

SVS PRACTICE GUIDELINES

From the Society for Vascular Surgery

SVS practice guidelines for the care of patients with an abdominal aortic aneurysm: Executive summary

Elliot L. Chaikof, MD, PhD,^a David C. Brewster, MD,^b Ronald L. Dalman, MD,^c Michel S. Makaroun, MD,^d Karl A. Illig, MD,^e Gregorio A. Sicard, MD,^f Carlos H. Timaran, MD,^g Gilbert R. Upchurch Jr, MD,^h and Frank J. Veith, MD,ⁱ *Atlanta, Ga; Boston, Mass; Palo Alto, Calif; Pittsburgh, Pa; Rochester, NY; St. Louis, Mo; Dallas, Tex; Ann Arbor, Mich; and Cleveland, Ohio*

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, initially 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 article is an executive summary of the main practice guidelines document and 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.²

From the Department of Surgery, Emory University,^a the Department of Surgery, Massachusetts General Hospital,^b the Department of Surgery, Stanford University,^c the Department of Surgery, University of Pittsburgh,^d the Department of Surgery, University of Rochester,^e the Department of Surgery, Washington University-St. Louis,^f the Department of Surgery, University of Texas-Southwestern,^g the Department of Surgery, University of Michigan,^h and the Department of Vascular Surgery, Cleveland Clinic Foundation.ⁱ

Dr Chaikof received research (principal investigator) and educational support (program director) from WL Gore, and research and educational support (program director) from Medtronic and Cook, Inc. Dr Makaroun received research and educational support (program director) from WL Gore, Cook Inc, Medtronic, Cordis, BSCI, Abbott, Bolton, and Lombard. In the last two years, Dr. Makaroun had consulting agreements with WL Gore, Medtronic, Cook, Inc and Cardiometrics. Dr Illig received proctoring, travel expenses, and honoraria from Cook, Inc, speaking and travel expenses from Cook, Inc and WL Gore, and also University of Rochester Department of Vascular Surgery has received unrestricted educational grant support from Cook, Inc, WL Gore, Medtronic, and Boston Scientific. Dr Timaran received fees for consulting and speaking from W.L. Gore and Associates, Inc. Dr Veith is a stockholder in Vascular Innovation, a company that may in the future make aortic endografts. Nothing in this article was influenced by their ownership.

Reprint requests: Elliot L. Chaikof, MD, PhD, Emory University, 101 Woodruff Circle, Rm 5105, Atlanta, GA 30322 (e-mail: echaiko@emory.edu).

0741-5214/\$36.00

Copyright © 2009 Published by Elsevier Inc. on behalf of the Society for Vascular Surgery.

doi:10.1016/j.jvs.2009.07.001

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).³ 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 on in the

Table. 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 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.

estimate of effect; or **low** when further research is very likely to change the estimate of the effect.

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.⁴ An AAA is over seven times more likely to develop in a smoker than a non-smoker, with the duration of smoking, rather than total number of cigarettes smoked, being the key variable.⁵ Family members are also at significant risk with 12% to 19% of those undergoing aneurysm repair having a first-degree relative with an AAA.⁶ The risk for developing an AAA is lower in women, African Americans, and diabetic patients. Risk factors for rupture have also been identified, including female gender, large initial aneurysm diameter, low forced expiratory volume in one second (FEV1), current smoking history, and elevated mean blood pressure.

Physical examination

Only 30% to 40% of aneurysms are noted on physical examination, with detection dependant on aneurysm size and limited by truncal obesity. An abdominal aneurysm may be present in up to 85% of patients with a femoral artery aneurysm and in up to 60% of those with a popliteal aneurysm. In contrast, approximately 15% patients with an abdominal aneurysm have either a femoral or a popliteal artery aneurysm.

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. 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. A substantial portion of patients with AAA have underlying CAD and postoperative myocardial infarction (MI) carries with it a substantially increased risk of death, as well as a high risk for later cardiovascular events and death. Indeed, while elective open surgical repair (OSR) can generally be considered to carry a higher risk for a perioperative cardiovascular event than endovascular aortic aneurysm repair (EVAR), the latter is associated with intermediate to high cardiac risk in the range of 3% to 7%. Thus, it is critical to minimize the risk of cardiac morbidity during the course of OSR or EVAR for AAA.

In the absence of an active cardiac condition (unstable or severe angina, recent MI <1 month, decompensated heart failure, significant arrhythmia, or severe valvular heart disease), further non-invasive testing is only indicated if it will change management. 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. However, those patients who present with three or more cardiac risk factors (history of heart disease, congestive heart failure, cerebral vascular disease, diabetes, creatinine > 2 mg/dL) and have an unknown or low activity level (MET < 4) may benefit from stress testing.

Noninvasive stress testing should be considered for patients with a history of three or more clinical risk 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 EVAR or OSR, if it will change management.

Level of recommendation:	Strong
Quality of evidence:	Moderate

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. However, it bears emphasis that coronary revascularization is indicated for those patients who present 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.

Perioperative medical management of coronary artery disease. Perioperative heart rate control with beta blockade appears appropriate for patients with known cardiovascular disease or at least one clinical risk factor, but should be started days to weeks before elective surgery with a target heart rate of less than 65 beats per minute.⁷ Recent clinical data also supports the notion that statins, alpha-2 agonists for perioperative control of hypertension, and calcium channel blockers reduce perioperative cardiac morbidity and death.

Areas in Need of Further Research

- Improved strategies to identify patients at risk for postoperative MI or cardiovascular related death.
-

Pulmonary disease. Between 7% and 11% of patients with chronic obstructive pulmonary disease (COPD) have an aneurysm and failure to optimize COPD management is associated with increased morbidity and mortality. If COPD is severe, formal pulmonary consultation is recommended for prediction of short- and long-term prognosis and 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.

Areas in Need of Further Research

- Perioperative management recommendations for patients with preexistent pulmonary disease.
-

Renal impairment. Preoperative renal insufficiency is known to be a risk factor for a poor outcome after open aneurysm repair. To minimize deterioration in renal function among these patients, preoperative intravenous hydration is recommended and angiotensin-converting enzyme inhibitors and angiotensin receptor antagonists should be held the morning of surgery and restarted only after the patient is euvoletic.⁸ Recent meta-analyses have not identified a significant benefit from intraoperative use of mannitol alone,⁹ but have found beneficial effects from fenoldopam, particularly when administered at ~0.1 µg/kg/min beginning at the initiation of surgery.¹⁰ Likewise, patients at increased risk for contrast-induced nephropathy (CIN) should be hydrated both prior (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) receipt of a contrast dye load as in EVAR. While fenoldopam, dopamine, theophylline, or calcium channel blockers do not appear to be beneficial in preventing CIN, N-acetylcysteine and ascorbic acid may be of benefit.¹¹ The incidence of CIN appears to be lower with iodixanol (Visipaque) and iopamidol (Isovue-370) than with iohexol (Omnipaque). Use of CO₂ gas as an alternate imaging agent may be considered. 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 euvoletic.

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

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

Areas in Need of Further Research

- Perioperative management recommendations for patients with renal insufficiency.
 - Optimal preoperative hydration regimen for patients with renal insufficiency undergoing OSR.
 - Recommendations to reduce the risk of contrast-induced nephropathy among patients with renal insufficiency undergoing EVAR.
-

Hematologic disorders. A number of studies have documented that even in the elective setting, anemia or a low hemoglobin level is associated with increased mortality following OSR. Ho and colleagues¹² 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. Therefore, we recommend perioperative blood transfusion if the preoperative hematocrit is less than 28%. Ho et al have also documented that a platelet count of 130,000 platelets/µL or less was associated with increased risk of bleeding among patients undergoing OSR,¹² and Matsumura and colleagues noted that a lower preoperative platelet count was an independent predictor of two-year mortality among patients under-

going OSR and EVAR ($P = .012$).¹³ Thus, further hematologic assessment is recommended if the preoperative platelet count is less than 130,000 platelets/ μ L.

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/ μ L.

Level of recommendation: Weak
Quality of evidence: Low

Areas in Need of Further Research

- Perioperative management recommendations for patients with preexistent anemia.

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. Isolated AAA, unrelated to a prior aortic dissection, is uncommonly associated with Marfan syndrome. Population screening for single nucleotide polymorphisms (SNPs) to identify patients at risk for AAA have identified a number of genetic variants proposed to be associated with AAA, but 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.¹⁴

Areas in Need of Further Research

- 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.

Aneurysm imaging

Image derived criteria to predict risk of AAA rupture. Maximum AAA diameter remains the most widespread criterion to predict risk of AAA rupture, but a variety of alternate parameters have been proposed as more sensitive predictors of rupture risk including AAA expansion rate, increase in intraluminal thrombus thickness, wall stiffness, wall tension, and peak AAA wall stress.¹⁵ 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. Further validation of these tools, however, will be required before they can be applied with confidence in clinical practice.

Areas in Need of Further Research

- Applicability of estimates of AAA tensile stress and wall strength or other computed tomography (CT), magnetic resonance imaging (MRI), or positron emission tomography (PET) derived parameters to identify patients at risk for rapid AAA growth or rupture.

Recommendations for aneurysm screening and surveillance. 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.¹⁶⁻²¹ Thus, 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 also be performed for women at or older than age 65 who have smoked or have a family history. If an AAA is identified, we recommend follow-up imaging at 12-month intervals for patients with an AAA of 3.5 cm to 4.4 cm in diameter and at six-month intervals for patients with an AAA diameter between 4.5 cm and 5.4 cm. For otherwise healthy patients, imaging is recommended at three-year intervals for those between 3.0 cm and 3.4 cm in diameter and at five-year intervals if the aortic diameter measures between 2.6 cm and 2.9 cm. It bears noting that these recommendations are based upon maximum external aortic diameter.

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

Surveillance imaging at 12-month intervals is recommended for patients with an AAA of 3.5 cm 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 cm 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 cm 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 cm and 2.9 cm.

Level of recommendation: Weak
Quality of evidence: Low

Areas in Need of Future Research

- 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).
-

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. 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 saccular 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. Long-term survival was equivalent in the United Kingdom Small Aneurysm Trial (UKSAT)²² and the Aneurysm Detection and Management (ADAM) Trial²³ for both immediate surgery and surveillance groups. Nonetheless, a trend towards a beneficial effect of early surgery was observed in both studies in the younger patient and for those with larger aneurysms. 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)²⁴ and Positive impact of endovascular options for treating aneurysm early (PIVOTAL) trials compare immediate EVAR with surveillance and selective EVAR, but neither trial has been 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. At present, surveillance with selective repair is most appropriate for older male patients with significant co-morbidities. 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 should be considered 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

Areas in Need of Further Research

- 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 specific 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 AAA in women and minorities.
-

Medical management during the period of AAA surveillance

During the surveillance period, patients should be counseled to cease smoking if tobacco products are being utilized and encouraged to seek appropriate management for hypertension, hyperlipidemia, diabetes, and other atherosclerotic risk factors. A statin and angiotensin-converting enzyme (ACE) inhibitor should be initiated given their broad potential benefits and acceptable safety profile. Insufficient data exists to recommend use of doxycycline or roxithromycin. Likewise, 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.^{25,26} Patients should be counseled that moderate physi-

cal activity does not precipitate rupture and may limit AAA growth rate. 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

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

Areas in Need of Further Research

- 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.
-

Open surgery

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 to be suitable for EVAR undergo standard OSR. It is clear, therefore, that a vascular surgeon should be familiar and experienced with both an anterior transperitoneal (TP) and left-flank retroperitoneal (RP) approaches, utilizing each as determined by patient anatomy and clinical needs. Although advocates of a RP approach claim various physiologic benefits, including reductions in fluid losses, cardiac stress, postoperative pulmonary complications, and severity of ileus, randomized prospective studies have generated conflict-

ing results.^{27,28} A RP approach is generally preferable for patients with a “hostile abdomen” secondary to multiple prior intra-abdominal operations, a history of irradiation, or stoma, or for repair of inflammatory aneurysms or AAA associated with a horseshoe kidney. 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.

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

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

Level of recommendation: Strong
Quality of evidence: High

Aortic clamping. 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. 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. Although suprarenal clamping is associated with an increased risk of postoperative decrease in renal function and overall adverse events, overall 30-day mortality is comparable to those patients repaired with infrarenal crossclamping.

It is usually recommended that the proximal clamp be applied first, in order to minimize the occurrence of atheromatous embolization. Irrespective of clamp location and method, however, 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, with surgeon preference and cost the dominant determinants in aortic graft choice. 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. Certainly bifurcated grafts are advisable if clinically significant concomitant iliac aneurysms (>2.0 cm to 2.4 cm) are present, which may be present in 20% to 30% of patients with infrarenal AAA.²⁹ If coexistent symptomatic aortoiliac occlusive disease exists with limiting claudication, both aneurysmal and obliterative disease can be corrected with an aortobifemoral graft.

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. Although multifactorial in origin, ligation of a patent inferior mesenteric artery (IMA) is the most commonly noted risk factor for development of colon ischemia in many series. 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, but its value has not been clearly established. 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.³⁰ It seems reasonable to conclude that IMA reimplantation should be considered in the presence of associated celiac or superior mesenteric artery (SMA) occlusive disease, an enlarged meandering mesenteric artery, a history of prior colon resection, inability to preserve hypogastric perfusion, substantial blood loss or intraoperative hypotension, poor IMA backbleeding when graft open, poor Doppler flow in colonic vessels, or should the colon appear ischemic.

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 lead to erectile dysfunction, symptomatic hip and buttock claudication, or occasionally colon ischemia, buttock necrosis, or spinal cord (cauda equina) ischemia.²⁹ Recently, the necessity to preserve perfusion of a least one hypogastric artery has been questioned as EVAR has become more widely utilized. Although 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;³¹ buttock claudication was noted in 42% and new onset of erectile dysfunction in 14%. Firm conclusions on this topic cannot be reached with certainty at present, however, it seems prudent to make every effort to preserve hypogastric perfusion on at least one side.

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

Timing of surgery. 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. The reason for such differences in outcome are multifactorial, but include the fact that emergent 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. Thus, each clinical situation must be approached individually and, in selected circumstances, it may be prudent to delay 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 intensive care unit (ICU) setting prior to operation.

A related practical concern regarding timing of operation, which frequently arises but for which there is little data relates to scheduling of elective repair in asymptomatic patients with large AAA. There is certainly no standard of care in regard to the time period within which elective repair must be carried out. While there is little advantage in delay, it is appropriate to obtain pertinent preoperative studies in a timely fashion, particularly in older, high-risk individuals. On occasion, however, rupture may occur during this interval. Although completely unpredictable, it is recommended that risks and benefits of a planned course of action be clearly communicated to the patient and family.

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

Perioperative outcomes of open AAA repair. 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%.³²⁻³⁴ 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 experience.³⁵ Complications after open AAA repair are observed in 15% to 30% of

patients. Multiple reports have identified a strong relationship between outcomes following AAA repair and both hospital and individual surgeon case volume and experience.³⁶ We recommend that OSR be 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

Areas in Need of Further Research

- 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.
-

Endovascular repair

Endovascular aortic aneurysm repair (EVAR) is progressively replacing OSR for the treatment of infrarenal AAA and 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.³⁷

Suprarenal and infrarenal fixation. Pooled results from EVAR devices support the safety and efficacy for endografts with infrarenal fixation. Likewise, several observational studies have reported the efficacy and safety of suprarenal endograft fixation. Concerns have been raised regarding the short- and long-term risks of renal or mesenteric artery embolization and occlusion after EVAR with suprarenal endograft fixation. However, 50% and 87% of endografts used in the Dutch Randomised Endovascular Aneurysm Management (DREAM) and Endovascular aneurysm repair (EVAR) trials, respectively, were performed with endografts that used suprarenal fixation and onrates of renal dysfunction appear to be no different when compared with those patients treated with devices that rely upon infrarenal fixation.

Areas in Need of Further Research

- Long-term safety of endografts with suprarenal fixation.
-

Recommended management of the internal iliac artery. Several studies have revealed that unilateral embolization of the hypogastric artery (HA) can be performed

during EVAR with minimal adverse events. Moreover, concomitant unilateral HA embolization during EVAR has been shown to be safe and effective, as compared with staged procedures. 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.³⁸ Patients should be aware that in at least one large series, buttock claudication persisted in 12% of unilateral and 11% of bilateral hypogastric artery interruptions, whereas impotence occurred in 9% of unilateral and 13% of bilateral HA occlusions.³⁹

Bilateral hypogastric artery occlusion with endograft extension into both external iliac arteries is occasionally required in patients at high-risk for OSR. Initial concerns about life-threatening pelvic or colonic ischemia and neurologic deficits may have been overestimated as several recent reports suggest that such devastating complications rarely occur. Technical considerations that may reduce the incidence of adverse events when bilateral HA embolization is required, include staging bilateral HA embolization, 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.

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

Areas in Need of Further Research

- Improvements in branched EVAR devices to maintain pelvic perfusion.
-

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. 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.⁴⁰ In this regard, 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 three-month mortality (40% and 42%).⁴¹ Identical mortality rates (53%) were also reported in a study of 32 patients randomized to EVAR or OSR.⁴² Nonetheless, recent studies analyzing national trends in the United States have observed that EVAR is being used with increasing frequency in the emergency management of ruptured AAA, with decreasing mortality. Results in non-teaching centers and low volume institu-

tions, however, were substantially worse than those in teaching hospitals and high volume centers. Establishing a protocol for urgent or emergent EVAR for ruptured AAAs appears to be essential to obtain optimal results. In particular, “hypotensive hemostasis,” which refers to restricting aggressive fluid resuscitation as long as the patient remains conscious and systolic blood pressure exceeds 50 mm Hg to 70 mm Hg, appears to be beneficial. In hemodynamically unstable patients that do not have a preoperative CT scan, intraoperative angiography and intravascular ultrasound may assist in device selection. Abdominal compartment syndrome may occur after EVAR for ruptured AAAs among hemodynamically unstable patients with a large retroperitoneal hematoma and diffuse visceral edema. Early recognition through measurement of bladder pressure and surgical decompression are necessary to improve survival.

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

Level of recommendation:	Strong
Quality of evidence:	Moderate

Areas in Need of Further Research

- Effectiveness of EVAR for ruptured AAA.
-

Role of EVAR in high-risk and unfit patients for open repair. 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.⁴³ Although a number of preoperative risk prediction methods have been reported, 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

Areas in Need of Further Research

- Management recommendations for EVAR versus no intervention in high-risk patients unfit for OSR with an AAA \geq 5.5 cm.
 - Improvement in medical management of patients with large AAA considered unfit for OSR.
-

Perioperative outcomes of elective EVAR. 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; Medtronic, Inc, Minneapolis, Minn [n = 416] 1.7%;⁴⁴ Excluder; W.L. Gore, Inc, Flagstaff, Ariz [n = 235] 1.3%;⁴⁵ Zenith; Cook Medical, Inc, Bloomington, Ind [n = 352] 1.1%;^{46,47} Powerlink; Endologix, Inc, Irvine, Calif [n = 192] 1%⁴⁸). However, pooled trial data representing the OSR cohorts was associated with a comparable 30-day mortality rate of 1.4%. 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, $P = .32$).⁴⁹ 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.^{50,51} These differences, however, did not achieve statistical significance ($P = .1$). It is recommended that elective EVAR be 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%.

It is noteworthy that with rapid adoption of this technology in the United States, much lower mortality rates have been reported for EVAR than OSR in analyses of large statewide and multi-state population-based databases. In a recent analysis of 45,000 propensity-score-matched Medicare beneficiaries, mortality was significantly lower after EVAR (1.2% vs 4.8%; $P < .001$), with reduction in mortality most pronounced for those of advanced age (80-84 years: 1.6% vs 7.2%; ≥ 85 years: 2.7% vs 11.2%; $P < .001$).⁵² Likewise, major medical complications are lower after EVAR than OSR. It is possible, however, that differences in outcome reflect the inclusion of anatomically more complex aneurysms among the cohort treated by OSR. It is noteworthy that disparities in EVAR outcome have been identified between patients of varying ethnicity and insurance type.

Despite the reduction in mortality and medical morbidity that appears associated with EVAR, 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%).⁵¹ Similar findings have been reported in observational studies with local or vascular complications occurring in 9% to 16% of patients after EVAR. 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.⁵⁰

The DREAM and EVAR-1 trials have provided mid-term follow-up data at two and four years, respectively.^{53,54} The initial reduction in all-cause mortality observed after EVAR 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. A population-based study of 45,660 Medicare beneficiaries undergoing either EVAR or OSR demonstrates similar findings.⁵²

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, aneurysm features, gender, ethnicity, and socioeconomic status; 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

Areas in Need of Further Research

- To identify whether EVAR outcomes vary with respect to endograft type, aneurysm features, gender, ethnicity, or socioeconomic status.
 - 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.
-

ANESTHETIC CONSIDERATIONS AND PERIOPERATIVE MANAGEMENT

Intraoperative fluid resuscitation and blood conservation

Intraoperative blood salvage during OSR can be achieved using either red blood cells (RBC) processors or hemofiltration devices. However, cell salvage techniques during vascular surgery have not prevented the need for transfusion and have not proven cost-effective. Routine use of cell salvage and ultrafiltration devices cannot be recommended, but is recommended if large blood loss is anticipated.

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. Likewise, optimal blood replacement therapy during complex OSR has not been defined and research in this area is encouraged.

Intravenous 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. 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.

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

Areas in Need of Further Research

- 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.
 - Optimal use of ICU after OSR.
-

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. However, patients at increased risk of a cardiac event following EVAR or OSR should be considered for electrocardiogram (ECG) monitoring and measurement of postoperative troponin levels, since troponin elevation is predictive of adverse short- and long-term outcomes.^{55,56} Otherwise, troponin measurement is only recommended for patients with postoperative ECG changes, chest pain, or other signs of cardiovascular dysfunction. Overall, improved strategies to identify postoperative patients at risk for MI are needed, and this is an area recommended for further study.

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

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. However, DVT after open AAA repair appears to be underappreciated. A Cochrane analysis of all non-randomised prospective studies in aortic surgery identified an average incidence of 9.2% among patients without DVT prophylaxis.⁵⁷ 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.⁵⁸ Consistent with these findings, deMaistre et al⁵⁹ 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. Indeed, aortic surgery may be associated with a higher risk of DVT than infrainguinal bypass.

Overall, most patients undergoing EVAR or OSR can be considered at moderate to high risk for DVT, given advanced age, duration of surgery >45 minutes, 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/pulmonary embolism [PE], obesity [BMI >25], limited mobility status, malignancy, hypercoagulable 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.

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

Areas in Need of Further Research

- Benefits of DVT prophylaxis and optimal prophylactic measures among patients undergoing OSR or EVAR.
-

POSTOPERATIVE AND LONG-TERM MANAGEMENT

Late outcomes after open surgery and EVAR

Both EVAR and OSR are associated with late complications. 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.

Areas in Need of Further Research

- Improvements in EVAR devices and related techniques to reduce complications and long-term follow-up.
-

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. 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, which appears to be substantially more common after treatment of AAA than aortic occlusive disease.

Areas in Need of Further Research

- Strategies to reduce hernia formation and small bowel obstruction after OSR.
-

Para-anastomotic aneurysm. 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.⁶⁰ 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. 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 is a welcome approach to this difficult problem.

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. Graft infection, however, is the indication for intervention in up to 25% of redo aortic surgery. The risk of graft infection after EVAR appears to be similar to that of OSR. Primary aortic graft infection usually presents late, on the average three years after implantation and on occasion much later. Femoral extension of the abdominal grafts increases the incidence of graft infection from 1% to nearly 3%. Presentations can be quite diverse including generalized sepsis, groin purulence and drainage, pseudoaneurysm formation, or ill defined pain. 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. Reiley 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.⁶¹ 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.

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

Areas in Need of Further Research

- Infection-resistant aortic prostheses.
-

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. 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. Endografts are at a higher risk for limb thrombosis than prostheses placed during the course of OSR, as observed in the EVAR-1 trial.⁵³ Endograft limbs, including stented limbs, can be narrowed by a calcified small diameter aortic bifurcation or tortuous, angulated and diseased iliac arteries.

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 may be less 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 ankle-brachial index (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. It is one of the most common abnormalities identified on late imaging and used to justify lifelong follow-up of these patients. Four types of endoleak have been described, independent of graft type:

Type I endoleak occurs in the absence or loss of complete sealing at the proximal (Type 1A) or distal (Type 1B) 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. 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.

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. For those detected at the time of EVAR, further treatment is not indicated, since spontaneous resolution is possible. 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. 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. 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.

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. Translumbar direct puncture of the aneurysm sac can also be utilized successfully. 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.

Type III endoleaks arise from poorly seated modular connections, disconnection and separation of components, or, infrequently, fabric erosion related to material fatigue. All Type III endoleaks should be treated.

Type IV endoleaks represents self-limiting blood seepage through the graft material due to porosity and treatment is not required.

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

Areas in Need of Further Research

- Management strategies for Type II endoleaks.
 - Durability of EVAR after additional interventions for treatment of Type I or III endoleak or device migration.
-

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. Additionally, a serous ultrafiltrate across a microporous fabric can fill the aneurysm and increase pressure. 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. 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 postoperative device migration appears related to the duration of follow-up. Most series evaluating device migration have reported increases after 24 months.

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. Thus, we recommend follow-up CT imaging at five year intervals after OSR.

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. 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.⁶² 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. Recent studies, however, have suggested that the lower sensitivity of color Doppler ultrasound (CDU) as compared to CT imaging is offset by a high degree of correlation between CDU and CT imaging in detection of clinically significant endoleaks.⁶³ Moreover, recent small studies evaluating the role of contrast-enhanced ultrasound in the detection of endoleaks report increased sensitivity, negative predictive value, accuracy, and specificity when compared with CDU. 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.⁶⁴ A significant increase in aneurysm size or new onset of endoleak, if detected by CDU at later follow-up, would prompt CT imaging. Eliminating the traditional six-month CT scan has also been recommended, if CT imaging one month after EVAR does not identify an endoleak.^{64,65} 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. 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. 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.

The Acute pressure measurement to confirm aneurysm exclusion (APEX) trial confirmed that intrasac pressure and pressure changes could be reliably measured non-invasively by a pressure sensor.⁶⁶ 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 the 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

Areas in Need for Further Research

- Postoperative surveillance protocols, including optimal use of CDU, contrast enhanced CDU, and CT imaging at various time periods after OSR or EVAR (0-5, 5-10, 10-15 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, KI, GS, CT, GU, FV

Writing the article: EC, DB, RD, MM, KI, GS, CT, GU, FV

Critical revision of the article: EC, DB, RD, MM, KI, GS, CT, GU, FV

Final approval of the article: EC, DB, RD, MM, KI, GS, CT, GU, FV

Statistical analysis: Not applicable

Obtained funding: Not applicable

Overall responsibility: EC

REFERENCES

1. Brewster DC, Cronenwett JL, Hallett JW Jr, Johnston KW, Krupski WC, Matsumura JS. Guidelines for the treatment of abdominal aortic aneurysms. Report of a subcommittee of the Joint Council of the American Association for Vascular Surgery and Society for Vascular Surgery. *J Vasc Surg* 2003;37:1106-17.
2. Chaikof EL, Brewster DC, Dalman RL, Makaroun MS, Illig KA, Sicard GA, et al. The care of patients with an abdominal aortic aneurysm: The Society for Vascular Surgery practice guidelines. *J Vasc Surg* 2009; 50(Suppl 8S)2S-49S.

3. 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.
4. Lederle FA, Johnson GR, Wilson SE, Chute EP, Hye RJ, Makaroun MS, et al. The aneurysm detection and management study screening program: validation cohort and final results. *Aneurysm Detection and Management Veterans Affairs Cooperative Study Investigators. Arch Intern Med* 2000;160:1425-30.
5. Wilmink TB, Quick CR, Day NE. The association between cigarette smoking and abdominal aortic aneurysms. *J Vasc Surg* 1999;30:1099-105.
6. van Vlijmen-van Keulen CJ, Pals F, Rauwerda JA. Familial abdominal aortic aneurysm: A systematic review of a genetic background. *Eur J Vasc Endovasc Surg* 2002;24:105-16.
7. Feringa HH, Bax JJ, Boersma E, Kertai MD, Meij SH, Galal W, et al. High-dose beta-blockers and tight heart rate control reduce myocardial ischemia and troponin T release in vascular surgery patients. *Circulation* 2006;114:1344-9.
8. Bertrand M, Godet G, Meerschaert K, Brun L, Salcedo E, Coriat P. Should the angiotensin II antagonists be discontinued before surgery? *Anesth Analg* 2001;92:26-30.
9. Hersey P, Poullis M. Does the administration of mannitol prevent renal failure in open abdominal aortic aneurysm surgery? *Interact Cardiovasc Thorac Surg* 2008;7:906-9.
10. Landoni G, Biondi-Zoccai GG, Tumlin JA, Bove T, De Luca M, Calabro MG, et al. Beneficial impact of fenoldopam in critically ill patients with or at risk for acute renal failure: A meta-analysis of randomized clinical trials. *Am J Kidney Dis* 2007;49:56-68.
11. Pannu N, Wiebe N, Tonelli M. Prophylaxis strategies for contrast-induced nephropathy. *JAMA* 2006;295:2765-79.
12. Ho P, Ting AC, Cheng SW. Blood loss and transfusion in elective abdominal aortic aneurysm surgery. *ANZ J Surg* 2004;74:631-4.
13. Matsumura JS, Katzen BT, Sullivan TM, Dake MD, Naftel DC. Predictors of survival following open and endovascular repair of abdominal aortic aneurysms. *Ann Vasc Surg* 2009;23:153-8.
14. Helgadottir A, Thorleifsson G, Magnusson KP, Gretarsdottir S, Steinthorsdottir V, Manolescu A, et al. The same sequence variant on 9p21 associates with myocardial infarction, abdominal aortic aneurysm and intracranial aneurysm. *Nat Genet* 2008;40:217-24.
15. Fillinger MF, Marra SP, Raghavan ML, Kennedy FE. Prediction of rupture risk in abdominal aortic aneurysm during observation: wall stress versus diameter. *J Vasc Surg* 2003;37:724-32.
16. Scott RA, Bridgewater SG, Ashton HA. Randomized clinical trial of screening for abdominal aortic aneurysm in women. *Br J Surg* 2002;89:283-5.
17. Lindholt JS, Juul S, Fasting H, Henneberg EW. Hospital costs and benefits of screening for abdominal aortic aneurysms. Results from a randomised population screening trial. *Eur J Vasc Endovasc Surg* 2002;23:55-60.
18. Ashton HA, Buxton MJ, Day NE, Kim LG, Marteau TM, Scott RA, et al. The Multicentre Aneurysm Screening Study (MASS) into the effect of abdominal aortic aneurysm screening on mortality in men: A randomised controlled trial. *Lancet* 2002;360:1531-9.
19. Scott RA, Wilson NM, Ashton HA, Kay DN. Influence of screening on the incidence of ruptured abdominal aortic aneurysm: 5-year results of a randomized controlled study. *Br J Surg* 1995;82:1066-70.
20. Vardulaki KA, Walker NM, Couto E, Day NE, Thompson SG, Ashton HA, Scott RA. Late results concerning feasibility and compliance from a randomized trial of ultrasonographic screening for abdominal aortic aneurysm. *Br J Surg* 2002;89:861-4.
21. Norman PE, Jamrozik K, Lawrence-Brown MM, Le MT, Spencer CA, Tuohy RJ, et al. Population based randomised controlled trial on impact of screening on mortality from abdominal aortic aneurysm. *BMJ* 2004;329:1259.
22. United Kingdom Small Aneurysm Trial Participants. Long-term outcomes of immediate repair compared with surveillance of small abdominal aortic aneurysms. *N Engl J Med* 2002;346:1445-52.
23. Lederle FA, Wilson SE, Johnson GR, Reinke DB, Littooy FN, Acher CW, et al. Immediate repair compared with surveillance of small abdominal aortic aneurysms. *N Engl J Med* 2002;346:1437-44.
24. Cao P. Comparison of surveillance vs Aortic Endografting for Small Aneurysm Repair (CAESAR) trial: study design and progress. *Eur J Vasc Endovasc Surg* 2005;30:245-51.
25. Propranolol Aneurysm Trial Investigators. Propranolol for small abdominal aortic aneurysms: Results of a randomized trial. *J Vasc Surg* 2002;35:72-9.
26. Lindholt JS, Henneberg EW, Juul S, Fasting H. Impaired results of a randomised double blinded clinical trial of propranolol versus placebo on the expansion rate of small abdominal aortic aneurysms. *Int Angiol* 1999;18:52-7.
27. Cambria RP, Brewster DC, Abbott WM, Freehan M, Megerman J, LaMuraglia G, et al. Transperitoneal versus retroperitoneal approach for aortic reconstruction: a randomized prospective study. *J Vasc Surg* 1990;11:314-24.
28. Sicard GA, Reilly JM, Rubin BG, Thompson RW, Allen BT, Flye MW, et al. Transabdominal versus retroperitoneal incision for abdominal aortic surgery: report of a prospective randomized trial. *J Vasc Surg* 1995;21:174-81.
29. Johnston KW, Scobie TK. Multicenter prospective study of nonruptured abdominal aortic aneurysms. I. Population and operative management. *J Vasc Surg* 1988;7:69-81.
30. Senekowitsch C, Assadian A, Assadian O, Hartleb H, Ptakovsky H, Hagmuller GW. Replanting the inferior mesentery artery during infrarenal aortic aneurysm repair: influence on postoperative colon ischemia. *J Vasc Surg* 2006;43:689-94.
31. Mehta M, Veith FJ, Darling RC, Roddy SP, Ohki T, Lipsitz EC, et al. Effects of bilateral hypogastric artery interruption during endovascular and open aortoiliac aneurysm repair. *J Vasc Surg* 2004;40:698-702.
32. Conrad MF, Crawford RS, Pedraza JD, Brewster DC, Lamuraglia GM, Corey M, et al. Long-term durability of open abdominal aortic aneurysm repair. *J Vasc Surg* 2007;46:669-75.
33. Hertzner NR, Mascha EJ, Karafa MT, O'Hara PJ, Krajewski LP, Beven EG. Open infrarenal abdominal aortic aneurysm repair: The Cleveland Clinic experience from 1989 to 1998. *J Vasc Surg* 2002;35:1145-54.
34. Berry AJ, Smith RB 3rd, Weintraub WS, Chaikof EL, Dodson TF, Lumsden AB, et al. Age versus comorbidities as risk factors for complications after elective abdominal aortic reconstructive surgery. *J Vasc Surg* 2001;33:345-52.
35. Lederle FA, Kane RL, MacDonald R, Wilt TJ. Systematic review: repair of unruptured abdominal aortic aneurysm. *Ann Intern Med* 2007;146:735-41.
36. Birkmeyer JD, Siewers AE, Finlayson EV, Stukel TA, Lucas FL, Batista I, et al. Hospital volume and surgical mortality in the United States. *N Engl J Med* 2002;346:1128-37.
37. Giles KA, Pomposelli F, Hamdan A, Wyers M, Jhaveri A, Schermerhorn ML. Decrease in total aneurysm-related deaths in the era of endovascular aneurysm repair. *J Vasc Surg* 2009;49:543-50.
38. Farahmand P, Becquemin JP, Desgranges P, Allaire E, Marzelle J, Roudot-Thoraval F. Is hypogastric artery embolization during endovascular aortoiliac aneurysm repair (EVAR) innocuous and useful? *Eur J Vasc Endovasc Surg* 2008;35:429-35.
39. Mehta M, Veith FJ, Ohki T, Cynamon J, Goldstein K, Suggs WD, et al. Unilateral and bilateral hypogastric artery interruption during aortoiliac aneurysm repair in 154 patients: a relatively innocuous procedure. *J Vasc Surg* 2001;33:S27-32.
40. Mastracci TM, Garrido-Olivares L, Cina CS, Clase CM. Endovascular repair of ruptured abdominal aortic aneurysms: a systematic review and meta-analysis. *J Vasc Surg* 2008;47:214-21.
41. Peppelenbosch N, Geelkerken RH, Soong C, Cao P, Steinmetz OK, Teijink JAW, et al. Endograft treatment of ruptured abdominal aortic aneurysms using the Talent aortouniiliac system: an international multicenter study. *J Vasc Surg* 2006;43:1111-23; discussion 1123.
42. Hinchliffe RJ, Bruijstens L, MacSweeney STR, Braithwaite BD. A randomised trial of endovascular and open surgery for ruptured abdominal aortic aneurysm - results of a pilot study and lessons learned for future studies. *Eur J Vasc Endovasc Surg* 2006;32:506-13.
43. EVAR trial participants. Endovascular aneurysm repair and outcome in patients unfit for open repair of abdominal aortic aneurysm (EVAR trial 2): Randomised controlled trial. *Lancet* 2005;365:2187-92.

44. Wilt TJ, Lederle FA, MacDonald R, Jonk YC, Rector TS, Kane RL. Comparison of endovascular and open surgical repairs for abdominal aortic aneurysm. Evidence report. Rockville, MD: AHRQ Publication No. 06-E017, 2006.
45. Matsumura JS, Brewster DC, Makaroun MS, Naftel DC. A multicenter controlled clinical trial of open versus endovascular treatment of abdominal aortic aneurysm. *J Vasc Surg* 2003;37:262-71.
46. Greenberg RK, Chuter TA, Sternbergh WC 3rd, Fearnot NE. Zenith AAA endovascular graft: intermediate-term results of the US multicenter trial. *J Vasc Surg* 2004;39:1209-18.
47. Greenberg RK, Chuter TA, Cambria RP, Sternbergh WC 3rd, Fearnot NE. Zenith abdominal aortic aneurysm endovascular graft. *J Vasc Surg* 2008;48:1-9.
48. Carpenter JP. The Powerlink bifurcated system for endovascular aortic aneurysm repair: four-year results of the US multicenter trial. *J Cardiovasc Surg (Torino)* 2006;47:239-43.
49. Sicard GA, Zwolak RM, Sidawy AN, White RA, Siami FS. Endovascular abdominal aortic aneurysm repair: Long-term outcome measures in patients at high-risk for open surgery. *J Vasc Surg* 2006;44:229-36.
50. Greenhalgh RM, Brown LC, Kwong GP, Powell JT, Thompson SG. Comparison of endovascular aneurysm repair with open repair in patients with abdominal aortic aneurysm (EVAR trial 1), 30-day operative mortality results: randomised controlled trial. *Lancet* 2004;364:843-8.
51. Prinszen M, Verhoeven EL, Buth J, Cuypers PW, van Sambeek MR, Balm R, et al. A randomized trial comparing conventional and endovascular repair of abdominal aortic aneurysms. *N Engl J Med* 2004;351:1607-18.
52. Schermerhorn ML, O'Malley AJ, Jhaveri A, Cotterill P, Pomposelli F, Landon BE. Endovascular vs. open repair of abdominal aortic aneurysms in the medicare population. *N Engl J Med* 2008;358:464-74.
53. EVAR trial participants. Endovascular aneurysm repair versus open repair in patients with abdominal aortic aneurysm (EVAR trial 1): Randomised controlled trial. *Lancet* 2005;365:2179-86.
54. Blankensteijn JD, de Jong SE, Prinszen M, van der Ham AC, Buth J, van Sterkenburg SM, et al. Two-year outcomes after conventional or endovascular repair of abdominal aortic aneurysms. *N Engl J Med* 2005;352:2398-2405.
55. Ali ZA, Callaghan CJ, Ali AA, Sheikh AY, Akhtar A, Pavlovic A, et al. Perioperative myocardial injury after elective open abdominal aortic aneurysm repair predicts outcome. *Eur J Vasc Endovasc Surg* 2008;35:413-9.
56. Kertai MD, Boersma E, Klein J, Van Urk H, Bax JJ, Poldermans D. Long-term prognostic value of asymptomatic cardiac troponin T elevations in patients after major vascular surgery. *Eur J Vasc Endovasc Surg* 2004;28:59-66.
57. Bani-Hani M, Al-Khaffaf H, Titi MA, Jaradat I. Interventions for preventing venous thromboembolism following abdominal aortic surgery. *Cochrane Database Syst Rev* 2008; CD005509.
58. Eagleton MJ, Grigoryants V, Peterson DA, Williams DM, Henke PK, Wakefield TW, et al. Endovascular treatment of abdominal aortic aneurysm is associated with a low incidence of deep venous thrombosis. *J Vasc Surg* 2002;36:912-6.
59. de Maistre E, Terriat B, Lesne-Padieu AS, Abello N, Bouchot O, Steinmetz EF. High incidence of venous thrombosis after surgery for abdominal aortic aneurysm. *J Vasc Surg* 2009;49:596-601.
60. Mii S, Mori A, Sakata H, Kawazoe N. Para-anastomotic aneurysms: incidence, risk factors, treatment and prognosis. *J Cardiovasc Surg (Torino)* 1998;39:259-66.
61. Reiley LM, Stoney RJ, Goldstone J, Ehrenfeld WK. Improved management of aortic graft infection: the influence of operation sequence and staging. *J Vasc Surg* 1987;5:421-31.
62. Brenner DJ, Hall EJ. Computed tomography - an increasing source of radiation exposure. *N Engl J Med* 2007;357:2277-84.
63. Manning BJ, O'Neill SM, Haider SN, Colgan MP, Madhavan P, Moore DJ. Duplex ultrasound in aneurysm surveillance following endovascular aneurysm repair: a comparison with computed tomography aortography. *J Vasc Surg* 2009;49:60-5.
64. Sternbergh WC 3rd, Greenberg RK, Chuter TA, Tonnessen BH. Redefining postoperative surveillance after endovascular aneurysm repair: recommendations based on 5-year follow-up in the US Zenith multicenter trial. *J Vasc Surg* 2008;48:278-84.
65. Go MR, Barbato JE, Rhee RY, Makaroun MS. What is the clinical utility of a 6-month computed tomography in the follow-up of endovascular aneurysm repair patients? *J Vasc Surg* 2008;47:1181-6.
66. Ohki T, Ouriel K, Silveira PG, Katzen B, White R, Criado F, Diethrich E. Initial results of wireless pressure sensing for endovascular aneurysm repair: The APEX Trial - Acute Pressure Measurement to Confirm Aneurysm Sac EXclusion. *J Vasc Surg* 2007;45:236-42.

Submitted Jul 2, 2009; accepted Jul 2, 2009.