chance of injuring adjacent organs or structures is reduced and the flexibility of proximal clamp placement enhanced.[198,233]

In patients with inflammatory aneurysms who have otherwise suitable anatomy, EVAR may provide a particularly useful alternative to OSR. The potential technical difficulties of OSR are avoided, and multiple reports of successful endovascular repair leading to subsequent improvement in the extent and severity of the perianeurysmal and retroperitoneal fibrotic process, similar to that seen following conventional OSR, have been published.[234,235]

Horseshoe kidney. Advanced knowledge of a horseshoe, ectopic, or pelvic kidney associated with an AAA is particularly important in view of the frequently complex renal artery anatomy, possibility of anomalous ureteral drainage, and other technical challenges for OSR. The diagnosis is usually evident on preoperative CT scans. Because multiple renal arteries are encountered in 75% of cases, often arising from the aneurysmal aortic segment, detailed preoperative arteriography may be required to fully elucidate the aberrant origin and location of renal blood supply and facilitate operative planning.[197,236]

If a horseshoe kidney is encountered unexpectedly during open AAA repair via an anterior transperitoneal approach, the isthmus of the horseshoe should not be divided unless it is extremely thin and atrophic, due to concern regarding injury to a renal collecting system and resultant urinary leakage. In such circumstances, the surgeon should try to mobilize and elevate the renal isthmus, and tunnel the graft beneath it. Careful dissection will be necessary to identify and preserve the principal renal circulation.

Ideally, preoperative recognition and appropriate delineation of renal artery anatomy will allow a retroperitoneal approach, which most surgeons prefer for repair of an AAA with horseshoe kidney. With a flank approach, the bulk of renal tissue can be easily displaced anteriorly and thereby avoided, and anomalous renal arteries originating from the AAA more easily preserved by reimplantation, short renal artery grafts, or revascularization from within the AAA sac.[197,236,237] In the 25% of cases with single main renal arteries originating in a normal location above the aneurysm, patients with suitable proximal and distal anat-

omy may benefit from endovascular stent graft repair and avoidance of the technical challenges associated with open operation.

Aortocaval fistula. Spontaneous aortocaval fistula is an infrequent complication of an AAA with a 2% to 4% incidence of ruptured aneurysms associated with a venous fistula. The mortality rate is approximately 30%.[238] Presumably the fistula forms as a result of necrosis of the aortic wall leading to an adventitial inflammatory reaction that eventually leads to rupture at the aortic-caval interface. Aortocaval fistulas most often present in men with an average age of 65 years. Patients may complain of confusion, lethargy, abdominal or low back pain, or dyspnea. A palpable abdominal mass with a loud machinery murmur is typically present on exam. High output failure with elevated central venous pressure, pulmonary edema, lower limb edema, pulsating varicose veins, hematuria, rectal bleeding, or priapism may also be present.

Both ultrasonography and CT scanning may demonstrate the presence of the fistula. Given the intense inflammatory reaction at the site of fistula formation, proximal and distal control is most safely achieved by direct compression above and below the fistula within the aneurysm sac. An aortocaval fistula carries an increased risk for massive hemorrhage or air embolism. The fistula is usually closed primarily with nonabsorbable suture. Several reports have described successful EVAR of AAA with spontaneous aortocaval fistula.[239-241]

Primary aortoenteric fistula. Primary aortoenteric fistula is a rare event often characterized by a classical triad of gastrointestinal bleeding, pain, and a pulsating mass that, in fact, is present in only a minority of patients.[242] Most primary aortoenteric fistulas are caused by an aneurysmal aorta and are almost always heralded by repetitive gastrointestinal bleeding. The time interval between the first herald bleed and massive exsanguination may range from hours to months. Computed tomography that demonstrates air within the aortic wall and contrast within the gastrointestinal tract strongly suggests the presence of a fistula. Indeed, gastrointestinal bleeding combined with a negative endoscopy in the presence of a known AAA suggests a primary aortoenteric fistula and mandates an urgent CT. Controlled hypotension with maintainenance of systolic blood pressure between 60 mm Hg and 100 mm Hg may reduce the risk of recurrent hemorrhage during the period leading up to repair. The fistula commonly involves the third or fourth portion of duodenum in over half of cases, but may also involve the stomach, esophagus, or jejunum. Surgical approaches continue to carry an attendant mortality of 30% to 40%. Extranatomic bypass with repair of the intestinal defect is recommended, while in situ graft repair using an ePTFE prosthesis is appropriate in the unstable patient. A portion of omentum should be wrapped around the vascular anastomosis or closure site and the overlying intestine. Closure of the gastrointestinal defect alone without treatment of the aneurysm is not recommended. Operative cultures should be obtained and appropriate antibiotic treatment initiated. EVAR may allow an unstable patient to undergo later definitive repair on an elective basis. A drawback of endovascular treatment is the risk of overwhelming sepsis because of a persistent source of untreated infection.

Primary mycotic abdominal aortic aneurysm. A mycotic aneurysm of the abdominal aorta may result from primary aortitis, septic emboli, or an adjacent infection, such as pancreatitis or a psoas muscle abscess. Mycotic aortic aneurysms may be suggested by preoperative imaging with associated rupture a common feature. Salmonella and staphylococcus are common organisms.[243] Recommended approaches for intervention are similar to those described for treatment of an aortoenteric fistula. Clinical results of medical treatment alone with antibiotic therapy remain poor.[244] Recently, EVAR has also been applied in the treatment of mycotic aortic aneurysms with mixed results. In a review of 48 cases from 22 reports, Kan et al[245] identified rupture or fever at presentation, as well as persistent fever after EVAR as predictive of poor outcome and, under these circumstances, recommended definitive surgical treatment or use of EVAR as a 'bridge' to surgical therapy. Nonetheless, better outcomes were noted in the absence of these feastures and when preoperative antibiotic suppression was associated with negative blood cultures before intervention. Further study of the role of EVAR in this difficult problem is warranted.

AAA secondary to Type IV Ehlers-Danlos syndrome (EDS). Diagnostic arteriography, in patients with type IV EDS has been associated with a 17% to 67% complication rate, including tears, dissection, perforation, and pseudoaneurysm formation, with 6% to 19% mortality.[246] Blood vessels display a "wet-tissue" consistency and may fail to hold sutures. Use of Teflon pledgets along with fibrin glue is recommended. Succesful endovascular repair has been reported and, if anatomically feasible, should be considered the preferred approach for treatment.[247]

Preoperative insertion of ureteral stents should be considered for patients undergoing OSR of an inflammatory aneurysm, particularly in the presence of hydronephrosis.

Level of recommendation:	Strong
Quality of evidence:	High

Timing of surgery. A ruptured AAA clearly represents a true surgical emergency. Documented rupture, particularly with associated hypotension, demands immediate transfer to the operating room as rapidly as possible. The timing of surgical repair in patients with symptomatic but unruptured aneurysms remains more controversial. A patient with a known AAA or pulsatile mass on abdominal exam who presents with acute onset of back or abdominal pain should undergo an immediate contrast enhanced CT scan to determine if rupture has occurred. The timing of AAA repair for those patients with symptomatic but unruptured aneurysms represents a clinical dilemma. Many series have demonstrated a significantly higher operative mortality for patients with symptomatic but unruptured AAA who undergo emergent open repair.[248-250] For example, Sullivan et al noted a fivefold increase in mortality (26% vs 5.1%) for patients with

symptomatic aneurysms undergoing emergency operation in contrast to elective repair.[249] Similarly, Haug and co-workers observed a mortality rate of 18% in patients with symptomatic AAA who underwent surgical repair within 24 hours versus 4.2% for those with semi-elective repair following emergency admission or patients having elective surgery.[248] In the Mayo Clinic series, no deaths from rupture occurred among those patients whose operation was delayed.[251]

The reason for such differences in outcome are multifactorial, but include the fact that truly emergency surgical repair is often carried out in less favorable circumstances without the usual surgical and anesthesia personnel or at times outside the typical workday. Similarly, some patients may benefit from preoperative preparation or interventions. Although definitive recommendations are not possible, and each clinical situation must be approached individually, it may be prudent to delay truly emergent repair of symptomatic but unruptured aneurysms for four to 24 hours until optimal conditions may be achieved. If such an approach is elected, blood should be available and the patient cared for in an ICU-setting prior to operation.

A related practical concern regarding timing of operation, which frequently arises but for which there is practically no data relates to the scheduling of elective repair in asymptomatic patients with large AAA. While the definition of "large" AAA is ill-defined, it is well accepted that the rupture risk of AAA 6 cm to 7 cm in diameter ranges from 10% to 20% per year, 7 cm to 8 cm 20% to 40%, and those >8 cm 30% to 50%.[1] However, these are estimates for rupture risk over a 12 month period, and the rupture risk within a period of weeks or one to two months remains completely unknown. There is certainly no standard of care in regard to the time period within which elective repair must be carried out. While clearly there is little advantage in excessive delay in performing elective repair of a large AAA, it is appropriate to obtain pertinent preoperative studies and tests in a timely fashion, particularly in older, high-risk individuals. Similarly, if preoperative intervention may potentially make such a patient a more favorable candidate for operation, then some delay appears warranted. On occasion, however, rupture may occur during this interval. Although completely unpredictable, it is recommended that this decision-making be made with the patient and family, and that they understand and accept such a small risk.

Immediate repair is recommended for patients that present with documented aneurysm rupture.

Level of recommendation:	Strong
Quality of evidence:	High

Should repair of a symptomatic AAA be delayed to optimize associated medical conditions, it is recommended that a patient be monitored in an ICU-setting and blood products be available.

Level of recommendation:	Strong
Quality of evidence:	High

Table VII. Estimated perioperative complications after elective open surgery for AAA

Complication	*Frequency (%)*
All cardiac	15
Myocardial infarction	2-8
All pulmonary	8-12
Pneumonia	5
Renal insufficiency	5-12
Dialysis	1-6
Bleeding	2-5
Wound infection	<5
Leg ischemia	1-4
Deep venous thrombosis	5-8
Colon ischemia	1-2
Stroke	1-2
Graft thrombosis	<1
Graft infection	<1
Ureteral injury	<1

From Schermerhorn ML, Cronenwett JL. Abdominal aortic and iliac aneurysms. In: Rutherford RB, editor. Vascular surgery. 6th ed. Philadelphia: Elsevier Saunders; 2005. p. 1431.

A final issue in regard to timing is whether or not a recent prior procedure, such as a laparotomy, coronary revascularization, or other operations can increase the likelihood of AAA rupture and represent an indication for earlier AAA repair. This has been a long-standing controversy, with anecdotal experience suggesting that this is indeed the case.[252,253] It has been suggested that a balance between the dynamic equilibrium of collagen synthesis and lysis might be altered by such operations, with injury, local inflammation, and possible nutritional depletion resulting in enhanced lysis of collagen and increased AAA rupture risk. However, Cohen et al[254] examined this question in rats, and found no evidence of increased aortic collagenase activity, and a prospective clinical study on this topic by Durham and colleagues found no evidence of this phenomenon.[255] It seems reasonable to conclude that the risk of AAA rupture is not increased by such unrelated surgeries, and that a period of four to six weeks should be allowed to enable a satisfactory recovery to occur prior to elective AAA repair. Certainly this should be modified if the aneurysm is symptomatic, large, or other worrisome features are identified on CT scan. Similarly, if endovascular repair is feasible, a shorter interval between operative procedures may be justified.

Perioperative outcomes of open AAA repair. Perioperative 30-day mortality of conventional OSR varies considerably in the literature. Much of this variability appears related to the type of study reported, that is, hospital-based versus population-based series, and whether or not the reports were prospective or retrospective studies.[256] Over the past two decades, mortality risk of elective infrarenal AAA repair in referral-based single institution reports from selected centers of excellence has ranged from 1% to 4%.[1,76,257-259] In multiple population-based series, however, employing state-wide or nation-wide data bases, reported perioperative mortality rates have generally been in the 4% to 8% range even in contemporary experi-

ence.[1,4,101,212,256,260-265] This is clearly higher than the perioperative mortality risk of endovascular repair. Similarly, perioperative morbidity associated with OSR, particularly, cardiac, pulmonary, or renal complications, as well as the incidence of colonic ischemia exceeds that of EVAR. Complications after open AAA repair are observed in 15% to 30% of patients (Table VII).[265-267]

Multiple reports have identified a strong relationship between outcomes following AAA repair and both hospital and individual surgeon case volume and experience.[101,259,262,268,269] For example, Birkmeyer and colleagues,[268] using national Medicare claims data and the Nationwide Inpatient Sample 1994-1999, found a 7.8% mortality rate following elective AAA repair in very low (<17/year) volume hospitals versus 4.4% in very high (>79/year) volume hospitals. Dimick et al[270] reviewed 3,912 patients undergoing AAA repair in 1997 and noted a 4.2% overall in-hospital mortality. Mortality was 3% at high volume hospitals, in contrast to 5.5% at low volume hospitals. Surgeon specific volume and specialized training have also been observed to be of significance. Dimick et al[270] found that lowest perioperative AAA mortality was associated with operations performed by vascular surgeons (2.2%), compared with cardiac surgeons (4.0%) and general surgeons (5.5%) ($P < .001$). In risk-adjusted analysis, high-volume hospital, vascular surgery specialty, and high–volume surgeon were all independently associated with lower risk of in-hospital operative mortality. Risk reduction was 30% for high-volume hospitals and 40% for operation by a high-volume surgeon. AAA repair by general surgeons compared with vascular surgeons was associated with 76% greater risk of death.[270] Similar training and specialty-related outcome relationships were noted by Cronenwett and Birkmeyer in the Dartmouth Atlas of Healthcare.[271] The overall 30-day mortality rate for open AAA repair in 1996 was 5.5%, but ranged from 4% to 7.9% among surgeons performing more than 10 per year and those performing fewer than four per year. Mortality was 4.4% for vascular surgeons, 5.4% for cardiothoracic surgeons, and 7.3% for general surgeons. We recommend that OSR is best performed at centers that have a documented in-hospital mortality of less than 5% for elective repair.

Elective OSR for AAA should be performed at centers with a documented in-hospital mortality of less than 5% for open repair of infrarenal AAA.

Level of recommendation:	Strong
Quality of evidence:	High

Endovascular repair

Endovascular aortic aneurysm repair (EVAR) has rapidly expanded since Parodi's first report[272] and is progressively replacing OSR for the treatment of infrarenal abdominal aortic aneurysms (AAAs).[273] According to nationwide hospital databases, there has been a 600% increase in the annual number of EVAR procedures performed in the United States since 2000.[266] Although long-term durability and associated costs have not been completely established, EVAR now accounts for more than half of all AAA repairs. Moreover, since the introduction of EVAR, the annual number of deaths from intact and ruptured AAA has significantly decreased in the United States. This has coincided with an increase in elective AAA repair after the introduction of EVAR and a decrease in the diagnosis and repair of ruptured AAA.[274]

Infrarenal fixation. EVAR usually requires adequate nonaneurysmal proximal and distal attachment sites. Proximal fixation may be obtained through infrarenal or suprarenal fixation. According to the instructions for use of endografts with infrarenal fixation, an infrarenal neck at least 15 mm in length and less than 32 mm in diameter with an angulation < 60° is required for optimal sealing. Pooled results from EVAR devices included in the published Lifeline report (ie, Guidant AnCure [Indianapolis, Ind], Medtronic AneuRx [Santa Rosa, Calif], Gore Excluder [Flagstaff, Ariz], and Endologix PowerLink [Irvine, Calif], provide safety and efficacy data for endografts with infrarenal fixation.[275,276] The number of EVAR devices with infrarenal fixation submitted for FDA review ranged from 121 for Ancure to 416 for AneuRx. The pooled results as well as device-specific data obtained from FDA websites indicates that 30-day mortality rates with these devices ranged from 1% with Gore Excluder to 4.2% for the Guidant Ancure endograft. Major complications or adverse events within 30 days were not provided in the Lifeline report but ranged from 13.6% in FDA reports with the Gore Excluder to 35.6% with Guidant Ancure.[277] In device-specific published studies, early ruptures were only reported for the AneuRx device and were 0.3%.[278] Primary conversion ranged from 0% for the Excluder device up to 9.7% for the Ancure system. The overall incidence of Type I endoleaks within 30 days following EVAR with endografts with infrarenal fixation was 4.2% and ranged from 0.9% to 11%.[279]

Suprarenal fixation. Suprarenal fixation has been proposed as a more effective means of proximal fixation when the morphologic features of the proximal neck are unfavorable, including, short length, severe angulation, reverse taper, a barrel-shaped neck, circumferential mural thrombosis, or calcification. Despite the potential advantages of suprarenal fixation, concerns have been raised regarding the short- and long-term risks of renal or mesenteric artery embolization and occlusion. To date, several observational studies have reported the efficacy and safety of suprarenal endograft fixation.[280-284] Moreover, almost 50% and 87% of endografts used in the DREAM and EVAR-1 trials, respectively, were performed with endografts that used suprarenal fixation. Significantly, rates of renal dysfunction appear to be equivalent for endografts with transrenal nitinol or stainless steel stents and not significantly different among patients undergoing EVAR with infrarenal or suprarenal fixation.[283] Although suprarenal fixation may produce a higher incidence of small renal infarcts, in most patients these do appear to be clinically

significant. The presence of renal dysfunction after EVAR with suprarenal fixation appears to be multifactorial and transient in most patients.[285] Nonetheless, renal artery occlusion and infarctions have been reported in patients with preexisting renal artery occlusive disease[286,287] and, while infrequent, visceral dysfunction and celiac or mesentery artery occlusion may occur secondary to suprarenal fixation.[288,289]

Recommended management of the internal iliac artery. Exclusion of the hypogastric artery (HA) to prevent a type II endoleak is usually required during endovascular repair of aortoiliac aneurysms that involve either the distal common iliac artery or the hypogastric artery itself.[290-294] Embolization of the hypogastric artery increases by 16% the proportion of AAAs suitable for EVAR. Several observational studies have revealed that unilateral embolization of the hypogastric artery can be performed during EVAR with minimal adverse events.[291,292] Although buttock claudication and erectile dysfunction occur in up to 40% of patients after unilateral hypogastric artery embolization, these symptoms tend to improve over time.[295] Indeed, one of the largest series of patients undergoing HA interruption during AAA repair revealed that persistent buttock claudication developed in 12% of unilateral and 11% of bilateral hypogastric artery interruptions, whereas impotence occurred in 9% of unilateral and 13% of bilateral HA occlusions.[296] Despite concerns about prolonged procedural time and increased amount of contrast, concomitant unilateral HA embolization during EVAR has been shown to be safe and effective, as compared with staged procedures.[297]

Bilateral hypogastric artery occlusion with endograft extension into both external iliac arteries is occasionally required in high-risk patients when there is no distal fixation zone in either common iliac artery or the aneurysm involves both common iliac and hypogastric arteries. Although antegrade flow into at least one of the hypogastric arteries should be attempted, if possible, bilateral HA embolization may be necessary in some situations. Initial concerns about life-threatening pelvic or colonic ischemia and neurologic deficits after bilateral hypogastric artery interruption during EVAR may have been overestimated as several recent reports suggest that such devastating complications rarely occur.[224,291,298,299] The risks associated with bilateral HA occlusion are restricted to a more severe, persistent and frequent buttock claudication and erectile dysfunction.

Technical considerations that may reduce the incidence of adverse events when bilateral HA embolization is required, include staging bilateral HA embolization when possible, embolization of the main trunk of the HA so as to preserve pelvic collateral vessels, preserving collateral branches from the common and deep femoral and external iliac arteries, and maintaining adequate anticoagulation during these procedures.[291,299]

Alternative strategies to avoid bilateral HA occlusion during EVAR include open or endovascular revascularization of at least one HA by transposition or bypass grafting or endografting from the external iliac artery.[300,301] More recently, iliac branched endografts designed to preserve flow to the HA have been used with satisfactory preliminary results.[302-304] Combining two bifurcated endografts has also be reported.[305] Long-term durability of iliac branched devices is not well defined, although a recent study has reported 87% patency at 60 months.[304] At this time, branched endografts are not commercially available in the United States.

As an adjunct to EVAR, bilateral hypogastric artery occlusion may be acceptable in certain anatomic situations for patients at high-risk for OSR.

Level of recommendation:	Strong
Quality of evidence:	Moderate

Role of EVAR in patients requiring urgent or emergent repair. In an effort to improve outcomes for patients presenting with symptomatic or ruptured AAAs, the impact of urgent or emergent EVAR has been recently evaluated. A randomized trial comparing EVAR and OSR for ruptured AAAs revealed that the suitability for endovascular repair was 46%, but the application rate was lower (30%).[306] Observational studies have revealed improved outcomes after emergent EVAR for ruptured AAAs, but significant selection bias and lack of uniform inclusion criteria and reporting standards confounds these analyses.[307-312] In contrast, a recent industry-sponsored study of emergent EVAR and OSR in 100 consecutive patients across 10 institutions in Europe failed to demonstrate improved in-hospital (35% and 39%, respectively) or 3-month mortality (40% and 42%).[313] Open repair was performed in 80% of cases because of unfavorable neck anatomy. Identical mortality rates (53%) were also reported in a study of 32 patients randomized to EVAR or OSR.[314] Nonetheless, recent studies analyzing national trends in the United States have observed that endovascular repair is being used with increasing frequency in the emergency management of ruptured AAA, with decreasing mortality.[315,316] Results in non-teaching centers and low volume institutions, however were substantially worse than those in teaching hospitals and high volume centers and fewer women who present with ruptured AAA are treated by EVAR.[316]

Establishing a protocol for urgent or emergent EVAR for ruptured AAAs appears to be essential to obtain optimal results.[309] Such protocols require that emergency department personnel alert the vascular team and operating room staff as soon as a ruptured AAA is suspected and a CT angiogram is obtained in hemodynamically stable patients. All other patients should be directly transferred to the operating room. "Hypotensive hemostasis," which refers to restricting aggresive fluid resuscitation as long as the patient remains conscious and systolic blood pressure exceeds 50 mm Hg to 70 mm Hg, should be implemented to limit hemorrhage.[312] The question of whether to use an aortic occlusion balloon remains unresolved with some

advocating its use in patients in circulatory collapse whereas others recommend its use in the presence of hemodynamic instability or anatomic limitations that prevent expeditious EVAR.[317] A femoral artery approach with use of a long sheath may be more expeditious than use of brachial artery access. The sheath may be advanced into the supraceliac aorta to support the balloon and permit its removal after endograft placement.[317] In hemodynamically unstable patients that do not have a preoperative CT scan, device selection and evaluation of the proximal and distal endograft sealing zones can be based on intraoperative angiography, and ideally, on intravascular ultrasound. Both bifurcated and aortouniliac endografts have been used for emergent EVAR of ruptured AAAs.[309-311]

Abdominal compartment syndrome is a well-recognized complication after EVAR for ruptured AAAs.[309,311,318] It typically occurs among hemodynamically unstable patients in which a large retroperitoneal hematoma and diffuse visceral edema results in intra-abdominal hypertension and multiple system organ dysfunction. Early recognition through measurement of bladder pressure and surgical decompression are necessary to improve survival. Use of an aortic occlusion balloon, coagulopathy, massive transfusion, and conversion to an aortouniliac device, are predictors of abdominal compartment syndrome.[318] Although colonic ischemia is less frequent after EVAR, it remains an acknowledged risk, particularly among patients who have undergone repair of a ruptured AAA.[319,320] Regardless, of the type of repair, the mortality rate in the presence of colonic ischemia is approximately 50%.

Emergent EVAR should be considered for treatment of a ruptured AAA, if anatomically feasible.

Level of recommendation:	Strong
Quality of evidence:	Moderate

Role of EVAR in high-risk and unfit patients for open repair. Several observational studies have reported excellent technical success and low rates of morbidity and mortality after EVAR in high-risk patients with significant co-morbidities.[1,321-324] However, the EVAR-2 trial found no survival advantage for patients deemed medically unfit for OSR when randomized between elective EVAR or no intervention.[325] Substantial in-hospital (9%) and preprocedural (8%) mortality was observed in the group randomized to EVAR, which persisted when urgent or emergent EVAR cases were excluded from the analysis (30-day mortality 7%). Several limitations of this study have been noted. First, nine ruptures and 14 deaths occurred in the EVAR group (n = 166) prior to elective repair with a median time from randomization to EVAR of 57 days. Second, 47 patient crossovers occurred from the no intervention group (n = 172) to EVAR. Finally, inclusion criteria were subjective with the potential for introduction of bias by the treating surgeon.[75] In summary, a number of reports have documented that EVAR can be performed with low rates of perioperative mortality and morbidity in patients at high risk for OSR. Nonetheless, the ability of EVAR to provide a survival advantage for patients considered truly unfit for open repair is uncertain. Although a number of preoperative risk prediction methods have been reported,[326] additional research is needed to define objective criteria that identify patients who are unfit for OSR and whose anticipated life expectancy limits benefit from EVAR.

EVAR may be considered for high-risk patients unfit for surgical repair.

Level of recommendation:	Weak
Quality of evidence:	Low

Perioperative outcomes of elective EVAR

Incidence of 30-day and in-hospital mortality. It would seem to be axiomatic that EVAR, as a minimally invasive technology would be associated with lower in-hospital and 30-day mortality rates as compared to OSR. Indeed, among nonrandomized but controlled trials, 30-day mortality rates of less than 2% were reported among all FDA pivotal study populations (AneuRx [n = 416] 1.7%;[277] Excluder [n = 235] 1.3%;[327] Zenith [n = 352] 1.1%;[328,329] Powerlink [n = 192] 1%[330]). However, pooled trial data representing the OSR cohorts was associated with a comparable 30-day mortality rate of 1.4%.[277] In an analysis of pooled trial data for patients considered at high risk for OSR, 30-day mortality for patients treated by EVAR or OSR was comparable (2.9% EVAR vs 5.1% OSR, $P = .32$).[321] Among randomized, prospective trials, lower mortality was observed among those patients treated by EVAR. Specifically, in-hospital mortality rates in the EVAR-1 trial and the DREAM trial were 1.7% and 1.2% for EVAR and 6% and 4.6% for OSR, respectively.[36,331] These differences, however, did not achieve statistical significance ($P = .1$). Moreover, only 23% of screened patients in the EVAR-1 trial were eligible for randomization with more than half of all patients excluded from the trial due to the presence of anatomic unsuitability for EVAR (54%).

It is noteworthy that with rapid adoption of this technology in the United States, much lower mortality rates have been reported for EVAR in analyses of large statewide and multi-state population-based databases.[277] These trends were noted early after initial FDA approval of first generation devices. In-hospital mortality of 4.2% for OSR and 0.8% for EVAR was observed among approximately 1,600 patients treated in New York State treated in 2002.[33] Lee at al[265] analyzed 4,607 patients who underwent OSR and 2,565 treated by EVAR that were enrolled in the Nationwide Inpatient Sample (NIS) database in 2002. In-hospital mortality was significantly lower following EVAR (1.3% vs 3.8%), despite the presence of greater cardiovascular co-morbidity. In a review of 65,502 patients entered into the NIS database who underwent elective EVAR between 2001 and 2004, Timaran et al[324] noted an in-hospital mortality of 2.2%. Stratified analyses, including

only elective EVAR procedures, revealed that in-hospital mortality was 1.7% in patients with the most severe comorbidities and 0.4% among those with lower comorbidity. In this regard, analysis of a high risk cohort from the Veteran Affairs (VA) National Surgical Quality Improvement Program (NSQIP) revealed those patients who underwent elective EVAR (n = 788) had a significantly lower 30-day mortality than those treated by OSR (n = 1,580) (3.4% vs 5.2%, $P = .047$).[322] In a recent analysis of 45,000 propensity-score-matched Medicare beneficiaries treated by EVAR and OSR, mortality was significantly lower after EVAR (1.2% vs 4.8%; $P < .001$), with reduction in mortality most pronounced for those of advanced age (80 to 84 years: 1.6% vs 7.2%; ≥ 85 years: 2.7% vs 11.2%; $P < .001$).[34] While a variety of demographic features and clinic data were used for propensity matching, it is possible that differences in outcome reflect the inclusion of anatomically more complex aneurysms among the cohort treated by OSR. Nonetheless, the marked difference in outcome among octagenarians is consistent with the anticipated physiologic benefits of a minimally invasive intervention, particularly among patients at high risk for perioperative morbidity and mortality. It is noteworthy that disparities in EVAR outcome have been identified between patients of varying ethnicity and insurance type[332]

Procedural outcomes and primary conversion to OSR. Procedural blood loss is less for EVAR (414 mL, range, 96 mL to 783 mL) when compared to OSR (1,329 mL, range, 451 mL to 1,800 mL),[277] with reductions in ICU (0.7 vs 1.6 d) and hospital (4.2 days vs 9.9 days) stays.[277] In the initial experience with EVAR, early conversion to OSR was necessary in as many as 18% of patients.[333,334] In both the DREAM and EVAR-1 trials, primary conversion to open repair occurred in 1.8% of patients,[36,331] while in recent FDA pivotal trials, conversion ranged from 0% (Excluder and Zenith) to 1.6% (Endologix).[277] In the recent analysis of 45,000 propensity-score-matched Medicare beneficiaries treated between 2001 and 2004, Schermerhorn et al reported conversion to OSR at initial EVAR in 1.6%.[34] Thirty-day technical success, in which EVAR leads to complete exclusion of the AAA with or without prior secondary intervention, has ranged from 77% to 100% with a mean of 91% in eight studies (n = 1,493).[277]

Major medical adverse events. Major medical complications are lower after EVAR than OSR. A meta-analysis of observational studies conducted prior to 2002 demonstrated an incidence of systemic complications of 9% after EVAR, as compared with 22% after OSR, largely attributable to fewer cardiac and pulmonary events.[335] Major adverse events within 30 days of treatment ranged from 13.6% for Excluder and 18.8% for Powerlink to an incidence of adverse events of 24.4% for the Zenith endograft in controlled, non-randomized FDA sponsored pivotal trials. The incidence of major adverse events were substantially higher in the OSR cohorts (26% to 42.5%).[277] Likewise, the DREAM trial reported fewer systemic complications after EVAR as compared with OSR (12% vs 27%), although all cardiac morbidity (EVAR 5.3% vs OSR 5.7%) and severe cardiac complications (EVAR 1.8% vs OST 1.1%) were comparable among groups.[36] As noted earlier, Anderson et al[33] reported a lower incidence of cardiac complications in a statewide review of patients treated by EVAR in 2002 (3.3% vs 7.8%). A similar, though less dramatic difference, in the incidence of perioperative MI was reported by Schermerhorn et al (7% vs 9.4%, $P < .001$).[34] In addition, the latter study noted a reduction in the incidence of pneumonia (9.3% vs 17.4%, $P < .001$), acute renal failure (5.5% vs 10.9%, $P < .001$), and need for dialysis (0.4% vs 0.5%, $P = .047$) among those treated by EVAR.[34] Post-implantation syndrome, characterized by fever, malaise, back or abdominal pain after EVAR, may last up to 10 days, but appears to be a relatively rare phenomenon. It has been attributed to the release of cytokines after aneurysm sac thrombosis.[336] Contrast-induced nephropathy occurs infrequently after EVAR.

Device related complications and 30-day re-intervention. The incidence of local vascular or device related complications, as well as the 30-day re-intervention rate is greater after EVAR than OSR. The DREAM trial demonstrated that a higher incidence of local vascular or device related complications occurred after EVAR than OSR (16% vs 9%).[36] Similar findings have been reported in several observational studies with local or vascular complications occuring in 9% to 16% of patients after EVAR.[277] Moreover, a pooled analysis that included pivotal data from the Ancure, AneuRx, Excluder, Powerlink, and Zenith studies revealed a 30-day re-intervention rate of 15.6%.[277] In the EVAR-1 and EVAR-2 trials, reintervention within 30 days of EVAR occurred in 9.8% and 18% of patients, respectively.[325,331] Groin and wound complications are the most frequent event. Stents, endografts, or surgical repair may be required if severe vascular access injuries occur. Distal embolization is now rare with lower profile introducer systems. Limb occlusion occurs more frequently in patients with aortoiliac occlusive disease, a small (<14 mm) distal aorta and tortuous vessels and when unsupported endografts are used. The EVAR-1 trial revealed that almost 75% more secondary interventions were undertaken within 30 days of the procedure or within the same admission after EVAR as compared with OSR.[331]

Mid-term outcomes. The DREAM and EVAR-1 trials have provided mid-term follow-up data at two and four years, respectively.[337,338] Although a 3% reduction in aneurysm-related mortality persisted throughout the follow-up period, in both trials, the initial reduction in all-cause mortality was eliminated within one to two years with equivalent overall survival in both treatment groups. Moreover, EVAR was associated with a greater number of late complications and secondary reinterventions. In the EVAR-1 trial, reinterventions occurred three times as often, exceeding 20% at four years, whereas for OSR the reintervention rate was approximately 6%. The DREAM trial showed a similar pattern in the first nine months after randomization (hazard ratio, 2.9; 95% confidence interval

(CI), 1.1 to 6.2), with roughly parallel rates for reintervention for EVAR and OSR, thereafter (hazard ratio, 1.1; 95% CI, 0.1 to 9.3). A population-based study of 45,660 Medicare beneficiaries undergoing either EVAR or OSR demonstrates similar findings.[34] Survival curves converged three years after initial repair and by four years, AAA rupture (1.8% vs 0.5%) and AAA-related reinterventions (9.0% vs 1.7%) were more likely after EVAR than after OSR. In contrast, laparotomy-related complications that required surgical repair were more likely among patients who had undergone OSR (9.7%, vs 4.1%), as were hospitalizations for bowel obstruction or abdominal-wall hernia.

Although the DREAM trial reported that quality of life scores were greater for those patients treated by EVAR, within six months, differences were no longer apparent and sexual function scores were similar or better for those patients who underwent OSR.[338] Likewise, in the EVAR-1 trial, SF-36 mental component scores were similar for both treatment groups throughout the postoperative and follow-up periods, with differences only observed for the physical component scores, which were lower during the first three postoperative months for those treated by OSR.[337]

In summary, the recognized benefits of EVAR, including reduced morbidity, ICU and hospital length of stay, as well as observed lower perioperative mortality rates, especially among elderly patients, has led to widespread adoption of this technology. Nonetheless, it is recommended that elective EVAR is best performed at centers that have a documented in-hospital mortality of less than 3% and a primary conversion rate to OSR of less than 2% for elective repair. Further research is needed to improve EVAR devices and related techniques to reduce complications and long-term follow-up; to identify whether EVAR outcomes vary with respect to endograft type or aneurysm features; and to define the relationship of hospital and physician volume to outcomes after EVAR.

EVAR should be performed at centers with documented in-hospital mortality for elective EVAR of less than 3% and a perioperative conversion rate to OSR of less than 2%.

Level of recommendation:	Strong
Quality of evidence:	High

Management of associated vascular disease in patients undergoing OSR or EVAR. Coexistence of other vascular disease with an abdominal aortic aneurysm is common. Several series reporting observations of aortography have documented greater than 50% stenosis in 20% to 40% of renal arteries, 10% to 15% of celiac or SMA branches, and 20% to 30% of iliac vessels.[339,340] Whether or not to extend repair to include correction of such associated vascular disease remains uncertain.

Decision making for intervention is based upon a consideration of (1) the severity of associated lesions, (2) the presumed natural history of the disease, and (3) the anticipated morbidity and mortality risk of combined repair. While definitive long-term natural history data are not available for many associated lesions, some data is available in regard to renal artery stenosis. Tollefson et al evaluated sequential arteriographic findings in patients with renal artery stenosis undergoing aortograms for evaluation of aortic disease.[341] Progression of renal stenosis was noted in 53% of these patients over a mean follow-up of 4.5 years, with 9% of arteries progressing to total occlusion. All vessels that occluded had severe (>75%) pre-existant stenoses. Zierler and co-workers prospectively determined progression of renal artery stenosis with serial ultrasound duplex scans.[342] These investigators observed progression in 42% of renal artery lesions by two years, with progression to total occlusion in approximately 5% per year for arteries with >60% stenosis.

In terms of mesenteric occlusive disease, Thomas and colleagues examined 980 consecutive aortograms over a six-year period to identify patients with significant mesenteric stenosis but without symptoms of mesenteric ischemia. During subsequent follow-up, these authors noted that 27% of patients with significant three-vessel visceral artery disease developed clinically overt symptoms of mesenteric ischemia.[343]

Because of such data, it is often reasoned that concomitant repair of associated disease would be prudent in order to avoid the technical difficulty and potential risk of later reoperation should this prove necessary due to progression of the associated vascular lesions. While simultaneous aortic grafting and renal artery reconstruction have been reported in some series to have a negligible impact on the risk of death or major complications,[344-348] many other reports clearly document significantly increased morbidity and mortality risk of combined operation.[349-351]

From available data, it can be concluded that prophylactic repair of associated renal or mesenteric artery disease cannot be justified.[352] A decision to repair each lesion should be based upon its own individual merits and indications. When associated renal or mesenteric disease is severe and symptomatic, combined operation employing either bypass grafting or endarterectomy techniques in conjunction with aortic graft repair may well be appropriate, and is generally associated with good long-term functional outcomes. However, increased risks secondary to the more extensive procedure must be recognized.

An alternative strategy in the current era of increasing endovascular interventions is staged treatment of the associated vascular disease, employing catheter-based techniques for treatment of renal or visceral artery disease before AAA repair. While this may be an appealing and useful solution in many patients, especially high-risk individuals, definitive data as to the timing of renal or visceral angioplasty, as well as early and long-term risks and merits of such an approach are not currently available.

Accessory renal arteries are present in 15% to 20% of patients, and occasionally arise from the aneurysm itself.[339] Whether or not such accessory vessels require preservation and reimplantation into the graft at the time of open aneurysm repair remains uncertain. However, several recommendations should be considered. First, it is important to identify the presence of such arteries prior

to operation, in order to minimize the hazards of injury or ligation during operative dissection. Accessory renal arteries of a clinically significant size are detected in the majority of preoperative CT scans. Secondly, it may be possible to determine how much of the involved kidney is supplied by this branch. Preservation and reimplantation, similar to an IMA Carrel patch, may be considered for a sizable (>3 mm) accessory renal artery or those that supply one third or more of the renal parenchyma. Obviously, the presence of preoperative renal insufficiency makes sacrifice of any renal parenchyma and function less appealing.

Such recommendations have come into question with increasing use of EVAR. Since preservation of accessory renal arteries arising from the segment of aneurysmal disease being excluded is obviously not possible with this methodology, the frequency with which accessory renal arteries have been sacrificed has increased. Most reported experience in this regard has concluded that loss of such accessory renal branches has rarely led to any clinically significant sequelae, such as deterioration of renal function, development of postoperative hypertension, or symptomatic renal infarcts.[353,354] This suggests that preservation and reimplantation during OSR may not be as important as previously believed.

Concomitant surgical treatment of other vascular disease at the time of open AAA repair should be considered in selected patients.

Level of recommendation:	Strong
Quality of evidence:	Moderate

Preoperative renal artery or superior mesenteric angioplasty and stenting is recommended for selected patients with symptomatic disease.

Level of recommendation:	Strong
Quality of evidence:	Low

Preservation of accessory renal arteries that are 3 mm or larger in diameter or supply more than one-third of the renal parenchyma can be beneficial at the time of open aneurysm repair.

Level of recommendation:	Strong
Quality of evidence:	Low

Laparoscopic aneurysm repair

Several observational studies, case reports, and technical notes have reported that totally laparoscopic or laparoscopic-assisted repair of AAAs is feasible.[355-360] Most of these reports, however, originate in a few specialized centers with extensive experience in laparoscopic techniques. Because of the advanced technical skills and specialized instrumentation necessary for these procedures, laparoscopic repair of AAAs has not been widely adopted.

The surgical principles of laparoscopic AAA repair are the same as those of OSR (ie, endoaneurysmorrhaphy, aneurysm exclusion and anatomic reconstruction). In theory, the main advantage of laparoscopic AAA repair is the performance of the standard surgical repair using a less invasive approach, which potentially may result in less morbidity and excellent long-term results compared to conventional AAA repair. Moreover, laparoscopic AAA repair could also be used in patients with AAAs unsuitable for EVAR. Further evaluation of this technique is required to define its role in the treatment of elective AAAs.

Although laparoscopic-assisted AAA repair has been used for several years, totally laparoscopic repairs using transperitoneal and retroperitoneal approaches have only recently been reported.[355,358,360] Inflammatory and ruptured AAAs are considered contraindications for totally laparoscopic repair. Robotically assisted laparoscopic repair has also been described, although it appears that further refinements are necessary to reduce operative and aortic cross-clamping times.[361]

Prior experience with laparoscopic aortic reconstructions for aortoiliac occlusive disease is considered essential before performing total laparoscopic AAA repair. The learning curve is steep and about 50 procedures are considered necessary to obtain a good level of expertise.[358] Despite the technical challenges of laparoscopic AAA repair, postoperative complications and mortality appeared not to be affected by the technique itself. The main technical difficulties are the exposure of the abdominal aorta, as the small intestine limits the operative field, and the performance of laparoscopic anastomosis.[358] Hand-assisted laparoscopic aneurysm repair can be performed safely with operating times similar to those of OSR.[362] This technique has distinct advantages over a total laparoscopic approach as it can be applied to most repairs with fewer procedures required to gain proficiency.

Similar perioperative mortality rates have been reported for laparoscopic and standard open repairs, although mortality may decrease with experience.[358] Operative and clamp times are usually longer than those reported for open AAA repair. However, these times can be significantly reduced as individual experience is gained. Blood loss is comparable to that of OSR, although back bleeding from lumbar arteries requires intense suction and may collapse the abdominal cavity with immediate loss of visualization. Postoperative systemic, local, and vascular complications are similar for both laparoscopic and open repairs.[357] Postoperative ileus, return to normal diet, ambulation, and need for analgesics appeared to be reduced after laparoscopic repair compared with OSR. Lengths of ICU and hospital stay, however, are not significantly different.

The benefit of totally laparoscopic or laparoscopic-assisted AAA repair as compared with open repair is uncertain.

Level of recommendation:	Weak
Quality of evidence:	Low

Cost and economic considerations in aneurysm repair

Costs of AAA repair are substantial, and vary considerably based on complexity of the repair, patient co-morbidities, length of stay, incidence of complications, and overall outcome. These costs include those associated with pre-

operative assessment, risk stratification and surgical planning, operative materials and procedures, post-procedural recovery, lost productivity, and outpatient follow-up, as well as late secondary interventions required to maintain aortic integrity.[363] The majority of data from both outcome case series and decision modeling analysis identify EVAR procedural costs as being higher than those associated with OSR in the elective setting. Jonk and associates[364] reviewed all EVAR and OSR cost and cost comparison studies published through 2005 and noted that devices alone accounted for between 34% and 78% of total EVAR hospitalization expense, consistently the largest single item for such procedures. Given reduced procedural morbidity associated with EVAR, cost-effectiveness following the initial period of hospitalization might be expected to favor EVAR. However, in the EVAR-1 trial,[337] device-related secondary events and re-interventions accounted for a 33% increase in cost at four years. Retrospective case series data also demonstrate cost disparities between EVAR and OSR.[365] Nonetheless, Tarride et al[366] recently found that EVAR was cost-effective compared to OSR in a one-year prospective observational study of high-risk patients treated in a single payor health care system. All three Markov modeling studies published to date have also predicted higher lifetime costs associated with EVAR.[364] In the most recent analysis using data incorporated from the EVAR-1 and DREAM trials, a cost effectiveness ratio of £110,000/QALY was calculated for a 70-year-old man undergoing EVAR for a 5.5 cm AAA as compared with OSR; a result incompatible with the notion of overall cost-effectiveness. In this analysis, EVAR consistently produced an incremental cost-benefit ratio of > £30,000/QALY over a range of alternative cost and outcome scenarios.[367]

Previous analyses have suggested that cost effectiveness determinations for elective and urgent procedures should be calculated independently. Visser et al[368] evaluated the in-hospital and one-year follow-up costs of EVAR and OSR in patients with hemodynamically stable, acute symptomatic or ruptured AAA treated between 2001 and 2005. Total in-hospital costs and total overall costs, including one-year follow-up costs, were lower in patients who underwent EVAR. ICU admission and length of stay, primarily related to the incidence of complications, represented the single most important cost differential between EVAR and OSR.

In summary, given the evolutionary nature of EVAR devices, procedures, perceived procedural imperatives, as well as postoperative surveillance technologies and protocols, additional studies are required to identify approaches that improve cost effective care for patients with AAA. In the process of conducting these studies, we recommend that more comprehensive analytic strategies be employed to capture all costs associated with aneurysm repair, including societal costs associated with time away from work for patients and family members providing care. Moreover, detailed and disease-specific quality of life instruments should be developed, validated, and employed.

ANESTHETIC CONSIDERATIONS AND PERIOPERATIVE MANAGEMENT

Choice of anesthetic technique and agent

OSR. Except in unusual circumstances, open aneurysm repair requires general anesthesia, because of the required relaxation of the abdominal wall musculature.[369] Low doses of relaxing agents, such as a benzodiazepine plus an opiate, are recommended followed by conventional inhalation agent-based general endotracheal anesthesia. Insertion of monitoring lines prior to induction of anesthesia is appropriate if such monitoring devices improve the safety of induction.

Infusion of an analgesic through an epidural catheter, by controlling pain fiber input, appears to lower the required dose of general anesthetic agents and may be associated with a shorter time to extubation.[370] However, infection and bleeding risks along with the potential for delayed surgery in the event of a "bloody tap" may outweigh these benefits. Successful elective OSR using "mini-laparotomy" or by retroperitoneal exposure has been performed using epidural anesthesia with either low dose inhalation anesthesia or in the awake patient.[371-373] These techniques should be considered experimental.

EVAR. EVAR can be safely performed under general, epidural, or local anesthesia. Data from the EUROSTAR registry suggests lesser degrees of anesthesia may be of benefit.[374] From 1997 to 2004, 3,848 patients underwent EVAR using general anesthesia, 1,399 using epidural anesthesia, and 310 using local anesthesia. Local anesthesia was associated with shorter operative times, reduced ICU admission, shorter hospital stay, and fewer systemic complications. Epidural anesthesia showed some advantages over general anesthesia, but the effect was less dramatic. Several small single-institution reports have shown similar results.[375,376] Mortality differences have not been observed.

Ruptured AAA. OSR of a ruptured AAA will require general endotracheal anesthesia, as the benefits of expeditious total relaxation and pain control outweigh the risks of intubation, placement of a high epidural catheter, or administration of local anesthesia in an awake patient in significant pain. However, vasodilation on induction will often lead to sudden hypotension and the surgical team should consider prepping and draping the surgical field or placing a transbrachial or transfemoral balloon for thoracic aortic control prior to induction. Treatment of a ruptured AAA by EVAR may be performed under local anesthesia, if pain related to rupture can be appropriately controlled.

Open aneurysm repair should be performed in most patients under general endotracheal anesthesia.

Level of recommendation:	Strong
Quality of evidence:	High

Placement of an epidural catheter for postoperative analgesia is suggested for patients undergoing open aneurysm repair.

Level of recommendation:	Weak
Quality of evidence:	Low

Use of epidural and local anesthetic techniques along with conscious sedation is suggested for patients undergoing EVAR.

Level of recommendation:	Weak
Level of evidence:	Low

Antibiotic prophylaxis

Prophylactic treatment with systemic antibiotics, administered immediately preoperatively, reduces the risk of wound infection and almost certainly early graft infection among all forms of arterial reconstructive surgery by between three-quarters and two-thirds, respectively.[377] Stewart et al[377] reviewed 10 studies that randomised 1297 patients to receive either prophylactic antibiotic or placebo. Significantly, three studies demonstrated that antibiotic prophylaxis for more than 24 hours was without added benefit. No evidence existed of a significant advantage among comparable regimens of first or second-generation cephalosporins, penicillin/β-lactamase inhibitor, aminoglycosides, or vancomycin.

Intravenous administration of a first generation cephalosporin or, in the case of a history of penicillin allergy, vancomycin, is recommended within 30 minutes prior to skin incision. Prophylactic antibiotics should be continued for no more than 24 hours.

Level of recommendation:	Strong
Quality of evidence:	High

Intraoperative fluid resuscitation and blood conservation

Preoperative autologous blood donation avoids disease transmission and transfusion reactions, but is dependent on accurate labeling, storage, and retrieval. In addition, erythropoesis may be stimulated by this practice, but the magnitude of this benefit is unclear. Limitations include restricted use to elective surgery, limited volume of blood donation, increased clerical demands and expense, as well as waste of non-utilized donated blood.[378]

Intraoperative blood salvage during OSR can be achieved using either red blood cells (RBC) processors or hemofiltration devices and are attractive under conditions where the safety of banked blood is a concern.[379] Cell processors collect shed whole blood and wash and separate RBC by centrifugation. The resulting product is pure, with anaphylaxis, toxins, and other waste products removed with a high degree of efficiency. However, platelets and clotting factors are lost. In contrast, hemofiltration devices filter shed whole blood, such that water and some waste products are removed. The resulting whole blood reinfusion contains more clotting factors and cellular components, but at the cost of potential contaminants. While advantages should exist for RBC-salvage and ultrafiltration devices in the presence of potential contamination or coagulopathy, respectively, this has not been borne out clinically. After CABG, hemoglobin values were higher after direct RBC infusion and platelet counts higher after ultrafiltration, but coagulation parameters, blood loss, and outcomes were similar.[380] Likewise, cell salvage techniques during vascular surgery have not prevented the need for transfusion and have not proven cost-effective.[381] In a Cochrane review, cell salvage techniques reduced the need for RBC transfusion by 0.67 units per patient, but did not alter clinical outcome.[382] Routine use of cell salvage and ultrafiltration devices cannot be recommended, but is recommended if large blood loss is anticipated or disease transmission from banked blood likely. This is an area where further investigation is needed, particularly with regard to empiric benefits of different techniques and cost-benefit analysis.

The benefit of maintaining a hemoglobin of at least 10 gm/dL during OSR is unknown and randomized trials have not been conducted to address this question. It would seem prudent to have a lower threshold for transfusion in the presence of ongoing blood loss, but evidence is lacking.[383] Optimal blood replacement therapy during complex OSR has not been defined and research in this area is encouraged. In the trauma population, plasma and packed RBCs in equal proportions, plasma, packed RBCs, and platelets in a 1:1:1 ratio, and warm fresh whole blood instead of component therapy have all been advocated.[384-386] It is recommended that transfusion be considered during OSR if blood loss is ongoing or expected and the hematocrit is less than 30%. Blood product-based resuscitation strategies are preferred with liberal use of platelets, plasma, and perhaps fresh whole blood.

Intraoperative blood salvage during OSRIntravenous fluids for abdominal aortic surgery has been the topic of many investigations over the past three decades, however, there is no overwhelming evidence in favor of the preferential use of any specific type of fluid or fluid regimen.[387] Moreover, there is no evidence of the beneficial effects of combination fluid therapy, with colloid and crystalloid. Although the cost of fluid is small, a positive fluid balance after OSR may be predictive of major adverse events, increased ICU and overall hospital length of stay.[388]

Preoperative autologous blood donation may be beneficial for patients undergoing open aneurysm repair.

Level of recommendation:	Weak
Quality of evidence:	Moderate

Cell salvage or an ultrafiltration device is recommended if large blood loss is anticipated or the risk of disease transmission from banked blood considered high.

Level of recommendation:	Strong
Quality of evidence:	Weak

Intraoperative blood transfusion is recommended for a hematocrit <30% in the presence of ongoing blood loss.

Level of recommendation:	Strong
Quality of evidence:	Weak

If the intraoperative hematocrit is <30% and blood loss is ongoing, resuscitation fluids should consider use of FFP and platelets in a ratio with packed blood cells of 1:1:1.

Level of recommendation:	Strong
Quality of evidence:	Weak

Intra- and postoperative cardiovascular monitoring

Clinical studies have not demonstrated altered outcome from routine use of either a pulmonary artery catheter, transesophageal echocardiography, ST-segment monitoring, or intravenous nitroglycerin. Pulmonary artery catheters have been used since the 1980s to measure cardiac output and index, outflow resistance, and central venous and pulmonary artery pressure, and estimate contractility. They are moderately invasive, though safe when used correctly. However, multiple randomized trials have shown no significant benefit when used routinely in non-selected patients.[389-391] In one randomized study, central venous pressure (CVP) measurements were found to be just as helpful as formal pulmonary artery catheterization.[390] Peripherally placed central catheters (PICCs) have been shown to correlate with conventional CVP measurements in aortic surgery.[392] However, PICC lines are inadequate for high volume infusion of fluid. The risks, logistics, and expense of routine transesophageal echocardiography (TEE) during open AAA repair exceed the benefits. However, in an unstable patient TEE can be used to estimate volume status, stroke volume, and cardiac output,[393] and can identify tamponade or valvular dysfunction.[394]

Early postoperative MI (40%) most often occurs suddenly without a warning period of ischemia, presumably due to rupture of a vulnerable plaque. Delayed MI (60%), occurring 24 to 72 hours postoperatively, typically is preceded by a period of electrically and chemically detectable myocardial ischemia, and probably related to prolonged stress.[63] This suggests that early identification of myocardial ischemia may reduce the risk of MI, by identifying the need for aggressive control of heart rate and attention to analgesia, oxygenation, and volume status.[63] Improved strategies to identify postoperative patients at risk for MI are needed and is an area recommended for further study. Moreover, little is known about the ability of intervention at the time of identification of myocardial injury to alter clinical outcomes.

Currently, troponin measurement is recommended for all patients with postoperative ECG changes, chest pain, or other signs of cardiovascular dysfunction, since troponin elevation is predictive of adverse short- and long-term outcomes.[395-397] Conventional ECG monitoring uses five leads, of which only one is precordial and most often placed at V5. In a study of 185 consecutive patients undergoing vascular procedures, full 12-lead ECGs with computerized ST-segment analysis and routine troponin measurements for up to 72 hours postoperatively showed transient myocardial ischemia in 21% of patients and MI in 6.5%. Leads placed at V3 and V4 were most predictive and the combination of two precordial leads led to greater than 95% sensitivity when compared to troponin levels.[398] A similar study showed abnormal troponin levels in 14% and MI in 5% of patients following aortic surgery.[63] Patients at increased risk of a cardiac event following EVAR or OSR should be considered for ECG monitoring and measurement of postoperative troponin levels.

Pulmonary artery catheters should not be used routinely in aortic surgery, unless there is a high risk for a major hemodynamic disturbance.

Level of recommendation:	Strong
Quality of evidence:	High

Central venous access is recommended for all patients undergoing open aneurysm repair.

Level of recommendation:	Strong
Quality of evidence:	High

Intraoperative and postoperative ST-segment monitoring is recommended for all patients undergoing open aneurysm repair, but required in only selected patients undergoing EVAR.

Level of recommendation:	Strong
Quality of evidence:	Moderate

Transesophageal echocardiography can be beneficial in determining the cause of an intraoperative hemodynamic disturbance.

Level of recommendation:	Weak
Quality of evidence:	Moderate

Postoperative troponin measurement is recommended for all patients with ECG changes or chest pain after EVAR or OSR.

Levels of recommendation:	Strong
Quality of evidence:	High

Maintenance of body temperature

Maintenance of body temperature during aneurysm repair is beneficial. In one trial randomizing patients to two methods of warming, patients who were hypothermic (<36 °C) on arrival to the post-anesthesia care unit (PACU) had lower cardiac output and platelet counts, higher prothrombin times and APACHE II scores, and a greater incidence of sinus tachycardia and ventricular arrythmias.[399] Forced air warming blankets have been shown to be beneficial[400] when compared with circulating water mattresses alone.[399] Warmed inhaled gasses[401] and infused liquids[402] have also been shown to be of benefit. The benefit of forced air-warming blankets applied to the lower extremities, as opposed to the upper trunk, during prolonged aortic clamping is unknown.

Core body temperature should be maintained at or above 36 °C throughout OSR or EVAR.

Levels of recommendation:	Strong
Quality of evidence:	High

The role of the ICU

Typically, all patients are admitted to an ICU setting following OSR, while most patients undergoing EVAR are not. Although distinctions are increasingly blurred,

an ICU, by virtue of a higher nurse to patient ratio and more intensive monitoring, differs from a non-ICU setting in that it allows ventilatory support, arterial and venous pressure monitoring, and infusion of cardio- and vasoactive drugs. Thus, there is an unavoidably greater cost.[403] Some patients probably do not need ICU support even after OSR. One recent study showed that the majority of such patients could be managed without ICU admission, although "overnight intensive recovery" in the postoperative care unit was provided.[404] Another group reported selective ICU admission after four hours of observation in the PACU for patients eligible for "fast track" OSR. Although mean ICU stay in 30 patients was 0.87 ± 0.10 days, the percentage of patients sent directly to the floor was not reported.[405] In a second report from a large academic practice, selective use of the ICU following OSR was achieved in 56% of patients. While length of stay decreased, mortality and morbidity were not different.[406]

Postoperative ICU care can be useful for patients undergoing open aneurysm repair and in selected patients undergoing EVAR.

Level of recommendation:	Strong
Quality of evidence:	Moderate

Postoperative nasogastric decompression and perioperative nutrition

Open aneurysm repair involves laparotomy or a large retroperitoneal exposure and in either case the viscera are either extensively mobilized or firmly retracted. Thus, many clinicians routinely use nasogastric (NG) drainage for several days postoperatively. This practice, however, may be unnecessary and perhaps even detrimental. In a randomized trial, 80 patients undergoing OSR had their NG tube removed at extubation or left until flatus. No difference in outcome was observed, and only three of 40 patients without initial placement of a NG tube required insertion.[407] In a more recent study, a higher incidence of respiratory complications was noted among patients with nasogastric suction.[408]

Preoperative malnutrition adversely affects outcome after surgery. However, perioperative total parenteral nutrition is of benefit only if support is continued for several weeks. Patients undergoing elective EVAR would not be expected to have an ileus or require NG decompression.

Nasogastric decompression should be used intraoperatively for all patients undergoing open aneurysm repair, but in only selected patients postoperatively.

Level of recommendation:	Strong
Quality of evidence:	Moderate

Elective open aneurysm repair should not be performed unless nutritional status is optimized.

Level of recommendation:	Strong
Quality of evidence:	High

Parenteral nutrition is recommended if a patient is unable to tolerate enteral support seven days after OSR or EVAR.

Level of recommendation:	Strong
Quality of evidence:	High

Perioperative pain management

Guidelines for acute pain management published by the American Society of Anesthesiologists report similar efficacy of patient controlled anesthesia (PCA) as compared with nurse or other staff-administered intravenous (IV) analgesia.[409] Overall patient satisfaction with PCA is high. In order to avoid inadequate analgesia after a period of sleep or unconsciousness, continuous low dose infusion of an analgesic has been incorporated into most PCA protocols. Postoperative epidural anesthesia also provides excellent pain relief. A Cochrane review reports a meta-analysis of 13 randomized trials involving 1,224 patients having abdominal aortic surgery, 597 of whom were treated using epidural delivery and 627 using systemic opiates. Those receiving epidural anesthesia had significantly fewer overall cardiovascular complications, myocardial infarction, respiratory failure, gastrointestinal complications, and renal insufficiency. Patients spent 20% less time intubated and reported less subjective pain, especially with movement. A thoracic epidural approach appeared to yield better outcomes than lumbar. No mortality differences were noted.[410] These potential benefits are associated with increased costs, which in one study of 80 patients averaged $2,489 for those treated with epidural anesthesia versus $443 for those treated with IV PCA.[411] In addition, patients on antiplatelet or anticoagulant therapy may be at increased risk of epidural hematoma.[412]

Epidural anesthetic or intravenous PCA are recommended for postoperative pain control after OSR.

Level of recommendation	Strong
Quality of evidence	High

Intramuscular delivery of opiates is not recommended for ongoing analgesia following aortic surgery.

Level of recommendation:	Strong
Quality of evidence:	Moderate

Perioperative prophylaxis for deep vein thrombosis

It has been assumed that the risk of deep vein thrombosis (DVT) in patients undergoing vascular surgery with systemic heparinization is low.[413] However, DVT after open AAA repair appears to be underappreciated. In a series of 50 consecutive patients undergoing OSR without prophylaxis, postoperative venography revealed acute DVT in 21% of patients, nearly 80% of which involved the calf veins, none of whom were symptomatic.[414] A Cochrane analysis of all non-randomized prospective studies in aortic surgery identified an incidence of all DVT that ranged between 0% and 20.5%, averaging 9.2%, among patients without DVT

prophylaxis.[415] This incidence was 2.6% if calf DVT was excluded. Although a reduced risk would be anticipated after EVAR, the incidence of femoral or popliteal DVT after endovascular repair was 6% among 50 patients examined by Duplex ultrasonography.[413] Consistent with these findings, deMaistre et al[416] recently reported an incidence of lower extremity DVT of 10.2% after OSR and 5.3% after EVAR ($P = .28$), despite prophylaxis with thigh-length compression bandages or stockings, early mobilization, and daily subcutaneous injection of low-molecular-weight heparin beginning in most patients within the first day after OSR or EVAR. Symptomatic pulmonary embolism (PE) occurred in 1.4% of patients after OSR, but most patients identified with DVT were asymptomatic. There was a trend toward delayed initiation of low dose heparin prophylaxis among patients that developed a DVT (1.7 days vs 0.9 days, $P = .09$). Indeed, aortic surgery may be associated with a higher risk of DVT than infrainguinal bypass. Hollyoak et al[417] reported an incidence of DVT 41% after aortic surgery compared with 18% after peripheral artery surgery among patients who did not receive heparin prophylaxis. Among patients receiving low dose subcutaneous heparin or LMWH, Farkas et al[418] reported that 8% of patients developed DVT after aortic surgery with an incidence of 3% after distal bypass. Similarly, Fletcher et al[419] noted an incidence of DVT in 12% and 9% of patients receiving low dose heparin prophylaxis after aortic surgery and lower extremity bypass, respectively.

Overall, most patients undergoing EVAR or OSR can be considered at moderate to high risk for DVT, given advanced age, duration of surgery >45 min, and the increasing prevalence of obesity in the US population. Therefore, DVT prophylaxis consisting of intermittent pneumatic compression and early ambulation are recommended for all patients undergoing OSR or EVAR. Patients at high risk (eg, prior history of DVT/PE, obesity (BMI >25), limited mobility status, malignancy, hypercoaguable state) should receive either low molecular weight heparin (LMWH) (enoxaparin 40 mg SQ once a day) or unfractionated heparin (5000 IU SQ two or three times a day) initiated within 24 hours per the judgement of the treating surgeon. If a high-risk patient has a history of renal insufficiency, unfractionated heparin (5000 IU SQ twice a day) is preferred, which is also favored for those patients who have an epidural catheter. A detailed review of DVT and methods to reduce risk after surgery has recently been published.[420]

DVT prophylaxis consisting of intermittent pneumatic compression and early ambulation are recommended for all patients undergoing OSR or EVAR.

Level of recommendation:	Strong
Quality of evidence:	High

Low dose heparin prophylaxis should be considered for patients at high risk for DVT undergoing aneurysm repair.

Level of recommendation:	Strong
Quality of evidence:	High

POSTOPERATIVE AND LONG-TERM MANAGEMENT

Late outcomes after open surgery and EVAR

Both EVAR and OSR are associated with late complications.[19,257,421] Given the routine use of postoperative imaging after EVAR, the incidence of complications has been better documented than for OSR.[19,257] Clinically significant complications appear to occur more frequently after EVAR, but this technology continues to evolve and newer endografts are associated with a lower incidence of migration, disconnection, and material fatigue. Nonetheless, the incidence of certain procedure specific complications, such as Type II endoleaks, remains unchanged.[421,422]

Long-term complications related to the incision. Retroperitoneal incisions for AAA repair have been associated with weakened lateral abdominal wall musculature and a bulge in up to 15% of patients.[195] The more commonly used laparotomy for transperitoneal AAA repair is, however, associated with a higher incidence of late small bowel obstruction, and approximately one in five patients may develop a ventral hernia, a finding that appears to be substantially more common after treatment of AAA than aortic occlusive disease.[423] Surgical exposure of the femoral arteries is common to both EVAR and OSR. Postoperative seromas and femoral nerve injury, manifest by anterior and inner thigh paresthesias, are well documented but infrequent events. Percutaneous approaches for EVAR will likely reduce these problems, but increase the risk of hematoma or pseudoaneurysm formation.

Para-anastomotic aneurysm. Para-anastomotic aneurysms after AAA repair include both false aneurysms resulting from a disruption of the anastomosis and true aneurysms that develop adjacent to the anastomosis. True metachronous aneurysms reportedly occur at a 1.5 to threefold greater frequency than anastomotic pseudoaneurysms. However, the incidence of para-anastomotic aneurysms is not clear, since few studies have systematically followed patients after OSR with serial imaging. Predisposing factors include hypertension, COPD, and tobacco use.[424-428] In the era prior to CT imaging, Szilagyi analyzed a 15-year experience with OSR in which anastomoses in the femoral region were at highest risk (3%), followed by the iliac (1.2%) and infrarenal aorta (0.2%).[427] Subsequent studies have reported an incidence of between 4% and 10% at 10-year follow-up.[425] In one study of 511 patients, Kaplan-Meier analysis has shown a probability of a para-anastomotic aneurysm of 0.8% at five years, 6.2% at 10 years, and 35.8% at 15 years.[426] The likelihood that 15 years after OSR 20% to 40% of patients may have a para-anastomotic aneurysm has been confirmed by others, especially among those patients treated with an aortobifemoral graft.[425,428] Indolent graft infection should be suspected in all pseudoaneurysms.

Given the inability to precisely differentiate anastomotic disruption from degenerative aneurysmal dilatation, indications for repairing para-anastomotic aneurysms are not well defined. Clearly large size and rapid enlargement

are indications for intervention. Redo OSR carries a significant risk of major morbidity and mortality. Thus, the successful application of endovascular repair when anatomically appropriate has been a welcome approach to this difficult problem.[429,430]

Graft infection. All implanted prostheses, whether placed by OSR or EVAR, are at risk for infection either at implantation or later by hematogenous seeding. This complication is rare and represents about 0.3% of all aortic operations.[431] Graft infection, however, is the indication for intervention in up to 25% of redo aortic surgery.[432] Although controversial, the risk of graft infection after EVAR may be lower than OSR, perhaps due to delivery of the endoprosthesis through a completely enclosed system or decreased dissection around the viscera. The EUROSTAR registry reported only three procedures for endograft infection in almost 3000 patients followed up to five years; a rate of 0.1%.[421] The EVAR-1 trial, however, showed a comparable incidence between OSR and EVAR over a four year follow-up period.[337] Similarly, in a recent analysis of more than 45,000 Medicare beneficiaries, graft infection or fistula at four years was 0.2% among patients treated by EVAR and 0.3% for those who underwent OSR ($P = .13$).[34] Likeiwse, in a review of 14,000 patients undergoing AAA repair in Washington State, Vogel et al[433] reported a nearly identical two-year incidence of graft infection of less than 0.2% among those treated with OSR or EVAR. Graft infection after EVAR or OSR may present in isolation or in association with a fistula to a neighboring viscus, most commonly the duodenum.[434,435]

Primary aortic graft infection usually presents late, on the average three years after implantation and on occasion much later.[432] Femoral extension of the abdominal grafts increases the incidence of graft infection from 1% to nearly 3%, as determined by an 18-year review of 664 patients treated by OSR.[436] Other predisposing factors include surgical revision and emergency surgery. Presentations can be quite diverse including generalized sepsis, groin purulence and drainage, pseudoaneurysm formation, or ill defined pain.[437] Staphylococcal organisms are the most frequent bacterial isolates. Although the diagnosis may be obvious, CT scanning usually provides the most information about the nature of the problem, extent of infection, and other associated abnormalities. Angiography may be required to plan therapy, especially if the infection involves the femoral region precluding use of the common femoral artery as an outflow for the reconstruction.

Treatment traditionally includes excision of all infected graft material with extra-anatomic reconstruction, particularly in the presence of extensive contamination. Outcome of treatment is poor with elevated mortality and limb loss.[432,438-442] Rupture of the infected aortic stump after aortic closure does occur and is almost always uniformly fatal. Many treatment strategies have been explored with various degree of success. Reilly demonstrated improved survival after staging the procedure, starting with the extra-anatomic reconstruction and, in a separate procedure, performing the excision and debridement of the infected field.[441] In-situ reconstruction using femoral vein, silver or antibiotic impregnated grafts, or arterial homografts, have all been advocated as surgical options that may be associated with reduced overall mortality in selected patients with limited contamination.[438,443-447]

Aortoenteric fistula (AEF) can complicate a graft infection in 1% to 2% of patients.[434,437] Although the duodenum is most frequently affected, all viscera, including small and large bowel, have been implicated.[435,437] A common presentation is upper gastrointestinal (UGI) bleeding.[448] Herald bleeds may ultimately progress to an exsanguinating hemorrhage, if the source of the bleeding is not identified and treated promptly. All patients with a previous aortic graft and an UGI bleed should be suspected of having an AEF. The diagnosis of AEF is one of exclusion and is occasionally confirmed by endoscopy or CT scanning.[449-451] Bleeding presentations are more common when the anastomosis erodes into the GI tract, while sepsis and abscess formation may be more common with para-prosthetic fistula involving the body of the graft. Treatment strategies are similar to primary graft infections but must include closure of the visceral tear and on rare occasion diversion of the GI tract. Anastomotic erosion may be less contaminated than the paraprosthetic presentation and more suitable for in-situ replacement. Separation of the graft from the viscera, which is desirable at the original operation, is necessary during the repair of an AEF.[452]

Antibiotic prophylaxis of graft infection is required prior to bronchoscopy, gastrointestinal or genitourinary endoscopy, and any dental procedure that may lead to bleeding.

Level of recommendation: Strong
Quality of evidence: High

Generalized sepsis, groin drainage, pseudoaneurysm formation, or ill-defined pain after OSR or EVAR should prompt evaluation of graft infection.

Level of recommendation: Strong
Quality of evidence: High

GI bleeding after OSR or EVAR should prompt evaluation of an aortoenteric fistula.

Level of recommendation: Strong
Quality of evidence: High

Excision of all graft material along with aortic stump closure with an omental flap and extra-anatomic reconstruction is recommended for treatment of an infected graft in the presence of extensive contamination.

Level of recommendation: Strong
Quality of evidence: High

In situ reconstruction with deep femoro-popliteal vein after graft excision and debridement is a recommended option when contamination is limited.

Level of recommendation: Strong
Quality of evidence: High

In situ reconstruction with silver or antibiotic impregnated grafts, arterial homografts, or a PTFE graft may be considered in patients with an infected prosthesis and limited contamination.

Level of recommendation:	Weak
Quality of evidence:	Low

Limb occlusion. Nearly 25% of all arterial reinterventions after OSR are due to limb occlusion, and are most common in patients with associated occlusive disease.[432] Limb occlusion appears to be greater in women and in grafts extending to the femoral artery. Isolated limb occlusion usually presents with claudication, but occlusion of the entire graft may present with severe ischemia. On occasion, the patient presents before complete occlusion of the graft.

Endografts are at a higher risk for limb thrombosis than prostheses placed during the course of OSR, as observed in the EVAR-1 trial.[337] Endograft limbs can be narrowed by a calcified small diameter aortic bifurcation or tortuous, angulated, and diseased iliac arteries. Non-supported limbs, as was the case with the Ancure endograft, were at high risk of limb occlusion and required frequent stenting.[453] However, even stented limbs may occlude due to infolding, kinking between stents, or by the abutting the arterial wall in tortuous iliacs.[421] Stenotic limbs, occasionally noted by Duplex examination or as a result of a reduction in ankle-brachial index or new onset of claudication, can be successfully treated by additional stenting.

Treatment of an occluded limb after EVAR or OSR includes, thrombectomy or lytic therapy with secondary endovascular or local surgical intervention, or extra-anatomic bypass, such as femoral-femoral or axillo-femoral bypass. Standard mechanical balloon thrombectomy is less likely to be successful with EVAR grafts because of sharp edges produced by stents and concerns related to dislodging or disrupting the sealing zones.

Follow-up of patients after EVAR or open surgery should include a thorough lower extremity pulse exam or ABI.

Level of recommendation:	Strong
Quality of evidence:	High

New onset of lower extremity claudication, ischemia, or a reduction in ABI after OSR or EVAR should prompt an evaluation of graft limb occlusion.

Level of recommendation:	Strong
Quality of evidence:	High

Endoleak. Endoleak, or persistent blood flow in the aneurysm sac outside of the endograft, is the most frequent complication after EVAR and has been reported in nearly one in four patients at some time during follow-up.[421,454,455] It is one of the most common abnormalities identified on late imaging and used to justify lifelong follow-up of these patients. Diagnosis of endoleak is most commonly performed by CT imaging, although Duplex imaging can be effective.[456,457] An endoleak can connect an inflow source with an outflow vessel resulting in elevated systemic pressures in the sac. When an outflow path does not exist, diastolic pressure in the sac is higher than the systemic pressure and the net effect is a higher mean pressure in the sac.[458] Four types of endoleak have been described, independent of graft type:[459,460]

Type I endoleak occurs in the absence or loss of complete sealing at the proximal (**Type IA**) or distal (**Type IB**) end of the stent graft. Type I endoleak is associated with significant pressure elevation in the sac and has been linked to a continued risk of rupture.[461-463] Incidence of Type I endoleak increases with difficult anatomic situations, such as short or angulated necks, and landing zones with calcification, tortuosity, or uneven size. Every attempt should be made to resolve Type I endoleaks noted at the time of EVAR before the patient leaves the intervention suite. On occasion, small persistent Type I endoleaks may be observed and if endovascular intervention has been unsuccessful, the only alternative is surgical conversion. Some may seal spontaneously by the time of the first postoperative surveillance study.

Proximal Type IA endoleaks can respond to expansion with less compliant balloons, extension if any additional landing zone exists, or placement of a balloon expandable stent.[22] If none of these maneuvers work, obliteration of the luminal crease with coils, glue or other embolic agents may be effective.[464,465] Distal Type I endoleak are usually treated by distal extension. Should a Type I endoleak persist, conversion to OSR is appropriate, especially in patients with large aneurysms who can undergo OSR.[466]

Type II endoleaks are the most common form of endoleak and arise from retrograde filling of the sac by lumbar branches or the inferior mesenteric artery.[422,454,455] Detection of a Type II endoleak may be difficult if the endoleak is associated with low flow. For those detected at the time of EVAR, further treatment is not indicated, since spontaneous resolution is possible.[461,464,465,467] When noted at follow-up, many resolve spontaneously, but some may persist. Endoleaks arising from the inferior mesenteric artery are thought to resolve less frequently than those from lumbar vessels and may be associated with a greater risk of sac expansion.[468]

Although delayed AAA rupture secondary to a Type II endoleak has been reported, it is rare and many patients with Type II endoleaks are observed without treatment. Some patients will show some sac shrinkage, an indication of low pressure in the aneurysm sac. The majority will show no change in sac size and require continued surveillance. A risk benefit analysis of close follow-up versus early intervention should take into consideration the age of the patient, size of the aneurysm, the vessels involved, and the expected efficacy of treatment. A definite subset of patients with Type II leaks will demonstrate sac enlargement, an indication of elevated pressure, and increased risk of rupture. Treatment of these Type II endoleaks is recommended.[23,469,470]

Obliteration of Type II endoleaks can be difficult. Transarterial retrograde catheterization of the offending branches with occlusion by coiling or other embolic agents can be effective, but requires advanced endovascular skills.[465]

Translumbar direct puncture of the aneurysm sac can also be utilized successfully.[458] The principle of treatment is to eliminate the branches at their junction with the aneurysm. Laparoscopic ligation of the inferior mesenteric artery or lumbar arteries is a third option for treatment of a type II endoleak.[471]

Type III endoleaks arise from poorly seated modular connections or from disconnection and separation of components. Less often it is the result of fabric erosion related to material fatigue. Sac pressure can exceed arterial pressure. All Type III endoleaks should be treated, typically with limb components, as they represent a lack of exclusion of the aneurysm with repressurization of the aneurysm sac.[461,462,472]

Type IV endoleaks represents self-limiting blood seepage through the graft material due to porosity and treatment is not required. Typically, this form of endoleak is only noted at the time of repair on post-implantation intra-operative angiography. An endoleak noted on follow-up imaging should not be considered a Type IV endoleak.

Type I endoleaks should be treated.

Level of recommendation:	Strong
Quality of evidence:	High

Treatment is recommended for Type II endoleaks associated with AAA expansion.

Level of recommendation:	Strong
Quality of evidence:	Moderate

Treatment may be considered for Type II endoleaks not associated with AAA enlargement.

Level of recommendation:	Strong
Quality of evidence:	Moderate

Type III endoleaks should be treated.

Level of recommendation:	Strong
Quality of evidence:	High

Type IV endoleaks do not require treatment.

Level of recommendation:	Strong
Quality of evidence:	Moderate

Conversion to OSR of an AAA is recommended if a Type I or III endoleak does not resolve with endovascular treatment.

Level of recommendation:	Strong
Quality of evidence:	High

Conversion to OSR of an AAA is recommended for a Type II endoleak in association with a large or expanding aneurysm that does not resolve with endovascular or laparoscopic treatment.

Level of recommendation:	Strong
Quality of evidence:	High

Endotension. An AAA may continue to enlarge after endovascular repair, even in the absence of a detectable endoleak, and this enlargement may lead to aneurysm rupture. Explanations for persistent or recurrent pressurization of an aneurysm sac include blood flow that is below the sensitivity limits for detection with current imaging technology or pressure transmission through thrombus or endograft fabric.[473,474] Additionally, a serous ultrafiltrate across a microporous fabric can fill the aneurysm and increase pressure.[422,475] Since sources of endotension can be difficult to detect, treatment strategies must be individualized. Relining devices with low porosity alternatives may abolish sac growth or induce shrinkage of the sac.[476] On occasion, explantation and conversion may be required when no clear cause can be detected and endoleak, as a cause of sac expansion, cannot be excluded.

Treatment of endotension to prevent aneurysm rupture is suggested in selected patients with continued aneurysm expansion.

Level of recommendation:	Weak
Quality of evidence:	Low

Device migration. Device migration after EVAR is multi-factorial and can be asymptomatic. It is normally detected on CT scan by the presence of a Type I endoleak and can lead to repressurization of the aneurysm sac and rupture. Although cranial migration of distal iliac attachment can occur and may have a similar effect in pressurizing the aneurysm sac, the most common form is caudal migration of the proximal aortic neck attachment site. The incidence of device migration is influenced by its definition (5 mm or 10 mm) and technique used to measure displacement.[477,478] In this regard, the use of three-dimensional CT reconstruction of thin slices (<3 mm) using a center-line of flow methodology provides the most accurate measurements.

Device migration can occur intraoperatively or subsequent to device implantation. Intraoperative device migration of the proximal attachment component is usually managed by the addition of a proximal aortic cuff. Most concur that proximal attachment deployment should be performed as close as possible to the inferior border of the lowest renal artery. Appropriate magnification and angulation of the C-arm provides optimal views of the landing zones increases the reliability of deployment.

The incidence of postoperative device migration appears related to the duration of follow-up. Most series evaluating device migration have reported increases after 24 months.[273,478-480] Migration rates betwen 0% and 45% have been reported in selected series with the largest migration rates reported after more than 24 months and with devices that are no longer commercially available.[479-481] Some of the anatomic factors that influence proximal attachment device migration include: length of the aortic neck (<15 mm), shape of the aortic neck (conical vs. straight), angulation of the aortic neck relative to centerline (>45°), presence of thrombus in the aortic neck, excessive over sizing of the endografts (>20%), and characteristics of the device (suprarenal fixation, presence of hooks or barbs, radial force alone).[278,480,481]

Component dislocation or disruption. Device integrity was a major concern in a number of first generation endografts (Stentor, Vanguard, and Challenger). The five

current commercially available endografts in the United States have not demonstrated a significant problem with device integrity in short and mid-term follow-up.[329] Component dislocation has been reported and it is usually related to either insufficient overlap of the limb component in the main body cage, decreased overlap of the limb in the iliac artery or occasionally a combination of these factors. Shrinking of the aneurysm sac, creating upward forces can dislocate the distal iliac limb into the aneurysm sac causing a Type I endoleak with sac pressurization and potential rupture. It is recommended that the iliac limb be extended at least two to three cm into the common iliac artery and preferably down to the bifurcation. When a proximal aortic cuff is necessary, sufficient overlap with the main body is necessary to avoid component separation.

Material fatigue, including metallic fracture, fabric fatigue, and suture breakage, has been described as minor (asymptomatic) or major (defined as material disruption associated with deleterious outcomes such as endoleak and aneurysm expansion and requires intervention). Jacobs et al evaluated the role of material fatigue in a 10-year experience of 686 patients treated with EVAR and TEVAR, most of which were cases of stent fractures in devices no longer available.[482,483] The relationship of material fatigue to clinical outcome has been reviewed both radiographically,[484] and from careful examination of explanted grafts.[484,485] Zarins et al[484] could not identify a clear relationship between material fatigue and adverse clinical outcome. Nearly all explanted grafts demonstrated some type of material fatigue, including those explanted at autopsy in patients whose cause of death was unrelated to their aneurysm. Metallic fractures were found in 66%, fabric holes in 45%, and suture breaks in almost all explanted grafts. However, in those patients with or without fabric holes, there was no difference in rate of endoleak or aneurysm enlargement. Interestingly, in explanted grafts with metallic fracture, there was an increased rate of migration, although most structural abnormalities were remote from the fixation site. Material fatigue was also noted in patients with significant aortic angulation.

Recommendation for postoperative surveillance

The primary goal of AAA treatment is to prevent rupture. As opposed to EVAR, OSR is not associated with a risk of persistent sac enlargement, but may be associated with late paranastomotic aneurysm formation or graft infection. Although the later event is rare, late aneurysm formation may be noted in approximately 1%, 5%, and 20% of patients at five, 10, and 15 years after OSR, respectively.[425,428] Thus, we recommend follow-up CT imaging at five-year intervals after OSR.

EVAR using commercially available devices has been associated with a low rate of aneurysm related death. Five-year results of FDA clinical trials involving three commercially available devices (Excluder, Zenith, and AneuRx) demonstrate freedom from aneurysm rupture of 100%, 96.8%, and 100%, respectively.[329,422,486] Similar results have been reported at 48 months for the Powerlink endograft.[330] Nonetheless, late aneurysm rupture is a potential risk of all devices. Therefore, continued surveillance after EVAR to detect aneurysm growth, due to endoleak, device migration, or structural failure is recommended. Protocols for EVAR surveillance established as an outgrowth of initial FDA sponsored pivotal trials consist of CT imaging at one, six, and 12 months after initial repair and yearly thereafter.[259,487] However, the frequent use of CT scanning has raised concerns related to the added costs of these studies, as well as cumulative radiation exposure and potential lifetime cancer risk.[488] Although ultrasound avoids radiation exposure and use of nephrotoxic contrast agents, concerns have been raised in the past regarding the variable sensitivity of ultrasound in identifying endoleaks.[456,489-491] Ashoke and collaborators[456,492,493] in a meta-analysis of 10 published studies comparing contrast enhanced CT with color Duplex ultrasonography (CDU), found a sensitivity and specificity of 69% and 91%, respectively, with greater sensitivity in detecting Type I and III endoleaks than Type II endoleaks. Recent studies, however, have suggested that the lower sensitivity of CDU is offset by a high degree of correlation between CDU and CT imaging in detection of clinically significant endoleaks.[493-496] Moreover, recent small studies evaluating the role of contrast-enhanced ultrasound in the detction of endoleaks report increased sensitivity (97.5% vs 62.5%), negative predictive value (97.3% vs 65.1%), accuracy (89.3% vs 63.1%), and specificity (81.8% vs 63.6%) when compared with CDU.[497-499] The utility of ultrasound is primarily limited in obese patients or those presenting with substantial bowel gas or a large ventral hernia.

Based on these recent reports, some investigators have suggested that follow-up with CDU as the sole imaging modality is appropriate, if neither an endoleak nor AAA enlargement is documented on the first annual CT scan.[500] A significant increase in aneurysm size or new onset of endoleak, if detected by CDU at later follow-up, would prompt CT imaging.[501] Eliminating the traditional six month CT scan has also been recommended, if CT imaging one month after EVAR does not identify an endoleak.[500,502] Makaroun and colleagues[502,503] have reported that these protocols can be instituted safely with minimal risk of an adverse clinical event. Further research is needed to confirm the broader efficacy of these modified protocols. It should also be noted that while risk for endoleak declines as the number of negative postoperative scans increases, new endoleaks may be identified as late as seven years following EVAR.[504] Convention has also dictated that Type II endoleaks, in the absence of aneurysm enlargement, should be followed with CT imaging at six-month intervals. However, Type II endoleaks in the presence of a shrinking or small stable aneurysm are characterized by a relatively benign natural history in most cases.[505] Thus, further studies may demonstrate that CDU at six or even 12-month intervals may be a reasonable alternative, especially for patients whose aneurysms are less than 6.5 cm in diameter.[470]

In 2005, the FDA approved an AAA Pressure Management System. The sensor is implanted in the aneurysm sac at the time of EVAR deployment and the APEX trial confirmed that intrasac pressure and pressure changes could be reliably measured.[506] This system pro-

vides an intraoperative tool to assess aneurysm exclusion by EVAR. As an alternative to postoperative CT imaging, algorithms to identify a significant endoleak based on pressure changes are under evaluation.

We currently recommend contrast enhanced CT imaging one and 12 months during the first year after EVAR. Should CT imaging at one month after EVAR identify an endoleak or other abnormality of concern, postoperative imaging at six months should be added to further evaluate the proper exclusion of the aneurysm. If neither an endoleak nor aneurysm enlargement is documented during first year after EVAR, Color Duplex ultrasonography may be a reasonable alternative to CT imaging for postoperative surveillance. However, these studies should be performed by a skilled technician in an accredited non-invasive vascular laboratory. Likewise, follow-up with CDU and non-contrast CT imaging is reasonable for patients with renal insufficiency at any time after EVAR. The presence of a Type II endoleak should initially prompt continued CT surveillance to ascertain whether the aneurysm is increasing in size. If the aneurysm is shrinking or stable in size, follow-up with CDU may be a reasonable alternative to continued CT imaging. Detection of a new endoleak after prior imaging studies have suggested complete aneurysm sac exclusion should prompt evaluation for a Type I or Type III endoleak. Given the risk of paraanastomotic aneurysm, non-contrast CT imaging at five-year intervals is recommended for patients after OSR.

Surveillance during the first year after EVAR should consist of contrast enhanced CT imaging at one and 12 months.

Level of recommendation: Strong
Quality of evidence: High

If a Type II endoleak or other abnormality of concern is observed on contrast enhanced CT imaging at one-month after EVAR, postoperative imaging at six months is recommended.

Level of recommendation: Strong
Quality of evidence: High

If neither endoleak nor AAA enlargement is documented during first year after EVAR, Color Duplex ultrasonography is suggested as an alternative to CT imaging for annual postoperative surveillance.

Level of recommendation: Weak
Quality of evidence: Low

The presence of a Type II endoleak should initially prompt continued CT surveillance to ascertain whether the aneurysm is increasing in size. If the aneurysm is shrinking or stable in size, follow-up with CDU is suggested as an alternative to continued CT imaging.

Level of recommendation: Weak
Quality of evidence: Low

A new endoleak that is detected after prior imaging studies have suggested complete aneurysm sac exclusion should prompt evaluation for a Type I or Type III endoleak.

Level of recommendation: Strong
Quality of evidence: High

Color Duplex ultrasonography and a non-contrast CT scan are recommended as a substitute for contrast enhanced CT imaging for post-EVAR surveillance of patients with renal insufficiency.

Level of recommendation: Strong
Quality of evidence: High

Non-contrast CT imaging of the entire aorta is recommended at five-year intervals after OSR or EVAR.

Level of recommendation: Strong
Quality of evidence: High

CARE OF THE PATIENT WITH AN AAA: AREAS IN NEED OF FURTHER RESEARCH

A number of areas of uncertainty exist in the care of patients with AAA that would benefit from further investigation. While the following list is not meant to be comprehensive, future research efforts should consider addressing the following topics:

- Improved strategies to identify patients at risk for postoperative MI or cardiovascular related death.
- Perioperative management recommendations for patients with preexistent pulmonary disease.
- Perioperative management recommendations for patients with preexistent diabetes.
- Perioperative management recommendations for patients with renal insufficiency.
- Recommendations to reduce the risk of contrast-induced nephropathy among patients with renal insufficiency undergoing EVAR.
- Optimal preoperative hydration regimen for patients with renal insufficiency undergoing OSR.
- Perioperative management recommendations for patients with preexistent anemia.
- The genetic and molecular basis of familial AAA.
- Biomarkers and single nucleotide genetic polymorphisms that identify patients at risk for development, progression, or rupture of an AAA.
- Applicability of estimates of AAA tensile stress and wall strength or other CT, MRI, or PET derived parameters to idemtify patients at risk for rapid growth or rupture.
- Screening for AAA in women and minorities.
- Optimal methods for invitation to AAA screening, ease of access to initial ultrasound and follow up, costs and workforce needs, and methods for providing risk-benefit information to individuals offered screening.
- Psychological effects of screening on patients and their partners.
- Effectiveness of screening programs initiated outside of initial screening centers.
- Frequency of imaging surveillance for specific AAA size groups (3 cm to 4.0 cm, 4.0 cm to 4.4 cm, 4.5 cm to 4.9 cm, and 5.0 cm to 5.5 cm).
- Management recommendations for EVAR versus surveillance and selective treatment for AAA <5.5 cm.
- Examination of the survival effect of immediate treatment versus surveillance and selective treatment for spe-

cific AAA size (4.0 cm to 4.4 cm, 4.5 cm to 4.9 cm, and 5.0 cm to 5.5 cm), age, gender, and fitness subgroups.
- Scales of fitness for surgical or endovascular intervention.
- Management recommendations for EVAR versus no intevention in high-risk patients unfit for OSR with an AAA ≥5.5 cm.
- Improvement in medical management of patients with large AAA considered unfit for OSR.
- Management recommendations for AAA in women and minorities.
- Therapeutic strategies directed at reduction in AAA growth rate or rupture risk, including clarification of the potential role of doxycycline, roxithromycin, and statin therapy in the progression of aneurysmal disease.
- Therapeutic strategies directed at regression of AAA size.
- Biomarkers and genetic polymorphisms that identify new avenues for pharmacotherapy.
- To identify whether OSR outcomes vary with respect to aneurysm features, gender, ethnicity, or socioeconomic status.
- Studies of hospital and physician volume-OSR outcome relationship.
- Simulation training in OSR.
- Cost effectiveness strategies for OSR that include considerations of time away from work for patients and family members and disease-specific quality of life instruments.
- Recommendations for staged OSR and renal angioplasty or simultaneous OSR and renal artery bypass.
- Long-term safety of endografts with suprarenal fixation.
- Improvements in branched EVAR devices to maintain pelvic perfusion.
- Effectiveness of EVAR for ruptured AAA.
- To identify whether EVAR outcomes vary with respect to endograft type or aneurysm features.
- Studies of hospital and physician volume-EVAR outcome relationship.
- Simulation training in EVAR.
- Cost effectiveness strategies for EVAR that include considerations of time away from work for patients and family members and disease-specific quality of life instruments.
- Recommendations for staged or simultaneous EVAR and renal angioplasty.
- Improvements in laparoscopic approaches for AAA repair.
- Simulation training in laparoscopic AAA repair.
- Recommendations for cell salvage and ultrafiltration devices during OSR.
- Recommendations for intraoperative blood product-based resuscitation during OSR.
- Recommendations for intraoperative fluid resuscitation during OSR.
- Impact of of forced air-warming blanket position (ie, lower extremities vs upper trunk) during OSR.
- Optimal use of ICU after OSR.
- Benefits of DVT prophylaxis and optimal prophylactic measures among patients undergoing OSR or EVAR.
- Improvements in EVAR devices and related techniques to reduce complications and long-term follow-up.
- Strategies to reduce hernia formation and small bowel obstruction after OSR.
- Infection-resistant aortic prostheses.
- Management strategies for Type II endoleaks.
- Durability of EVAR after additional interventions for treatment of Type I or III endoleak or device migration.
- Postoperative surveillance protocols, including optimal use of CDU, contrast enhanced CDU, and CT imaging at various time periods after OSR or EVAR (zero to five years, five to 10 years, 10 to 15 years).
- Effectiveness of pressure sensors in reduction of postoperative surveillance costs.

AUTHOR CONTRIBUTIONS

Conception and design: EC, DB, RD, KI, MM, GS, CT, GU, FV
Analysis and interpretation: EC, DB, RD, MM, KI, GS, CT, GU, FV
Data collection: EC, DB, RD, MM, GS, KI, CT, GU, FV
Critical revision of the article: EC, DB, RD, MM, KI, GS, CT, GU, FV
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