The Care of Patients with an Abdominal Aortic Aneurysm: The Society for Vascular Surgery Practice Guidelines

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Special considerations
SUMMARY OF GUIDELINES FOR THE CARE OF PATIENTS WITH AN ABDOMINAL AORTIC ANEURYSM

Physical examination

*In patients with a suspected or known abdominal aortic aneurysm, we recommend performing physical examination that includes an assessment of femoral and popliteal arteries.*

*In patients with a popliteal or femoral artery aneurysm, we recommend evaluation for an abdominal aortic aneurysm.*

Level of recommendation: Strong
Quality of evidence: High

Assessment of medical comorbidities

*In patients with active cardiac conditions, including unstable angina, decompensated heart failure, severe valvular disease, or significant arrhythmia, we recommend cardiology consultation prior to EVAR or OSR.*

Level of recommendation: Strong
Quality of Evidence: High

*In patients with significant clinical risk factors, such as coronary artery disease, congestive heart failure, cerebrovascular disease, diabetes mellitus, chronic renal insufficiency and an unknown or poor functional capacity (MET < 4) who are to undergo OSR or EVAR, we suggest noninvasive stress testing.*

Level of recommendation: Weak
Quality of evidence: Moderate

We recommend a preoperative resting 12-lead ECG in all patients undergoing EVAR or OSR within 30 days of planned treatment.

Level of recommendation: Strong
Quality of evidence: High

We recommend echocardiography prior to planned operative repair in patients with dyspnea of unknown origin or worsening dyspnea.

Level of recommendation: Strong
Quality of evidence: High

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

Level of recommendation: Weak
Quality of evidence: Moderate

We suggest coronary revascularization prior to aneurysm repair in patients with stable angina and two-vessel disease that includes the proximal left descending artery and either ischemia on non-invasive stress testing or reduced LV function (EF < 50%).

Level of recommendation: Weak
Quality of evidence: Moderate
In patients who may need aneurysm repair in the subsequent 12 months and in whom percutaneous coronary intervention is indicated, we suggest a strategy of balloon angioplasty or bare metal stent placement, followed by four to six weeks of dual-antiplatelet therapy.

Level of recommendation: Weak
Quality of evidence: High

We suggest deferring elective aneurysm repair for 30 days after bare metal stent placement or coronary artery bypass surgery, if clinical circumstances permit. As an alternative, EVAR may be performed with uninterrupted continuation of dual-antiplatelet therapy.

Level of recommendation: Weak
Quality of evidence: Moderate

We suggest deferring open aneurysm repair for at least 6 months after drug-eluting coronary stent placement or, alternatively, performing EVAR with continuation of dual-antiplatelet therapy.

Level of recommendation: Weak
Quality of evidence: Moderate

In patients with a drug-eluting coronary stent requiring open aneurysm repair, we recommend discontinuing P2Y12 platelet receptor-inhibitor therapy 10 days preoperatively with continuation of aspirin. The P2Y12 inhibitor should be restarted as soon as possible after surgery. The relative risks and benefits of perioperative bleeding and stent thrombosis should be discussed with the patient.

Level of recommendation: Strong
Quality of evidence: Moderate

We suggest continuation of beta-blocker therapy during the perioperative period, if part of an established medical regimen.

Level of recommendation: Weak
Quality of evidence: Moderate

If a decision was made to start beta-blocker therapy (due to the presence of multiple risk factors such as coronary artery disease, renal insufficiency, and diabetes); we suggest initiation well in advance of surgery to allow sufficient time to assess safety and tolerability.

Level of recommendation: Weak
Quality of evidence: Moderate

We suggest preoperative pulmonary function studies, including room air arterial blood gases, in 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

We recommend smoking cessation for at least two weeks prior to aneurysm repair.

Level of recommendation: Strong
Quality of evidence: Low

We suggest administration of pulmonary bronchodilators for at least two weeks prior to aneurysm repair in patients with a history of COPD or abnormal pulmonary function testing.

Level of recommendation: Weak
Quality of evidence: Low
We suggest holding angiotensin-converting enzyme inhibitors and angiotensin receptor antagonists on the morning of surgery and restarting these agents after the procedure once euvoilema has been achieved.
Level of recommendation Weak
Quality of evidence Low

We recommend preoperative hydration in non-dialysis dependent patients with renal insufficiency prior to aneurysm repair.
Level of recommendation Strong
Quality of evidence High

We recommend pre- and post-procedure hydration with normal saline or 5% dextrose/sodium bicarbonate for patients at increased risk of contrast-induced nephropathy undergoing EVAR.
Level of recommendation Strong
Quality of evidence High

We recommend holding metformin at the time of contrast administration among patients with an eGFR of < 60 mL/min or up to 48 hours before contrast administration, if the eGFR is < 45 mL/min.
Level of recommendation Strong
Quality of evidence Low

We recommend restarting metformin no sooner than 48 hours after contrast administration as long as renal function has remained stable (< 25% increase in creatinine above baseline).
Level of recommendation Strong
Quality of evidence Low

We recommend perioperative transfusion of packed red blood cells if the hemoglobin level is less than 7 g/dL.
Level of recommendation Strong
Quality of evidence Moderate

We suggest hematologic assessment if the preoperative platelet count is less than 150,000/μL.
Level of recommendation Weak
Quality of evidence Low

Aneurysm imaging

We recommend using ultrasound, when feasible, as the preferred imaging modality for aneurysm screening and surveillance.
Level of recommendation Strong
Quality of evidence High

We suggest that the maximum aneurysm diameter derived from CT imaging should be based on an outer-wall to outer-wall measurement perpendicular to the path of the aorta.
Level of recommendation Good Practice Statement
Quality of evidence Ungraded
We recommend a one-time ultrasound screening for abdominal aortic aneurysms in men or women 65-75 years of age with a history of tobacco use.

Level of recommendation: Strong
Quality of evidence: High

We suggest ultrasound screening for AAA in first-degree relatives of patients who present with an AAA. Screening should be performed in first-degree relatives who are between 65-75 years of age or in those over 75 years of age and in good health.

Level of recommendation: Weak
Quality of evidence: Low

We suggest a one-time ultrasound screening for abdominal aortic aneurysms in men or women over 75 years of age with a history of tobacco use and in otherwise good health who have not previously received a screening ultrasound.

Level of recommendation: Weak
Quality of evidence: Low

If initial ultrasound screening identified an aortic diameter > 2.5 cm but less than 3 cm, we suggest re-screening after 10 years.

Level of recommendation: Weak
Quality of evidence: Low

We suggest surveillance imaging at three-year intervals for patients with an abdominal aortic aneurysm between 3.0 and 3.9 cm.

Level of recommendation: Weak
Quality of evidence: Low

We suggest surveillance imaging at 12-month intervals for patients with an abdominal aortic aneurysm of 4.0 to 4.9 cm in diameter.

Level of recommendation: Weak
Quality of evidence: Low

We suggest surveillance imaging at six-month intervals for patients with an abdominal aortic aneurysm between 5.0 and 5.4 cm in diameter.

Level of recommendation: Weak
Quality of evidence: Low

We recommend a CT scan to evaluate patients suspected to have AAA presenting with recent onset 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

The decision to treat

We suggest referral to a vascular surgeon at the time of initial diagnosis of an aortic aneurysm.

Level of recommendation: Good Practice Statement
Quality of evidence: Ungraded
We recommend repair for the patient who presents with an AAA and abdominal or back pain that is likely attributed to the aneurysm.

Level of recommendation: Strong
Quality of evidence: Low

We recommend elective repair for the patient at low or acceptable surgical risk with a fusiform AAA that is ≥ 5.5 cm.

Level of recommendation: Strong
Quality of evidence: High

We suggest elective repair for the patient who presents with a saccular aneurysm.

Level of recommendation: Weak
Quality of evidence: Low

We suggest repair in women with abdominal aortic aneurysm between 5.0 cm and 5.4 cm in maximum diameter.

Level of recommendation: Weak
Quality of evidence: Moderate

In patients with a small aneurysm (4.0 cm to 5.4 cm) who will require chemotherapy, radiation therapy, or solid organ transplantation, we suggest a shared-decision making approach to decide about treatment options.

Level of recommendation: Weak
Quality of evidence: Low

Medical management during the period of AAA surveillance

We recommend smoking cessation to reduce the risk of AAA growth and rupture.

Level of recommendation: Strong
Quality of evidence: Moderate

We suggest not administering statins, doxycycline, roxithromycin, ACE-inhibitors, or angiotensin receptor blockers for the sole purpose of reducing the risk of AAA expansion and rupture.

Level of recommendation: Weak
Quality of evidence: Low

We suggest not administering beta-blocker therapy for the sole purpose of reducing the risk of AAA expansion and rupture.

Level of recommendation: Strong
Quality of evidence: Moderate

Timing for intervention

We recommend immediate repair for patients who present with a ruptured aneurysm.

Level of recommendation: Strong
Quality of evidence: High
Should repair of a symptomatic AAA be delayed to optimize co-existing medical conditions, we recommend that the patient be monitored in an ICU-setting with blood products available.

Level of recommendation: Strong
Quality of evidence: Low

Assessment of operative risk and life expectancy

We recommend informing patients contemplating open repair or EVAR of their VQI perioperative mortality risk score.

Level of recommendation: Weak
Quality of evidence: Low

Endovascular aneurysm repair

We recommend preservation of flow to at least one internal iliac artery.

Level of recommendation: Strong
Quality of evidence: High

We recommend using FDA approved branch endograft devices in anatomically suitable patients to maintain perfusion to at least one internal iliac artery.

Level of recommendation: Strong
Quality of evidence: High

We recommend staging bilateral internal iliac artery occlusion by at least one to two weeks if required for EVAR.

Level of recommendation: Strong
Quality of evidence: High

We suggest renal artery or superior mesenteric angioplasty and stenting for selected patients with symptomatic disease prior to EVAR or OSR.

Level of recommendation: Weak
Quality of evidence: Low

We suggest prophylactic treatment of an asymptomatic, high-grade stenosis of the SMA in the presence of a meandering mesenteric artery based off of a large IMA, which will be sacrificed during the course of treatment.

Level of recommendation: Weak
Quality of evidence: Low

We suggest preservation of accessory renal arteries at the time of EVAR or OSR if the artery is 3 mm or larger in diameter or supplies more than one-third of the renal parenchyma.

Level of recommendation: Weak
Quality of evidence: Low
**Perioperative outcomes of elective EVAR**

*We suggest that elective EVAR be performed at centers with a volume of at least 10 EVAR cases each year and a documented perioperative mortality and conversion rate to OSR of 2% or less.*

Level of recommendation: Weak
Quality of evidence: Low

**Role of elective EVAR in the high-risk and unfit patient**

*We suggest informing high-risk patients of their VQI perioperative mortality risk score in order to make an informed decision to proceed with aneurysm repair.*

Level of recommendation: Weak
Quality of evidence: Low

**Open surgical repair**

*We recommend a retroperitoneal approach for patients requiring open surgical repair of an inflammatory aneurysm, horseshoe kidney, or an aortic aneurysm in the presence of a hostile abdomen.*

Level of recommendation: Strong
Quality of evidence: Low

*We suggest a retroperitoneal exposure or a transperitoneal approach with a transverse abdominal incision for patients with significant pulmonary disease requiring open surgical repair.*

Level of recommendation: Weak
Quality of evidence: Low

*We recommend a thrombin inhibitor, such as Bivalirudin or Argatroban as an alternative to heparin for patients with a history of heparin-induced thrombocytopenia.*

Level of recommendation: Strong
Quality of evidence: Moderate

*We recommend straight tube grafts for open surgical repair of AAA in the absence of significant disease of the iliac arteries.*

Level of recommendation: Strong
Quality of evidence: High

*We recommend performing the proximal aortic anastomosis as close to the renal arteries as possible.*

Level of recommendation: Strong
Quality of evidence: High

*We recommend that all portions of an aortic graft be excluded from direct contact with the intestinal contents of the peritoneal cavity.*

Level of recommendation: Strong
Quality of evidence: High
We recommend reimplantation of a patent inferior mesenteric artery (IMA) under circumstances that suggest an increased risk of colonic ischemia.

Level of recommendation: Strong
Quality of evidence: High

We recommend preserving blood flow to at least one hypogastric artery in the course of OSR.

Level of recommendation: Strong
Quality of evidence: High

We suggest concomitant surgical treatment of other visceral arterial disease at the time of open surgical repair in symptomatic patients who are not candidates for catheter-based intervention.

Level of recommendation: Weak
Quality of evidence: Moderate

We suggest concomitant surgical repair of an AAA and co-existent cholecystitis or an intraabdominal tumor in patients who are not candidates for EVAR or staged intervention.

Level of recommendation: Weak
Quality of evidence: Low

Perioperative outcomes of open AAA repair

We suggest that elective OSR for AAA be performed at centers with an annual volume of at least 10 open aortic operations of any type and a documented perioperative mortality of 5% or less.

Level of recommendation: Weak
Quality of evidence: Low

The patient with a ruptured aneurysm

We suggest a door-to-intervention time of < 90 minutes, based on a 30-30-30 minute framework, for the management of the patient with a ruptured aneurysm.

Level of recommendation: Good Practice Statement
Quality of evidence: Ungraded

An established protocol for the management of ruptured AAA is essential for optimal outcomes.

Level of recommendation: Good Practice Statement
Quality of evidence: Ungraded

We recommend implementing hypotensive hemostasis with restriction of fluid resuscitation in the conscious patient.

Level of recommendation: Strong
Quality of evidence: Moderate

We suggest that patients with ruptured AAA who require transfer for repair, be referred to a facility with an established rupture protocol and suitable endovascular resources.

Level of recommendation: Good Practice Statement
Quality of evidence: Ungraded
If anatomically feasible, we recommend EVAR over open repair for treatment of a ruptured AAA.

**Level of recommendation** Strong  
**Quality of evidence** Low

### Choice of anesthetic technique and agent

*We recommend general endotracheal anesthesia for patients undergoing open aneurysm repair.*  
**Level of recommendation** Strong  
**Quality of evidence** High

### Antibiotic prophylaxis

*We recommend intravenous administration of a first generation cephalosporin or, in the event of penicillin allergy, vancomycin, within 30 minutes prior to open surgical repair or EVAR. Prophylactic antibiotics should be continued for no more than 24 hours.*  
**Level of recommendation** Strong  
**Quality of evidence** High

*We recommend that any potential sources of dental sepsis be eliminated at least 2 weeks before implantation an aortic prosthesis.*  
**Level of recommendation** Good Practice Statement  
**Quality of evidence** Ungraded

### Intraoperative fluid resuscitation and blood conservation

*We recommend using cell salvage or an ultrafiltration device if large blood loss is anticipated.*  
**Level of recommendation** Strong  
**Quality of evidence** Moderate

*If the intraoperative hemoglobin is less than 10 gm/dL and blood loss is ongoing, we recommend transfusion of packed blood cells along with fresh frozen plasma and platelets in a ratio of 1:1:1.*  
**Level of recommendation** Strong  
**Quality of evidence** Moderate

### Cardiovascular monitoring

*We suggest using pulmonary artery catheters only if the likelihood for a major hemodynamic disturbance was high.*  
**Level of recommendation** Strong  
**Quality of evidence** Moderate

*We recommend central venous access and arterial line monitoring in all patients undergoing open aneurysm repair.*  
**Level of recommendation** Strong  
**Quality of evidence** Moderate
We recommend postoperative ST-segment monitoring for all patients undergoing open aneurysm repair and for those patients undergoing EVAR who are at high cardiac risk.
Level of recommendation    Strong
Quality of evidence     Moderate

We recommend postoperative troponin measurement for all patients with ECG changes or chest pain after aneurysm repair.
Levels of recommendation    Strong
Quality of evidence     High

Maintenance of body temperature

We recommend maintaining core body temperature at or above 36°C during aneurysm repair.
Levels of recommendation    Strong
Quality of evidence     High

Role of the intensive care unit

We recommend postoperative management in an intensive care unit for the patient with significant cardiac, pulmonary or renal disease, as well as for those requiring postoperative mechanical ventilation or who developed a significant arrhythmia or hemodynamic instability during operative treatment.
Level of recommendation    Strong
Quality of evidence     High

Nasogastric decompression and perioperative

We recommend optimization of preoperative nutritional status prior to elective open aneurysm repair, if repair will not be unduly delayed.
Level of recommendation    Strong
Quality of evidence     High

We recommend using nasogastric decompression intraoperatively for all patients undergoing open aneurysm repair, but postoperatively, only for those patients with nausea and abdominal distention.
Level of recommendation    Strong
Quality of evidence     High

We recommend parenteral nutrition if a patient is unable to tolerate enteral support seven days after aneurysm repair.
Level of recommendation    Strong
Quality of evidence     High

Prophylaxis for deep vein thrombosis

We recommend thromboprophylaxis that includes intermittent pneumatic compression and early ambulation for all patients undergoing OSR or EVAR.
Level of recommendation    Strong
Quality of Evidence     High
We suggest thromboprophylaxis with unfractionated or low molecular weight heparin for patients undergoing aneurysm repair at moderate to high risk for venous thromboembolism and low risk for bleeding.

Level of recommendation: Weak
Quality of evidence: Low

Postoperative blood transfusion

*In the absence of ongoing blood loss, we suggest a threshold for blood transfusion during or after aneurysm repair at a hemoglobin concentration of 7 gm/dL or below.*

Level of recommendation: Weak
Quality of evidence: Low

Perioperative pain management

*We recommend multimodality treatment, including epidural analgesia, for postoperative pain control after open surgical repair of an abdominal aortic aneurysm.*

Level of recommendation: Strong
Quality of evidence: High

Late outcomes

*We recommend treatment of Type I endoleaks.*

Level of recommendation: Strong
Quality of evidence: Moderate

*We suggest treatment of Type II endoleaks associated with aneurysm expansion.*

Level of recommendation: Weak
Quality of evidence: Low

*We recommend surveillance of Type II endoleaks not associated with aneurysm expansion.*

Level of recommendation: Strong
Quality of evidence: Moderate

*We recommend treatment of Type III endoleaks.*

Level of recommendation: Strong
Quality of evidence: Moderate

*We suggest no treatment of Type IV endoleaks.*

Level of recommendation: Weak
Quality of evidence: Low

*We recommend open repair if endovascular intervention fails to treat a Type I or III endoleak with ongoing aneurysm enlargement.*

Level of recommendation: Strong
Quality of evidence: Moderate

*We suggest open repair if endovascular intervention fails to treat a Type II endoleak with ongoing aneurysm enlargement.*

Level of recommendation: Weak
Quality of evidence: Low
We suggest treatment for ongoing aneurysm expansion, even in the absence of a visible endoleak.
Level of recommendation Weak
Quality of Evidence Low

We recommend that follow-up of patients after aneurysm repair include a thorough lower extremity pulse exam or ABI.
Level of recommendation Strong
Quality of evidence Moderate

We recommend a prompt evaluation for possible graft limb occlusion if patients develop new onset lower extremity claudication, ischemia, or a reduction in ABI after aneurysm repair.
Level of recommendation Strong
Quality of evidence High

We recommend antibiotic prophylaxis to prevent graft infection prior to any dental procedure involving the manipulation of the gingival or periapical region of teeth or perforation of the oral mucosa, including scaling and root canal procedures, for any patient with an aortic prosthesis, whether placed by open surgical repair or EVAR.
Level of recommendation Strong
Quality of evidence Moderate

We suggest antibiotic prophylaxis prior to respiratory tract procedures, gastrointestinal or genitorurinary procedures, dermatological or musculoskeletal procedures, for any patient with an aortic prosthesis, if the potential for infection exists or the patient is immunocompromised.
Level of recommendation Good practice statement
Quality of evidence Ungraded

After aneurysm repair, we recommend prompt evaluation for possible graft infection if a patient presents with generalized sepsis, groin drainage, pseudoaneurysm formation, or ill-defined pain.
Level of recommendation Strong
Quality of evidence High

We recommend prompt evaluation for possible aortoenteric fistula in a patient presenting with gastrointestinal bleeding after aneurysm repair.
Level of recommendation Strong
Quality of evidence High

In patients presenting with an infected graft in the presence of extensive contamination with gross purulence, we recommend extra-anatomic reconstruction followed by excision of all graft material along with aortic stump closure covered by an omental flap.
Level of recommendation Strong
Quality of evidence Moderate

In patients presenting with an infected graft with minimal contamination, we suggest in situ reconstruction with cryopresevered allograft.
Level of recommendation Weak
Quality of evidence Moderate
In a stable patient presenting with an infected graft, we suggest in situ reconstruction with femoral vein after graft excision and debridement.
Level of recommendation    Weak
Quality of evidence     Moderate

In unstable patients with infected graft, we recommend in situ reconstruction with a silver or antibiotic impregnated graft, cryopreserved allograft, or a PTFE graft.
Level of recommendation    Strong
Quality of evidence     Moderate

Recommendation for postoperative surveillance

We recommend surveillance during the first year after EVAR using contrast enhanced CT at one month and in the absence of an endoleak or sac enlargement, contrast enhanced CT or color duplex sonographic imaging at 12 months.
Level of recommendation    Strong
Quality of evidence     Moderate

If a Type II endoleak is observed one month after EVAR, we suggest postoperative surveillance with contrast enhanced CT and color duplex sonographic imaging at six months.
Level of recommendation    Weak
Quality of evidence     Moderate

If neither endoleak nor AAA enlargement is observed one year after EVAR, we suggest color Duplex ultrasonography when feasible, or CT imaging if ultrasound is not possible, for annual surveillance.
Level of recommendation    Weak
Quality of evidence     Low

If a Type II endoleak is associated with an aneurysm sac that is shrinking or stable in size, we suggest color Duplex ultrasonography for continued surveillance at 6 month intervals for 24 months and then annually thereafter.
Level of recommendation    Weak
Quality of evidence     Low

If a new endoleak is detected, we suggest evaluation for a Type I or Type III endoleak.
Level of recommendation    Weak
Quality of evidence     Low

We suggest non-contrast CT imaging of the entire aorta at five-year intervals after open repair or EVAR.
Level of recommendation    Weak
Quality of evidence     Low
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 and subsequently updated in 2009, for surgeons and physicians who are involved in the preoperative, operative, and postoperative care of patients with abdominal aortic aneurysms (AAA). This document provides recommendations for evaluating the patient, including risk of aneurysm rupture and associated medical co-morbidities, guidelines for 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 graded based on the GRADE approach. 3
The quality of evidence derived from randomized trials has an initial rating of high whereas evidence derived from observational studies has an initial rating of low. GRADE domains are then used to modify this initial rating; these domains include risk of bias, consistency of the results across studies, directness of the populations and interventions of the studies to the question at hand, precision of the estimates of effect and the size of the observed effect. 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. Guideline developers used the terms “we recommend” to denote strong recommendations, whereas for weak recommendations they used the less definitive wording “we suggest.” Thus, quality of evidence was rated 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. On occasions, the guideline committee made good practice statements; which are ungraded recommendations advising about performing certain actions considered by surgeons to be essential for patient care and supported by only indirect evidence.

Literature search and evidence summary

Three systematic reviews were conducted to support this guideline. Two focused on evaluating the best modalities and optimal frequency for post EVAR surveillance. A third umbrella systematic review (overview of reviews) was focused on identifying the best available evidence on the diagnosis and management of AAA. The date range of this search was from 1996 to September 19th, 2016 and included Ovid Medline In-Process & Other Non-Indexed Citations, Ovid MEDLINE, Ovid EMBASE, Ovid Cochrane Central Register of Controlled Trials, Ovid Cochrane Database of Systematic Reviews, and Scopus. The search strategy was
designed and conducted by an experienced librarian with input from the guideline methodologist. Controlled vocabulary supplemented with keywords was used to search for meta-analyses and randomized controlled trials of diagnosis and therapy for abdominal aortic aneurysm. The actual strategy is available as Appendix. This search yielded 1,206 references; from which 29 evidence synthesis reports (systematic reviews and meta-analyses) were used to grade the quality of evidence (Table 1) in various topics that relate to AAA such as screening, diagnosis, surveillance and treatment.

GENERAL APPROACH TO THE PATIENT

History and Risk Factors for Abdominal Aortic Aneurysm

The risk of an abdominal aortic aneurysm (AAA), as well as aneurysm enlargement and rupture, for each patient and related family members, can be largely determined by a thorough medical, family and social history. Abdominal aortic ultrasound screening studies obtained in the United States between 2003 and 2008 were analyzed from more than three million men and women, from diverse racial, ethnic and socioeconomic backgrounds. Participants completed a 36-item questionnaire on demographic and medical, social and family history information, as well as self-reported weight and height.

These data were used to generate a multivariable model for risk, which confirmed age as the most significant risk factor for development of an abdominal aortic aneurysm with a significant increase in risk between the ages of 65 to 69 years (Odds Ratio (OR) 5.4) and 75 to 79 years (OR 14.5). Consistent with prior estimates, an abdominal aortic aneurysm was more likely among men (OR 5.7) and less common in Hispanic (OR 0.7), African American (OR 0.7) and Asian Americans (OR 0.7).

This study also confirmed the close epidemiologic association of cigarette smoking and aneurysmal disease. A smoking history of < 0.5 pack per day for up to 10 years carried a significant increased risk of an abdominal aortic aneurysm (OR 2.6), which increased in a dose-
dependent manner such that smoking more than 1 pack per day for greater than 35 years was associated with a 12-fold increased risk (OR 12.1). Reduced risk was noted for smoking cessation, diabetes mellitus, eating fruits and vegetables more than 3 times a week, and exercise more than once a week. The protective effects of healthy diet and physical activity have been confirmed in other reports. Increasing risk has also been noted with increased salt intake, high blood pressure, concomitant peripheral arterial disease and cerebrovascular disease, and a family history of abdominal aortic aneurysm (Table 2).

Given the prevalence of abdominal aortic aneurysm-related risk factors in the United States, the prevalence of abdominal aortic aneurysm, as defined by an aortic diameter greater than 3 cm, was estimated at 1.4% among those between 50 and 84 years of age, or 1.1 million adults. Importantly, these findings largely concur, and expand upon, prior prevalence and association estimates derived from more homogenous populations such male military veterans. First degree relatives of patients with an abdominal aortic aneurysm have an approximately 20% likelihood of developing an AAA.

The association between cigarette smoking and AAA disease deserves special emphasis. More than 90% of patients with abdominal aortic aneurysm have smoked cigarettes at some point in their lifetime, and AAA is second only to lung cancer in epidemiologic association to cigarette smoking – more closely associated than either cerebrovascular or coronary artery disease. For patients with early aneurysmal disease, a recent meta-analysis concluded that smoking increased the rate of aneurysm enlargement by 35%. Current smokers are over seven times more likely to have an aneurysm than nonsmokers, with duration of smoking the most important variable. Each year of smoking increases the relative risk of developing an aneurysm by 4%. The decades-long decline in per capita cigarette consumption in American adults has been paralleled by a similar decline in deaths from ruptured AAA (Figure 1).
Estimates of the incidence of death from ruptured abdominal aortic aneurysm have declined by more than 50% in the last twenty years, likely due to multiple factors including declining cigarette consumption, increased public awareness of AAA disease, improved surgical outcomes and access to treatment afforded by endovascular repair techniques, and general improvement in management of cardiovascular disease risk factors.\textsuperscript{22-24} However, in countries where cigarette consumption remains high or is increasing, aneurysm-related mortality continues to increase.\textsuperscript{25} Although the risk of inhaled, vaporized nicotine from e-cigarettes and similar nicotine delivery devices has yet to be determined, multiple investigations suggest that exposure to nicotine alone may promote the development and progression of an abdominal aortic aneurysm.\textsuperscript{26-28}

Risk factors for rupture are also relevant when evaluating and managing patients with a known or suspected abdominal aortic aneurysm. In the U.K. Small Aneurysm Trial, the annual risk of rupture was 2.2%. Factors significantly and independently associated with rupture included female gender, large initial aneurysm diameter, low forced expiratory volume in one second (FEV1), current smoking history, and elevated mean blood pressure.\textsuperscript{29, 30} Multiple studies have suggested that women are at greater risk for rupture,\textsuperscript{31, 32} as are patients receiving immunomodulatory therapy following major organ transplantation.\textsuperscript{33-35} Women who smoke are at high risk for an abdominal aortic aneurysm. In a recent Swedish population study, women with a history of smoking of more than a 20 pack-years were nearly twice as likely to develop AAA as men with a similar smoking history.\textsuperscript{36} However, the risk of AAA following smoking cessation declines more rapidly in women than men.\textsuperscript{36} Increased aortic mural calcification has also been suggested as a risk factor for rupture.\textsuperscript{37}

Rupture risk for those unfit for repair in the ADAM trial was 9% per year for patients with aortic diameters between 5.5 and 5.9 cm, 10% for aneurysms between 6.0 and 6.9 cm, and 33% for those $\geq$ 7.0 cm.\textsuperscript{38} More recent experience suggests that rupture estimates based on aortic diameter may need revision downward. Pooled analysis from natural history studies and
control arms of interventional trials indicate that current rupture risk for AAAs between 5.5 and 7.0 cm in diameter may be as low as 5.3% per year and for AAA > 7.0 cm, 6.3% per year. Among asymptomatic patients, the risk of death from causes other than AAA, regardless of aneurysm diameter, was higher than the risk of death from aneurysm rupture.39 Careful review of the surgical history is also essential for accurate and timely recognition of AAA disease. Cholecystitis, appendicitis, or pancreatitis may mimic the presentation of a symptomatic aneurysm. In addition, the nature and extent of previous abdominal surgery may influence the operative approach. When a pulsatile mass is discovered in a patient following prior open surgical repair (OSR) of an AAA, the presence of an anastomotic pseudoaneurysm,40 iliac artery aneurysm,41 or suprarenal aortic aneurysm42 should be considered. Abdominal or back pain after endovascular aneurysm repair (EVAR) should also prompt evaluation of potential aneurysm expansion or rupture.43-45

Physical examination

An abdominal aortic aneurysm is generally defined as an enlargement of the abdominal aorta to ≥ 3.0 cm in diameter. The abdominal aorta begins at the diaphragm, typically at the 12th thoracic vertebra, and lays in the retroperitoneum just anterior and slightly left of the vertebral column. With increasing age, the aorta elongates and enlarges, so the location of a pulsatile mass on physical examination can be variable in location. At the level of the umbilicus and fourth lumbar vertebra, the aorta bifurcates into the right and left common iliac arteries. The focused exam for an aortic aneurysm should be directed at the upper abdominal quadrants.

Physical examination has only a moderate sensitivity for detecting AAA, depending on the extent of abdominal girth and aneurysm size.46 The common iliac arteries may also become aneurysmal and palpable in the lower abdominal quadrants. A number of theories have been proposed to explain the predilection of aneurysmal degeneration to the abdominal aorta and common iliac arteries, but none are definitive.47 Palpation does not precipitate rupture and the
concern for a symptomatic aneurysm should not preclude thorough examination. An abdominal 202
aneurysm is common (37-40%) in patients with popliteal artery aneurysms\(^{48-50}\), as are the 204
presence of concurrent distal arterial aneurysms in patients with an AAA.\(^{51-53}\)

In patients with a suspected or known abdominal aortic aneurysm, we recommend performing 207
physical examination that includes an assessment of femoral and popliteal arteries.

In patients with a popliteal or femoral artery aneurysm, we recommend evaluation for an 211
abdominal aortic aneurysm.

Level of recommendation      Strong
Quality of evidence           High

Assessment of medical comorbidities

Preoperative evaluation of cardiac risk. Despite improvements in cardiovascular risk 219
factor management, five-year survival following successful aneurysm repair remains under 220
70%.\(^{54-56}\) Cardiovascular and pulmonary disease remain the leading causes of early and late 221
death following OSR or EVAR.\(^{57}\) EVAR is associated with a three-fold reduction in perioperative 222
mortality when compared to propensity-matched patients undergoing elective OSR,\(^{58}\) including 223
even younger patients with fewer co-morbidities.\(^{59, 60}\) For both patients with advanced chronic 224
renal insufficiency\(^{61}\) and oxygen-dependent COPD,\(^{62}\) EVAR outcomes are superior to those 225
achieved with contemporary OSR, particularly when performed under local or regional 226
anesthesia. However, despite the reduced risk as compared to OSR, EVAR remains an 227
intermediate to high-risk procedure.

Given the risk associated with either OSR or EVAR, it is essential to evaluate the overall 229
operative risk associated with either method of repair. The first step should be to determine 230
whether an active cardiovascular condition exists (Table 3), which would mandate further 231
assessment and management prior to planned aneurysm repair. In the absence of an active 232
cardiovascular condition, further testing, as dictated by functional capacity and cardiovascular
risk factors, is indicated only if the results will change the planned treatment approach.

Functional capacity can be estimated from a simple activity assessment (Table 4). Patients capable of moderate physical activities, such as climbing two flights of stairs or running a short distance (metabolic activity unit (MET) ≥ 4) will not benefit from further testing. Those who do not function at this level or in whom physical reserve cannot be assessed will benefit from cardiac testing, if it will change operative management. Recent studies suggest that low anaerobic threshold (≤ 10 mL O$_2$/kg/min) during exercise testing, as a measure of aerobic capacity, is predictive of cardiovascular complications, as well as early and late death after aortic aneurysm repair. All patients should be evaluated with a 12-lead ECG within 30 days of planned repair.

Although resting LV function, as determined by an echocardiogram, does not predict postoperative MI or death, echocardiography is recommended for those patients with dyspnea of unknown origin or worsening dyspnea in the setting of a history of congestive heart failure.

In patients with active cardiac conditions, including unstable angina, decompensated heart failure, severe valvular disease, or significant arrhythmia, we recommend cardiology consultation prior to EVAR or OSR.

In patients with significant clinical risk factors, such as coronary artery disease, congestive heart failure, cerebrovascular disease, diabetes mellitus, chronic renal insufficiency and an unknown or poor functional capacity (MET < 4) who are to undergo OSR or EVAR, we suggest noninvasive stress testing.

We recommend a preoperative resting 12-lead ECG in all patients undergoing EVAR or OSR within 30 days of planned treatment.
We recommend echocardiography prior to planned operative repair in patients with dyspnea of unknown origin or worsening dyspnea.

Level of recommendation: Strong
Quality of evidence: High

Preoperative coronary revascularization. A meta-analysis of 22 studies examining more than 13,000 patients with coronary artery disease (CAD) identified an abdominal aortic aneurysm in 8.4% of patients. Routine coronary artery revascularization in patients with stable cardiac symptoms and absent left main coronary artery disease or severe aortic stenosis does not alter the risk of MI, death, or long-term survival among patients undergoing elective vascular surgery. Coronary revascularization is indicated for acute coronary syndrome with or without ST-segment elevation, unstable angina, stable angina in the presence of left main coronary artery or three-vessel disease, as well as for two-vessel disease, including the proximal left anterior descending artery and either ischemia on non-invasive testing or reduced LV function.

The risk for perioperative stent thrombosis for both bare-metal stents (BMS) and drug-eluting stents (DES) in the coronary arteries is highest in the first 4 to 6 weeks after implantation. Surgery should be delayed for 14 days after coronary angioplasty and 30 days after a BMS, if dual-antiplatelet therapy cannot be continued through the perioperative period. Likewise, OSR should not be performed within 6 months following implantation of a drug-eluting stent, if cessation of dual antiplatelet therapy is required. This recommendation assumes the use of newer generation drug eluting stents in patients with stable ischemic heart disease. Thus percutaneous EVAR should be considered the operative method of choice if aneurysm treatment becomes necessary within 6 months after placement of a drug-eluting stent, as dual antiplatelet therapy can typically be continued using this approach.
In summary, a recommendation for percutaneous or surgical intervention for coronary artery disease should follow established clinical practice guidelines, regardless of the need for aneurysm repair. While simultaneous open aneurysm repair and CABG has been reported for select symptomatic patients with critical coronary artery disease, if anatomically feasible, EVAR under local anesthesia would be a preferred option.

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

Level of recommendation: Weak
Quality of evidence: Moderate

We suggest coronary revascularization prior to aneurysm repair in patients with stable angina and two-vessel disease that includes the proximal left descending artery and either ischemia on non-invasive stress testing or reduced LV function (EF < 50%).

Level of recommendation: Weak
Quality of evidence: Moderate

In patients who may need aneurysm repair in the subsequent 12 months and in whom percutaneous coronary intervention is indicated, we suggest a strategy of balloon angioplasty or bare metal stent placement, followed by four to six weeks of dual-antiplatelet therapy.

Level of recommendation: Weak
Quality of evidence: High

We suggest deferring elective aneurysm repair for 30 days after bare metal stent placement or coronary artery bypass surgery, if clinical circumstances permit. As an alternative, EVAR may be performed with uninterrupted continuation of dual-antiplatelet therapy.

Level of recommendation: Weak
Quality of evidence: Moderate

We suggest deferring open aneurysm repair for at least 6 months after drug-eluting coronary stent placement or, alternatively, performing EVAR with continuation of dual-antiplatelet therapy.

Level of recommendation: Weak
Quality of evidence: Moderate
In patients with a drug-eluting coronary stent requiring open aneurysm repair, we recommend discontinuing P2Y12 platelet receptor-inhibitor therapy 10 days preoperatively with continuation of aspirin. The P2Y12 inhibitor should be restarted as soon as possible after surgery. The relative risks and benefits of perioperative bleeding and stent thrombosis should be discussed with the patient.

Perioperative medical management of coronary artery disease. The initiation of beta-blockade prior to non-cardiac surgery has been associated with an increased risk of stroke and all-cause mortality. The use of an alpha-2 agonist is no longer recommended to prevent cardiac events, nor are calcium channel blockers, such as diltiazem and verapamil, given their potential to impair myocardial function in patients with reduced left ventricular function. Continuation of angiotensin-converting enzyme inhibitors and angiotensin receptor blockers is based on individual clinical circumstances.

Aspirin reduces adverse cardiovascular events among patients with coronary artery disease and can be continued during the perioperative period. Both warfarin and the new oral anticoagulants (non-vitamin K antagonist oral anticoagulants) should be discontinued at least 5 days and 2 days, respectively, prior to major surgery. The need for low molecular weight heparin as a bridge depends on the indication for anticoagulation.

We suggest continuation of beta-blocker therapy during the perioperative period, if part of an established medical regimen.

If a decision was made to start beta-blocker therapy (due to the presence of multiple risk factors such as coronary artery disease, renal insufficiency, and diabetes); we suggest initiation well in advance of surgery to allow sufficient time to assess safety and tolerability.
Pulmonary disease. Between 7 and 11% of patients with COPD have an AAA. The prevalence of chronic obstructive pulmonary disease (COPD) in patients presenting with ruptured AAA has largely been attributed to cigarette smoking as a common risk factor.

Common genetic, inflammatory and remodeling pathways may also be present that predispose patients to both conditions. Several studies have reported that COPD is an independent predictor of mortality after open repair with the severity of pulmonary disease and the capacity to optimize preoperative respiratory function influencing outcome. EVAR is better tolerated than OSR, particularly if EVAR is performed under local anesthesia. However, patients with severe COPD exhibit increased in-hospital mortality, pulmonary complications, major adverse events, and decreased 5-year survival whether treated with open repair or EVAR.

If COPD is suspected or present, room air arterial blood gases and standard pulmonary function testing should be performed prior to surgery. In the setting of oxygen-dependent COPD, pulmonary consultation should be obtained for assessment of prognosis and optimization of medical therapy. Smoking cessation prior to aneurysm repair and administration of pulmonary bronchodilators for at least two weeks is recommended. The diagnosis of an aortic aneurysm can be a strong motivator for smoking cessation and efforts to begin smoking cessation prior to surgery can result in long term benefits. Nicotine replacement and use of nortriptyline and bupropion, alone or in combination, along with inpatient and outpatient counseling have proven beneficial for smoking cessation.

We suggest preoperative pulmonary function studies, including room air arterial blood gases, in 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
We recommend smoking cessation for at least two weeks prior to aneurysm repair.

Level of recommendation: Strong
Quality of evidence: Low

We suggest administration of pulmonary bronchodilators for at least two weeks prior to aneurysm repair in patients with a history of COPD or abnormal pulmonary function testing.

Level of recommendation: Weak
Quality of evidence: Low

Renal insufficiency. Preoperative renal insufficiency is an established risk factor for poor outcome after aneurysm repair. Among patients with moderate renal dysfunction (estimated glomerular filtration rate (eGFR) 30-60 mL/min), mortality and cardiovascular events are more likely for patients treated by open surgical repair than by EVAR. However, outcomes are uniformly poor in the presence of severe renal dysfunction (eGFR < 30 mL/min), regardless of the type of repair. Outcomes are equally poor after EVAR or OSR for the patient requiring dialysis with a 30-day mortality of 11% with Kaplan-Meier survival estimates of 66% at 1 year and 37% at three years. Median survival was two years.

Significant declines in renal mass and eGFR have been documented after OSR and EVAR, even in the setting of age-adjusted normal renal function prior to surgery. For example, acute and chronic kidney injury have been noted after complex EVAR with snorkel or renal stent placement, with an increased risk among women. Even transient postoperative renal dysfunction is associated with an increase in mortality, morbidity, and the need for additional intensive care unit support.

Several strategies have been recommended to minimize renal injury after EVAR or OSR. Hydration with either normal saline or sodium bicarbonate is recommended to ensure euvolemia. Similarly, given the association of angiotensin-converting enzyme inhibitors and angiotensin receptor antagonists with hypotension on induction of anesthesia, these medications should be held the morning of surgery and restarted after the patient is
While the administration of many agents have been evaluated, none have proven of value in limiting renal injury after AAA repair. Antioxidants, such as mannitol, prior to or during OSR have demonstrated no benefit. Likewise, fenoldopam, dopamine, atrial natriuretic peptide, diuretics, as well as anti-platelet and anti-inflammatory agents are of no value in the prevention or treatment of acute kidney injury. Lastly, remote ischemic preconditioning (RIPC) has been studied as a strategy for reducing the risk of renal dysfunction. However, systematic review and meta-analysis of the current literature does not confirm the efficacy of this technique in patients undergoing major vascular surgery.

Contrast-induced nephropathy (CIN) is defined as a 25% increase in serum creatinine, or an absolute increase of 0.5 mg/dL, two to seven days following contrast administration. Patients with renal disease (eGFR ≤ 45 mL/min/1.73 m²), diabetes, congestive heart failure, ejection fraction < 40%, hypertension, anemia, advanced age, proteinuria, and gout are at increased risk for CIN. Gadolinium is not a safe alternative to iodinated contrast, given the risk for nephrogenic systemic fibrosis in patients with a GFR of < 30 mL/min/1.73 m².

There is a linear relationship between the volume of contrast administered and the onset and severity of CIN. For every 100 mL of contrast infused during coronary artery interventions, there is a 12% increase in the risk for CIN. Pre-procedural hydration may be beneficial, but fenoldopam, dopamine, atrial natriuretic peptide, theophylline and calcium channel blockers are not. Pre-procedural administration of oral N-acetylcysteine is recommended for at-risk patients, given its low cost, safety profile, and mild protective effect. However, a randomized trial of N-acetylcysteine did not reduce the incidence of CIN after EVAR. Recent evidence suggests that statin therapy may be of benefit in preventing CIN. For example, two studies suggest that initiating high dose statin therapy two days prior to contrast exposure, and continuing for three days afterward, may reduce the risk for CIN undergoing coronary interventions. A recent meta-analysis concluded that the administration of statins along with N-acetylcysteine and intravenous saline had clinically important and statistically significant
benefits as a prevention strategy for CIN when compared to the use of N-acetylcysteine and saline alone.$^{108}$

Contrast agents with osmolality of > 780 mOsm/kg display increased nephrotoxicity.

Additional nephroprotection through further reduction in osmolality was suggested by a study comparing iohexol (Omnipaque, a low-osmolar agent; 600 – 800 mOsm/kg) with iohexanol (Visipaque, an iso-osmolar agent; 290 mOsm/kg).$^{109}$ However, rates of CIN for iopamidol (Iovue-370, 796 mOsm/kg, non-ionic) are similar to iodixanol, which suggests that other physiochemical properties, apart from osmolarity, are important determinants of CIN.$^{110}$

Likewise, several randomized trials of ionic and nonionic contrast agents have demonstrated no difference in CIN.$^{111}$

In summary, minimizing the volume of any type of contrast agent is essential to reducing the risk of CIN, and all nephrotoxic drugs should be stopped at least 48 hours before contrast administration. Patients at increased risk for CIN should be hydrated prior to and after EVAR.

Normal saline at 1 mL/kg/h can be administered for 6 to 12 hours prior to and after the procedure. Alternatively, D5W/sodium bicarbonate can be administered at 3 mL/kg/h for 1 hour prior to EVAR and at 1 mL/kg/h for six hours afterwards. Peri-procedural N-acetylcysteine may be of benefit to reduce CIN. Additional strategies to limit contrast load include use of CO₂,$^{112}$ intravascular or Duplex ultrasound,$^{113}$ or fusion imaging.$^{114}$

We suggest holding angiotensin-converting enzyme inhibitors and angiotensin receptor antagonists on the morning of surgery and restarting these agents after the procedure once euvoolemia has been achieved.

We recommend preoperative hydration in non-dialysis dependent patients with renal insufficiency prior to aneurysm repair.

Level of recommendation: Strong
Quality of evidence: High
We recommend pre- and post-procedure hydration with normal saline or 5% dextrose/sodium bicarbonate for patients at increased risk of contrast-induced nephropathy undergoing EVAR.

Diabetes mellitus. Diabetic patients have increased operative mortality following AAA repair with reduced survival two to five years following surgery, consistent with an increased burden of cardiovascular disease. However, whether diabetes is a distinct risk factor for major adverse events or death after OSR or EVAR is not well defined.

Metformin is a first-line medication for the treatment of type 2 diabetes and prescribed for over 100 million patients worldwide. Metformin is contraindicated if the eGFR is below 30 mL/min per 1.73 m$^2$ due to risk the lactic acidosis, which carries a mortality of up to 50%.

Given the risk of CIN after conventional or CT angiography and the association of metformin with lactic acidosis among patients with renal insufficiency, an eGFR of less than 60 mL/min should prompt the cessation of metformin either at the time of contrast administration or up to 48 hours before, if eGFR is less than 45 mL/min. Metformin should be restarted no sooner than 48 hours after contrast administration as long as renal function has remained stable (less than 25% increase in creatinine above baseline).

We recommend holding metformin at the time of contrast administration among patients with an eGFR of < 60 mL/min or up to 48 hours before contrast administration, if the eGFR is < 45 mL/min.

We recommend restarting metformin no sooner than 48 hours after contrast administration as long as renal function has remained stable (< 25% increase in creatinine above baseline).
Hematologic disorders. The presence of an aortic aneurysm influences both platelet count and function. Low platelet counts and high glycoprotein levels have been observed in patients with an abdominal aortic aneurysm, which has been attributed to increased platelet destruction within the aneurysm sac. While a threshold of platelet count below which elective AAA repair should be deferred has not been addressed, further evaluation is warranted if the platelet count is less than 150,000 platelets/μL. Following elective OSR, a platelet count of less than 130,000/μL is associated with an increased risk of bleeding. Platelet sequestration and thrombocytopenia may occur after OSR, which may persist for several weeks, especially when proximal clamping necessitates periods of renal or visceral ischemia. Matsumura and colleagues suggested that a lower preoperative platelet count was an independent predictor of two year mortality among patients undergoing both EVAR and OSR.

Elevated levels of homocysteine, plasminogen activator inhibitor 1, and lipoprotein(a) have been observed among patients with aortic aneurysms, but their role in aneurysm progression is uncertain. Anticardiolipin antibodies, MTHFR C677T polymorphism, prothrombin gene G20210A variant, and Factor V Leiden mutation are no more common in patients with an aortic aneurysm.

We recommend perioperative transfusion of packed red blood cells if the hemoglobin level is less than 7 g/dL.

We suggest hematologic assessment if the preoperative platelet count is less than 150,000/μL.
Biomarkers and heritable risks for an abdominal aortic aneurysm

Biomarkers for the presence and expansion of an aortic aneurysm. The identification of circulating biomarkers for AAA disease remains an area of active investigation. Such markers may assist in identifying new targets for pharmacotherapy, and may improve both the diagnosis of AAA disease and monitoring the response to medical or surgical therapy. Among 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. A meta-analysis has reported that fibrinogen, D-dimer and thrombin-anti-thrombin III complex levels are increased in patients with AAA. Matrix metalloproteinase 9 (MMP-9), tissue inhibitor of matrix metalloproteinase 1 (TIMP-1), interleukin 6 (IL-6), C-reactive protein (CRP), α1-antitrypsin, triglycerides, lipoprotein (a) (Lp(a)), apolipoprotein A, and high-density lipoprotein (HDL) are also differentially expressed in patients with AAA. While a linear correlation has been noted between CRP and aortic diameter, this observation is at odds with at least one prior report. A number of microRNAs (miRs) related to smooth muscle cell function and collagen formation have also been suggested as possible AAA biomarkers. At the current time, none of these candidates has the sensitivity, specificity, or rigorous clinical validation to be relied upon as a diagnostic or prognostic indicator for rupture risk.

Genetic markers identifying risk of aortic aneurysm. Genetic influences in AAA disease, first suggested by Clifton, has been demonstrated by twin studies and by formal segregation analyses. Genetic predisposition likely represents small contributions from a large number of risk alleles with the effect dependent on the population under consideration, as well as relevant environmental considerations, such as cigarette smoking. Most genomic studies have investigated single nucleotide polymorphisms (SNPs) in genes related to AAA pathogenesis. Potential AAA-related SNPs have been identified in genes encoding for ACE,
5,10-methyltetrahydrofolate reductase (MTHFR), angiotensin II type 1 receptor, interleukin-10, MMP-3 and transforming growth factor beta receptor II. The use of genome-wide DNA linkage analyses relies on traditional proband and family tree studies. Examination of families with two or more members with an AAA has identified variations on chromosomes 4 and 19. Allelic variation at the q31 locus on chromosome 4 may influence endothelin signaling and respiratory epithelial response to injury, such as cigarette smoking. Several genome-wide association studies (GWAS) have also been conducted for various cardiovascular diseases, at least three of which have focused on AAA. A number of genetic loci have been implicated in AAA pathogenesis (Table 5). The non-coding region of chromosome 9p21 has been identified as an important source of heritable risk coronary and peripheral arterial disease, as well as AAA, independent of smoking, hypertension and hyperlipidemia. The at-risk allele may mediate this effect by down regulating the cell-cycle regulatory gene, CDKN2B. Epigenetic regulation of gene expression through microRNA production and post-translation regulation of gene expression may also influence inflammation, fibrosis, or other mechanisms relevant to AAA pathogenesis.

Some monogenic diseases increase the risk of AAA, including mutations within the COL3A1 gene, associated with an autosomal dominant defect in type III collagen synthesis present in patients with the Ehlers Danlos phenotype or mutations in fibrillin 1, responsible for Marfan Syndrome. In Ehlers Danlos and Marfan Syndromes, isolated AAA is uncommon in the absence of other arterial aneurysms or aortic dissection, respectively.

**Aneurysm imaging**

**Modalities for aneurysm imaging.** Among asymptomatic patients, ultrasound detects the presence of an abdominal aortic aneurysm accurately, reproducibly, and efficiently. Sensitivity and specificity approach 100%, but in 1% to 3% of patients, the aorta cannot be visualized due to bowel gas or obesity. Transabdominal ultrasound is ideal for screening
and surveillance, but insufficiently precise for procedural planning or more complex morphological analyses.

Computed tomographic (CT) imaging is more reproducible than ultrasound, with > 90% of measurements within 2 mm of the initial reading. Both techniques suffer from a lack of standardization in terms of determining the degree and rate of disease progression. An aneurysm measured by standard axial CT is generally more than 2 mm larger in diameter than when measured by ultrasound. Most commonly, the cross-sectional measurement obtained by CT is not necessarily perpendicular to the path of the aorta, which presumably contributes to an over-estimation of aneurysm size. There is also significant variability in reporting of aneurysm diameter, particularly in research studies, which have included diameter measurements based on outer-wall to outer-wall, inner-wall to inner-wall, and anterior outer-wall to posterior inner-wall. Lack of precision of diameter measurements based on orthogonal rendering, as well as path lengths and centerline measurements have been largely superseded by the adoption of three-dimensional reformatting software and dedicated computer workstations to obtain curved multiplanar reformatted images.

The increasingly small size and low cost of portable ultrasound units and absence of radiation or nephrotoxic contrast administration with ultrasound has made it the preferred technique for aneurysm screening and surveillance. For operative planning, however, CT remains an essential tool, given its precision, reproducibility, resolution, capacity for complete anatomic examination, and data conversion into numerous reformatting and measurement programs. It bears noting that plain abdominal films and catheter-based digital subtraction angiography have low sensitivity for the detection of AAA. The luminal contour of the aneurysmal aorta visualized by angiography may be obscured by accumulated mural thrombus, particularly in the case of larger aneurysms.

Ultrasound has become a mainstay of emergency medical practice and used with increasing accuracy and facility in the differential diagnosis of abdominal pain. High sensitivity
and specificity have been reported in detecting non-ruptured aneurysms\textsuperscript{167, 168} and use of bedside ultrasound significantly reduces time to diagnosis and treatment.\textsuperscript{169, 170} However, regions of the retroperitoneum may not be well visualized in non-fasting patients or those with ileus or excessive intestinal gas.\textsuperscript{171}

The accuracy of CT imaging for diagnosis of symptomatic and ruptured AAA has also improved due to advances in coordinated timing and appropriate dosage of contrast, as well as through the use of multi-detector arrays and post-image processing. Timed contrast boluses greatly increases the sensitivity and specificity of CT imaging.\textsuperscript{172} With modern equipment and imaging techniques, false positive CT interpretation is very low\textsuperscript{173} and radiographic findings of rupture are well-characterized.\textsuperscript{174}

We recommend using ultrasound, when feasible, as the preferred imaging modality for aneurysm screening and surveillance.

Level of recommendation Strong
Quality of evidence High

We suggest that the maximum aneurysm diameter derived from CT imaging should be based on an outer-wall to outer-wall measurement perpendicular to the path of the aorta.

Level of recommendation Good Practice Statement
Quality of evidence Ungraded

**Prediction of aneurysm expansion and rupture risk.** A significant unmet need in the assessment of AAA disease is a determination of rupture risk. Maximum AAA diameter remains the most widely used and validated criteria for prediction of rupture risk. The adoption of maximum diameter as a measure of rupture risk was based, in part, on a retrospective review of 24,000 consecutive autopsies performed over 23 years at a single institution.\textsuperscript{175} Of the 473 non-resected AAA identified in this series, 118 were ruptured. Approximately 40\% of AAA greater than 5 cm in diameter were ruptured. However, 40\% of AAA between 7 and 10 cm were not ruptured, while 13\% of AAA less than 5 cm in size were ruptured.\textsuperscript{175} Thus, a variety of
potentially more sensitive predictors of rupture risk have been proposed, including AAA expansion rate,\textsuperscript{30, 38, 176, 177} wall stiffness,\textsuperscript{178-180} wall tension,\textsuperscript{181} and peak AAA wall stress.\textsuperscript{182-184}

Hall and colleagues have suggested that rupture is imminent above a critical aortic wall stress, predicted by the Law of Laplace and maximum AAA diameter.\textsuperscript{181} Several other investigators subsequently demonstrated that wall stress is highly dependent on AAA shape rather than diameter alone.\textsuperscript{182-188} With ultrasound-based assessment, determination of peak wall stress may soon be translatable to real-time patient assessment.\textsuperscript{189} Modeling by computational fluid dynamics has suggested that intraluminal hemodynamic conditions also influence AAA growth, remodeling, and risk of rupture.\textsuperscript{190-194}

The peak wall rupture index considers both peak wall stress and residual wall strength and has been proposed as more predictive than estimates of peak wall stress alone.\textsuperscript{195-197} Further analyses have also incorporated the influence of intraluminal hemodynamic conditions on wall stress/strength indices through fluid structure interaction simulations.\textsuperscript{198, 199} The value of CT-determined intraluminal thrombus volume\textsuperscript{200 201}, as well as PET-CT imaging in predicting rupture risk remains uncertain.\textsuperscript{202, 203} The utility of PET imaging may require the development of AAA-specific radiotracer agents.\textsuperscript{204}

Beyond an assessment of rupture risk, there is a clear role for imaging-based criteria for the prediction of disease progression. As revealed in surveillance studies, many small AAAs do not enlarge.\textsuperscript{205} Molecular imaging of pathologic processes characteristic of aortic degeneration, including angiogenesis,\textsuperscript{206} matrix disruption,\textsuperscript{207} activated macrophage localization,\textsuperscript{208} and proteolysis\textsuperscript{209} are being translated to the clinic\textsuperscript{210-213} and may help to identify those patients with an increased likelihood of disease progression.

Gender-specific rupture indicators have been evaluated given the increased risk for rupture in women at any given aortic diameter. A recent computational study of patient-specific anatomy using finite element analysis was unable to identify significant differences in peak wall stress and peak wall rupture indices between men and women.\textsuperscript{214} Although aneurysm diameter
remains a well-established parameter for clinical decision-making, a retrospective analysis has suggested that aneurysm diameter indexed to body size (aortic size index (ASI) = aneurysm diameter (cm)/BSA (m²)) may represent a superior predictor of rupture risk for women.\(^{215}\)

**Recommendations for aneurysm screening.** Aneurysm screening has been motivated by a desire to reduce AAA-related mortality and prolong life expectancy. Overall, the probability of AAA in the general population is very low, but significantly increased when certain risk factors are present.\(^4\) Four randomized clinical trials that included 127,891 men and 9,342 women between 65 and 79 years of age provide evidence that ultrasound screening is effective in reducing aneurysm-related mortality.\(^{216-221}\) This benefit begins within 3 years of testing and persists for up to 15 years.\(^{222}\) In addition, screening is associated with a reduced risk for AAA rupture and emergency surgery.\(^{222}\)

The MASS group, the largest of the four randomized clinical trials, reported that over 13 years there was a 42% reduction in AAA-related mortality and a small reduction in all-cause mortality. It was estimated that 216 men needed to be invited to screening to save one death over 13 years. Of those aneurysms that did rupture, roughly half had an initial baseline diameter of 2.5 to 2.9 cm at initial screening.\(^{223}\)

Screening for AAA in women is more controversial. Since few women were included in these trials, a decrease in AAA-related mortality or incidence of rupture could not be identified.\(^{224}\) Although AAA prevalence is lower in women, the rate of rupture and overall life expectancy are higher, which suggest that screening may be more cost-effective in women.\(^{225}\)

In 2005 the U.S. Preventative Services Task Force (USPSTF) recommended a one time screening by abdominal ultrasonography for men aged 65 to 75 with a history of smoking.\(^{226}\) In 2014, the USPSTF updated their 2005 recommendations to include one time ultrasound screening for men aged 65 to 75 who have ever smoked (grade B) and selective screening of 65 to 75 year old men who have never smoked (grade C). Screening was not recommended for
women aged 65 to 75 who have never smoked (grade D) and evidence was insufficient to recommend for or against screening in women aged 65 to 75 who had a smoking history. The USPSTF recommendations are based on the assumption that 6 to 7% of men with a smoking history in the 65 to 75 year old age group will have an AAA. In the absence of a smoking history, the prevalence drops to 2%. The prevalence of AAA is 0.8% in a similar age group of women who have a past history of smoking, but for women who are current smokers the prevalence is 2%. In women who have never smoked, AAA prevalence is less than 0.60%.

In 2007, following passage of the SAAAVE (Screening Abdominal Aortic Aneurysms Very Efficiently) amendment in 2006, the U.S. Centers for Medicare and Medicaid Services began offering one time screening by ultrasonography for men aged 65 to 75 if they had smoked ≥ 100 cigarettes in their lifetime, and men and women with a family history of AAA disease, as part of their “Welcome to Medicare” physical examination. As originally mandated, the screening could only be ordered by the primary care physician, within 6 months of the activation of Medicare benefits. However, by 2012 it was apparent that fewer than 3% of abdominal ultrasound claims were for SAAAVE-specific AAA screening, and although abdominal ultrasounds in the affected age groups had increased, there was no discernible effect on AAA rupture or all-cause mortality. Subsequent Medicare eligibility guidelines have been modified to allow additional physician specialists to order the tests and increase the window of eligibility.

Many additional opportunities exist to improve screening and surveillance practices in the United States. Currently, 40% of operative repairs in Medicare beneficiaries are performed late in the course of the disease, suggesting that a prior screening opportunity had been missed. A significant portion of patients present with rupture despite known AAA status. In an analysis of a cohort of more than 3 million individuals, it was suggested that small changes in recommended eligibility requirements, such as accounting for the impact of accumulated
cardiovascular risk factors, would improve the screening yield for women, non-smokers, and
other groups traditionally considered at lower risk.4

Effective use of the electronic health record to improve screening of target populations
has been demonstrated by Kaiser Permanente of Southern California.230 “Best practice alerts”
for AAA screening criteria were integrated into the electronic health record and reduced the
percentage of unscreened patients within their 3.6 million subscriber system from 52% to 20%
within 15 months. These alerts were directed to nursing staff when patients checked in for any
visit with any provider. Automatic screening orders were then entered that clinicians would sign
when visit specific relevant orders were completed. Patients who had undergone any type of
cross-sectional abdominal imaging in the prior ten years (>54,000) were excluded from the alert
system.230

In addition to the United States, government-sponsored screening programs have been
implemented in the U.K. and Sweden and the results reflect the changing epidemiology of
aneurysmal disease. In Sweden the screening yield was less than half that expected, despite
widespread participation.231 Similarly, in the first three years of the U.K. National Abdominal
Aortic Aneurysm Screening Program AAA detection rate was only 1.6%, less than 50% of that
expected based on the results of the prior MASS cohort.232 However, cost-effectiveness
calculations derived from MASS (£7,600/life-year gained) suggest that AAA screening in
England and other European countries will remain cost-effective even with prevalence rates as
low as 1%.233-235

Other challenges to efficient implementation of screening include identifying and
excluding patients who have had prior abdominal cross-sectional imaging studies, accounting
for increasing longevity, the potential needs for late rescreening,236 and selective screening to
optimize yield at minimal cost.237 Reducing all-cause mortality and enhancing yield might be
increased by integrating AAA screening with concurrent echocardiograms238 or blood pressure
and peripheral vascular disease testing. Ensuring full participation for all patient groups at risk remains an ever-present challenge.

We recommend a one-time ultrasound screening for abdominal aortic aneurysms in men or women 65-75 years of age with a history of tobacco use.

Level of recommendation: Strong
Quality of evidence: High

We suggest ultrasound screening for AAA in first-degree relatives of patients who present with an AAA. Screening should be performed in first-degree relatives who are between 65-75 years of age or in those over 75 years of age and in good health.

Level of recommendation: Weak
Quality of evidence: Low

We suggest a one-time ultrasound screening for abdominal aortic aneurysms in men or women over 75 years of age with a history of tobacco use and in otherwise good health who have not previously received a screening ultrasound.

Level of recommendation: Weak
Quality of evidence: Low

If initial ultrasound screening identified an aortic diameter > 2.5 cm but less than 3 cm, we suggest re-screening after 10 years.

Level of recommendation: Weak
Quality of evidence: Low

Recommendations for aneurysm surveillance. The optimal frequency for surveillance following recognition of early-stage AAA disease has not been defined by randomized clinical study. Some authors have suggested that there is no need to follow patients with an initial aortic diameter less than 3 cm given their low risk for rupture.

However, in a 12 year analysis of 1,121 men aged 65 or older, 13.8% of aortas with an initial 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, 10.5% exceeded 5.5 cm or required surgery within two years. Rupture occurred in 1.4%.

Two randomized controlled trials, the U.K. Small Aneurysm Trial, and the US Veterans Affairs Aneurysm Detection and Management Trial, as well as a follow-up study of patients detected in the U.K. MASS trial, demonstrated that a policy of surveillance until aneurysm diameter exceeds 5.5 cm was safe and associated with a rupture rate of 1% per year. While aortic size was defined by the maximum external aortic diameter, the surveillance frequency differed among these studies.

In an analysis of expansion rates of 1,743 participants in the U.K. Small Aneurysm Trial, AAA growth rate increased with aneurysm size and among current smokers, was lower in those with low ankle-brachial index and diabetes, and was unaffected by lipids and blood pressure. Combining the results of 18 surveillance studies with similar imaging and assessment protocols, the RESCAN collaborators identified a pooled growth rate across all studies of 2.2 mm per year, with no significant difference between men and women. When estimates were pooled using random-effects meta-analysis, following further adjustment for all demographics, medical and drug history, rates of aneurysm enlargement were significantly increased in smokers and decreased in those with diabetes. Pooled meta-analysis failed to identify the effects of any class of drug on aneurysm expansion. After adjustment for initial aneurysm diameter, medical history and demographics, a strong association was noted between smoking and rupture (HR 2.02, 95% CI 1.33 - 3.06, p = 0.001), and a far higher risk for women than men (HR 3.76, 95% CI 2.58 - 5.47, p < 0.001). Rupture risk was increased in older participants, those enrolled in earlier studies, those with lower BMI and higher mean arterial or pulse pressure. The effect of any class of drug was difficult to evaluate based on the low incidence of rupture.

Thompson and associates performed a meta-regression of growth estimates based on aneurysm diameter and time to a 10% probability of attaining an aortic diameter of
Integrating cost-effectiveness data, the authors proposed recommendations for surveillance intervals based on aortic size. Several years was recommended for men with an initial AAA diameter between 3.0 and 4.0 cm range, whereas an interval of one year was recommended for AAAs between 4.0 and 4.9 cm and six months for those between 5.0 and 5.4 cm. However, the presence of diabetes, female sex, and current smoking history were not accounted by the model or considered within this set of recommendations. The increased risk of aneurysm rupture in women and patients with a smoking history has confounded attempts at standardizing surveillance intervals.

We suggest surveillance imaging at three-year intervals for patients with an abdominal aortic aneurysm between 3.0 and 3.9 cm.

We suggest surveillance imaging at 12-month intervals for patients with an abdominal aortic aneurysm of 4.0 to 4.9 cm in diameter.

We suggest surveillance imaging at six-month intervals for patients with an abdominal aortic aneurysm between 5.0 and 5.4 cm in diameter.

Recommendations for imaging the symptomatic patient. In patients with abdominal or back pain, ultrasound imaging is recommended to determine if an AAA is present and to evaluate for the presence of other causes of abdominal or back pain. If an aneurysm is detected, the patient should have a CT aortogram with timed intravenous contrast injection, if not contraindicated, to exclude rupture and facilitate operative planning. A patient presenting with a large AAA and back or abdominal pain should be referred for treatment, as soon as an
aneurysm is recognized, regardless of evidence for rupture, symptom evolution, or if a CT scan has been completed. If hemodynamic compromise is present or evolves during the process of evaluation, further imaging studies should be abandoned as care is escalated.

We recommend a CT scan to evaluate patients suspected to have AAA presenting with recent onset 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 ABDOMINAL AORTIC ANEURYSM

The decision to treat. It is recognized that the majority of patients will be asymptomatic at the time of diagnosis of an AAA. Less frequently, the first presentation of an unrecognized AAA may, in fact, be a symptomatic aneurysm manifested by abdominal or back pain or even rupture. Should this be the case, prompt treatment is recommended.

Most AAAs are fusiform rather than saccular and current recommendations for treatment of asymptomatic fusiform AAA rests primarily on the maximum transverse diameter as measured on ultrasound, computed tomography, or magnetic resonance imaging. Conventional arteriography can easily underestimate the true diameter by not accounting for luminal thrombus.

There is general agreement that small aneurysms, less than 4.0 cm in maximum diameter, are at low risk of rupture and should be monitored whereas an aneurysm greater than 5.4 cm in diameter should be repaired in an otherwise healthy patient. Elective repair is also recommended for patients who present with a saccular aneurysm and while size guidelines are currently lacking due to their infrequent presentation, repair is generally recommended at a smaller diameter.
Some controversy exists regarding treatment strategies for patients who present with an AAA between 4.0 and 5.4 cm. In the United Kingdom Small Aneurysm Trial (UKSAT)²⁴⁷ and the Aneurysm Detection and Management (ADAM) Trial,²⁴⁸ the 30-day operative mortality in the immediate surgery groups (5.5% UKSAT, 2.1% ADAM) led to an early disadvantage in survival. The investigators found no statistically significant difference in long-term survival between the immediate open surgical repair and surveillance groups. Currently nearly 80% of all AAAs are treated by EVAR in the United States.²⁴⁹,²⁵⁰ Given the less invasive nature of EVAR, two studies re-evaluated the appropriateness of intervention for small aneurysms. The Comparison of Surveillance versus Aortic Endografting for Small Aneurysm Repair (CAESAR)²⁵¹ and Positive Impact of Endovascular Options for Treating Aneurysms Early (PIVOTAL)²⁵² trials compared immediate EVAR or surveillance for AAA between 4.1 and 5.4 cm (CAESAR) and 4.0 and 5.0 cm (PIVOTAL) and found no survival benefit for early EVAR, but neither trial was designed to determine whether immediate EVAR might be beneficial or harmful for specific AAA size ranges or age subgroups. A Cochrane Database Review²⁵³ of these four studies demonstrated no advantage to immediate repair by open surgery or EVAR for small AAA (4.0 - 5.5 cm).

Patients with an asymptomatic fusiform AAA greater than 5.4 cm should be considered for repair and surveillance is recommended for smaller aneurysms. An individualized approach may be appropriate for patients with an AAA greater than 5.4 cm, but who are of advanced age or have significant co-morbid conditions. Alternatively, young, healthy patients, particularly women, with an AAA between 5.0 and 5.4 cm or those with rapid expansion of small fusiform AAAs may benefit from early repair.²⁴⁶,²⁵⁴-²⁵⁶

We suggest referral to a vascular surgeon at the time of initial diagnosis of an aortic aneurysm.

Level of recommendation: Good Practice Statement
Quality of evidence: Ungraded
We recommend repair for the patient who presents with an AAA and abdominal or back pain that is likely attributed to the aneurysm.

Level of recommendation    Strong
Quality of evidence     Low

We recommend elective repair for the patient at low or acceptable surgical risk with a fusiform AAA that is ≥ 5.5 cm.

Level of recommendation    Strong
Quality of evidence     High

We suggest elective repair for the patient who presents with a saccular aneurysm.

Level of recommendation    Weak
Quality of evidence     Low

We suggest repair in women with abdominal aortic aneurysm between 5.0 cm and 5.4 cm in maximum diameter.

Level of recommendation    Weak
Quality of evidence     Moderate

In patients with a small aneurysm (4.0 cm to 5.4 cm) who will require chemotherapy, radiation therapy, or solid organ transplantation, we suggest a shared-decision making approach to decide about treatment options.

Level of recommendation    Weak
Quality of evidence     Low

Medical management during the period of aneurysm surveillance. In the presence of a small aortic aneurysm, several approaches have been proposed to prevent further enlargement. Smoking cessation is the most important intervention for a patient with an aneurysm. Hemodynamic control with propranolol has not been shown to inhibit aneurysm expansion. Despite the benefits of statins in cardiovascular disease, their ability to limit aneurysm expansion is lacking. Angiotensin-converting enzyme (ACE) inhibitors and Losartan, an angiotensin receptor antagonist, decrease the rate of AAA expansion in mice,
but clinical investigations have reported conflicting results.\textsuperscript{266, 267} Clinical trials of beta-adrenergic receptor blockade demonstrate no effect on the rate of aneurysm progression.\textsuperscript{261, 268} Likewise, beta-blockade, lipid-lowering agents, and angiotensin receptor blockade do not appear to alter rupture risk, but an increased risk of rupture has been reported for patients who recently discontinue ACE inhibitors.\textsuperscript{269}

Some have suggested that serological evidence of \textit{Chlamydophili pneumonia} infection may be associated with AAA expansion,\textsuperscript{270} but a prospective randomized trial demonstrated that azithromycin had no effect aneurysm enlargement.\textsuperscript{271} Doxycycline can inhibit matrix metalloproteases in plasma and aneurysm tissue and, thus, has been proposed as an agent to limit AAA growth.\textsuperscript{272, 273} However, a randomized trial of low dose doxycycline (100 mg once daily) demonstrated no reduction in aneurysm growth during an 18 month period.\textsuperscript{274} An ongoing NIH trial is examining the effectiveness of a higher dose of doxycycline (100 mg twice daily).

In summary, during the surveillance period, patients should be counseled to cease smoking if tobacco products are being used. Patients should be encouraged to seek appropriate medical management for hypertension, hyperlipidemia, diabetes, and other atherosclerotic risk factors. A statin and ACE inhibitor should be considered, given the broad potential benefits to cardiovascular disease and acceptable safety profile. Insufficient data currently exist to recommend use of doxycycline or roxithromycin. Patients should be counseled that moderate physical activity does not precipitate rupture or influence AAA growth rate.\textsuperscript{12, 275}

\textit{We recommend smoking cessation to reduce the risk of AAA growth and rupture.}

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\textit{We suggest not administering statins, doxycycline, roxithromycin, ACE-inhibitors, or angiotensin receptor blockers for the sole purpose of reducing the risk of AAA expansion and rupture.}

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We suggest not administering beta-blocker therapy for the sole purpose of reducing the risk of AAA expansion and rupture.

Level of recommendation: Strong
Quality of evidence: Moderate

Timing of intervention. A patient with a known abdominal aortic aneurysm or pulsatile mass on abdominal exam who presents without hemodynamic instability and acute onset of back or abdominal pain should undergo an immediate CT scan to determine if rupture has occurred. While a ruptured AAA represents a surgical emergency, the timing of aneurysm repair for patients with a symptomatic but non-ruptured aneurysm represents a clinical dilemma. Under select circumstances, it may be appropriate to delay intervention for several hours to ensure conditions for successful repair, including optimizing anesthetic support, as well as blood product or device availability. If such an approach is elected, the patient should be closely monitored in an intensive care unit.

A more frequent concern is the timing for treatment of an asymptomatic, large abdominal aortic aneurysm, greater than 5.4 cm in diameter. In the ADAM trial, rupture risk was estimated at 10% per year for aneurysms between 5.5 and 6.9 cm diameter, but over 33% per year when aneurysms were 7 cm or larger in diameter. Recent reports, however, suggest that contemporary rupture rates may be lower than previously estimated. For example, a pooled analysis from natural history studies and control arms from interventional trials, calculated a rupture risk of 6.3% per year for aneurysms larger than 7 cm in diameter. In general, should a patient be considered a surgical candidate, repair of a large aneurysm should not be unduly delayed. Pertinent preoperative assessment should be conducted in a timely manner in order to optimize outcomes, especially for patients with associated co-morbid conditions. Given the risk of rupture, both patient and family need to understand and accept the rationale for any delay related to further evaluation.
Whether a recent surgical procedure, such as an abdominal colectomy, coronary artery bypass, or prostatectomy, can increase the likelihood of aneurysm rupture remains an unsettled question. It has been suggested that inflammation and the induction of a catabolic state may result in enhanced collagen proteolysis with an increased risk of rupture. However, animal studies have not found evidence of increased aortic collagenase activity nor has this notion been supported by a prospective clinical study. It seems unlikely that the risk of aneurysm rupture is substantially increased by an unrelated operation and that a several week delay to enable satisfactory recovery is acceptable prior to elective abdominal aortic aneurysm repair.

In summary, the optimal timing of abdominal aortic aneurysm repair is based on clinical presentation and aneurysm status: 1) a ruptured abdominal aortic aneurysm requires emergent repair; 2) a symptomatic, non-ruptured aneurysm is best treated urgently; and 3) an asymptomatic abdominal aortic aneurysm can be treated electively following completion of preoperative assessment. Delay in the treatment of an asymptomatic, large abdominal aortic aneurysm should be minimized.

We recommend immediate repair for patients who present with a ruptured aneurysm.

Assessment of operative risk and life expectancy. Several prediction models have been developed to estimate operative risk for open AAA repair and EVAR and hold the promise of better informing patients of their individual risk of perioperative mortality and provide surgeons a useful tool to ensure an informed discussion with patients and their families. Risk
prediction models for aneurysm repair were first developed in the 1990s; largely derived from relatively small cohorts of several hundred patients treated by open surgical repair. The most well-known of this first generation of risk model were the Glasgow Aneurysm Score, the Leiden Score, and the Hardman Index. As one example, the Glasgow Aneurysm Score was developed from a cohort of 268 open AAA repairs, where 41% of patients presented with ruptured aneurysms and the overall mortality was 20%. The risk score accounted for age, the presence of shock, renal disease, and a history of myocardial or cerebrovascular disease. The EUROSTAR collaborators suggested that the GAS could be used to estimate mortality for EVAR with 30 day mortality of 1.1% for GAS score < 74, 2.1% for GAS 74 to 84, and 5.3% for GAS > 84.

Over the past 7 years, a variety of new risk scoring schemes have been derived from an assessment of patients who have undergone either open repair or EVAR, in order to specifically account for the mortality risk associated with EVAR. Egarova used Medicare data to identify a EVAR patients with increased operative risk due to the presence of many of the same risk factors for mortality that had been previously identified among patients undergoing open repair, including age, renal failure, CHF, PAD, and liver disease. They found that only 3.4% of Medicare patients undergoing EVAR had an operative risk > 5%, but a subset, which represented <1% of patients undergoing EVAR, was identified with a predicted mortality of > 10%. In an analysis of Medicare patients undergoing open repair and EVAR, including a review of prior Medicare claims data to obtain a reliable assessment of pre-existing comorbidities, Schermerhorn found age, renal failure, heart failure, female sex, and peripheral or cerebral vascular disease to be predictive of perioperative mortality for either EVAR or open aneurysm repair (C-statistic 0.726). For the first time, a single scoring scheme was developed that could be applied to patients to assess risk for either EVAR or open surgical repair. Recently, Eslami and collaborators used the Vascular Study Group of New England (VSGNE) database to develop a new risk model, which included anatomic features, such as aneurysm diameter, neck...
length, and level of clamp placement that had not been incorporated in prior scoring schemes (C-statistic 0.822; Tables 6A and 6B). This model has since been validated using the Vascular Quality Initiative (VQI) database and has been recently endorsed by the VQI for risk stratification of patients under consideration for planned open repair or EVAR.

The delivery of clinically appropriate care requires balancing operative risk with the likelihood of late survival. Patients with aortic aneurysms suffer higher rates of heart attacks, strokes, and major amputation and have an increase in five-year mortality compared with age-matched controls. Bahia and colleagues recently conducted a systematic review of long-term survival after aneurysm repair. Patients with large aneurysms, at greatest risk of rupture, also had significantly worse five-year survival. As one would anticipate, many of the same risk factors for perioperative death also impact life expectancy. One-year survival after hospital admission for heart failure is 60%. One-year survival after initiation of dialysis is 85%, but decreases for those with significant comorbidities to 60%. The three-year survival after initiating home oxygen therapy for COPD is 60%. In the EVAR-2 trial, patients with severe coronary artery disease, COPD, or poor renal function were considered ineligible for open repair. While the study has been criticized for its trial design, EVAR did not impact overall survival. Two year survival was 60% and five year survival was 35%. De Martino et al. assessed survival after EVAR within the VSGNE population using the EVAR-2 trial criteria. Five-year survival for patients with aneurysms smaller than 6.5 cm was 46% and for patients with aneurysms larger than 6.5 cm, five-year survival was 28%. In a recent study, those patients declined for AAA repair had a 2-year survival of 35%. Thus, for a patient with high operative risk and shortened life expectancy, rupture risk must be high to benefit from EVAR.

We recommend informing patients contemplating open repair or EVAR of their VQI perioperative mortality risk score.

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Endovascular aneurysm repair

Endovascular aortic aneurysm repair has rapidly expanded as the preferred approach for treatment of AAA since the first report more than 25 years ago.\textsuperscript{288, 289} Since the introduction of EVAR, the annual number of deaths from intact and ruptured AAAs has significantly decreased in the United States. This has coincided with an increase in elective AAA repair and a decrease in the diagnosis and repair of ruptured AAAs.\textsuperscript{23}

**Considerations for percutaneous repair.** EVAR has evolved since its inception with the development lower profile delivery sheaths that are tapered, flexible, and coated for low-resistance introduction into the femoral arteries. Concomitantly, devices have been designed to facilitate percutaneous closure of femoral artery puncture sites of increasing dimension. Together, this has reduced the requirement for open surgical exposure of the femoral artery. A randomized study comparing open exposure and the percutaneous “preclose” technique using the Perclose ProGlide\textsuperscript{®} (Abbott Vascular, Inc.) device demonstrated both safety and effectiveness.\textsuperscript{290} Following femoral artery access, systemic anticoagulation with 100 U/kg of intravenous heparin is recommended with a target activated clotting time $\geq 300$ sec.

**Infrarenal fixation.** EVAR requires non-aneurysmal proximal and distal attachment sites or sealing zones as dictated by device specific instructions for use. Most endografts that are dependent upon infrarenal fixation have required a proximal sealing zone of at least 15 mm in length, a neck diameter less than 32 mm, and a neck angulation of less than 60°. Several devices now report efficacy with shorter neck lengths and more severe levels of angulations. Use of devices outside recommended parameters increase the risk of device migration, delayed Type IA endoleaks, and aneurysm rupture.

**Suprarenal fixation.** Suprarenal fixation has been proposed as a more effective means of proximal fixation when the morphologic features of the proximal aortic neck are unfavorable, including shortened neck length, severe angulation, reverse taper, barrel-shaped neck, circumferential mural thrombus, or extensive neck calcification. While concerns have
been raised regarding the risks of renal or mesenteric embolization, occlusion, and end-organ ischemia, observational studies have documented the efficacy and safety of suprarenal fixation. Rates of renal dysfunction appear to be equivalent for endografts that utilize nitinol or stainless steel transrenal stents and not significantly different than that observed with infrarenal fixation. Although suprarenal fixation may produce a higher incidence of small renal infarcts, in most patients these do not appear to be clinically significant. The presence of renal dysfunction after EVAR with suprarenal fixation is likely multifactorial and transient in most patients. Nonetheless, renal artery occlusion and infarctions have been reported in patients with pre-existing renal artery occlusive disease and, while infrequent, visceral dysfunction and celiac or mesenteric artery occlusion may occur secondary to suprarenal fixation. One report showed no difference in renal function between the two device types, whereas another study demonstrates a reduction in renal function after the use of a suprarenal fixation device. A recent meta-analysis examining the renal complications after standard EVAR with suprarenal and infrarenal fixation demonstrated no difference in renal complications.

Management of the internal iliac artery. Exclusion of the hypogastric artery (HA) to prevent a type II endoleak is usually required when the aneurysm involves either the distal common iliac artery or the HA itself. Several observational studies have revealed that unilateral embolization of the HA can be performed during EVAR with minimal adverse events as long as the contralateral HA is patent. Although ipsilateral buttock claudication and erectile dysfunction have been reported to occur in up to 40% of patients after unilateral HA embolization, these symptoms tend to improve and abate over time. 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 HA interruptions, whereas impotence occurred in 9% of unilateral and 13% of bilateral HA embolizations. In addition, the occurrence of these events is reduced if patency of the internal iliac artery bifurcation remains intact as illustrated in one small study using an Amplatzer vascular plug to
occlude only the main trunk of the HA. A more recent report demonstrates no clinical difference between coils and plug embolization. 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 to a staged approach.

Bilateral HA 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 and internal iliac arteries. Although antegrade flow into at least one of HA should be maintained, 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 HA interruption during EVAR may have been overestimated as several recent reports suggest that such devastating complications are exceedingly rare. The risks associated with bilateral HA occlusion are restricted to 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 a staged approach, embolization of only the proximal main trunk of the HA, and preservation of collateral branches from the common and deep femoral arteries. Alternative considerations to avoid bilateral HA embolization during EVAR include open or endovascular revascularization of at least one internal iliac artery. FDA approved iliac branch graft devices have been developed or under review to maintain ipsilateral internal iliac perfusion. These devices have displayed satisfactory early outcomes and should be considered prior to embolization in appropriate circumstances.

With the advent of endovascular repair techniques, the continued necessity of maintaining pelvic blood flow has been called into question. Several clinical series have used internal iliac artery embolization as an adjunct to extend the indications of EVAR in patients with aneurysms involving of the iliac bifurcation. Mehta and associates reported no mortality or increased morbidity in 48 patients who had interruption of both internal iliac arteries during open
or endovascular aortic repair.\textsuperscript{314} However, buttock claudication and new onset erectile
dysfunction were noted in 42\% and 14\% of the patients, respectively. The incidence of
postoperative sexual dysfunction and buttock claudication varies widely in the literature, ranging
from 16\% to 50\% for unilateral and 16\% to 80\% for bilateral internal iliac artery embolization,
underscoring the difficulty of causal association in the setting of significant co-morbidities
present in the older patient demographic at risk for AAA disease.\textsuperscript{323} Several endovascular
techniques have been described to preserve internal iliac artery flow, including the development
of commercially available aortoliac endografts that incorporate an iliac branch.\textsuperscript{324-327}

We recommend preservation of flow to at least one internal iliac artery.

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We recommend using FDA approved branch endograft devices in anatomically suitable patients
to maintain perfusion to at least one internal iliac artery.

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We recommend staging bilateral internal iliac artery occlusion by at least one to two weeks if
required for EVAR.

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Management of associated vascular disease. Co-existence of other vascular disease
with an AAA is common. Several series reporting observations of aortography have
documented greater than 50\% stenosis in 20-40\% of renal arteries, 10-15\% of celiac or superior
mesenteric branches, and 20-30\% of iliac vessels.\textsuperscript{328, 329}

The decision to intervene is based upon a consideration of severity of associated
lesions; presumed natural history of the diseased vessel and end organ; and anticipated
morbidity and mortality risk of combined repair. Prophylactic treatment of associated
asymptomatic renal or mesenteric artery disease cannot be justified.\textsuperscript{330, 331} The exception may
be the patient presenting with high grade stenosis of the superior mesenteric artery (SMA) and
a meandering mesenteric artery that is based off of a large inferior mesenteric artery (IMA),
which will be sacrificed during the course of treatment. A decision to repair each lesion should
be based upon its own individual merits and indications. If endovascular treatment is judged to
be beneficial, it is recommended that it be performed in a staged manner rather than
concomitantly with the planned EVAR procedure. Iliac and femoral artery lesions may be
treated at the time of EVAR to facilitate endograft delivery and to correct underlying pathology
that may be contributing to lower extremity ischemic symptoms.

Accessory renal arteries are present in 15 to 20\% of patients and occasionally may arise
from the aneurysm itself.\textsuperscript{328} Whether an accessory renal artery requires preservation is
dependent on the size of the artery, its contribution to the renal parenchyma, and the presence
of co-existing kidney disease. Renal infarction after occlusion of an accessory renal arteries is
common, occurring in 84\% of kidneys, but it is well tolerated in most patients, without significant
impact on long-term glomerular filtration rate.\textsuperscript{332} Nonetheless, preservation should be
considered for large accessory renal arteries (\geq 3 mm) or accessory renal arteries providing
more than one-third of arterial flow to the kidney, particularly in the presence of pre-existing
renal dysfunction.\textsuperscript{333, 334}

\textit{We suggest renal artery or superior mesenteric angioplasty and stenting for selected patients
with symptomatic disease prior to EVAR or OSR.}

Level of recommendation \hspace{1cm} Weak
Quality of evidence \hspace{1cm} Low
We suggest prophylactic treatment of an asymptomatic, high-grade stenosis of the SMA in the presence of a meandering mesenteric artery based off of a large IMA, which will be sacrificed during the course of treatment.

Level of recommendation     Weak
Quality of evidence     Low

We suggest preservation of accessory renal arteries at the time of EVAR or OSR if the artery is 3 mm or larger in diameter or supplies more than one-third of the renal parenchyma.

Level of recommendation     Weak
Quality of evidence     Low

Perioperative outcomes of elective EVAR

Incidence of 30-day and in-hospital mortality. The UK EVAR-1, Dutch DREAM, US Veterans Affairs OVER, and French ACE multicenter randomized trials collectively randomized 2,790 patients to EVAR or open repair. The two largest trials (EVAR-1 and OVER) demonstrated a statistically significant mortality benefit with EVAR and pooled analysis from all four trials confirmed the benefit of EVAR with a mortality of 1.4% as compared to 4.2% for open surgery (OR 0.3; 95%CI 0.22-0.50, p < 0.0001). A review of 79,932 Medicare patients confirmed that these results are representative of current outcomes with an overall mortality of 5.2% for open repair and 1.6% for EVAR (OR 3.2; 95%CI 2.95-3.51). Outcomes after AAA repair are related to experience. While earlier studies suggested that the minimum hospital threshold for optimal outcomes is 8 to 10 EVAR cases per year, a recent risk-adjusted analysis of 122,495 Medicare patients undergoing elective EVAR between 2001 and 2008 observed that operative mortality is directly related to medical center volume. The odds ratio for elective perioperative mortality adjusted for surgeon volume was lowest for centers that perform at least 30 EVAR cases per year.
We suggest that elective EVAR be performed at centers with a volume of at least 10 EVAR cases each year and a documented perioperative mortality and conversion rate to OSR of 2% or less.

Level of recommendation    Weak
Quality of evidence     Low

Perioperative morbidity. Estimated blood loss is significantly lower with EVAR than open repair. While major complications were not different in randomized controlled trials, in a review of Medicare patients, most major complications were lower with EVAR including pneumonia (3.8% vs. 12.9%, p < 0.001), acute renal failure (4.3% vs. 11.3%, p < 0.001), MI (2.5% vs. 5.2%, p < 0.001), and bowel ischemia (0.6% vs. 2.1%, p < 0.001). EVAR patients were also more likely to be discharged to home rather than to skilled nursing facility (95% vs. 83%, p < 0.001). The need to convert from EVAR to open repair decreased over time (2.2% in 2001 to 0.3% in 2008). Median length of stay was 2 days after EVAR as compared to 7 days after open repair (p < 0.001).

Endoleak. Type IA endoleak is noted in 6% of procedures at the time of implantation and may be due to mural thrombus, calcification, angulation, neck tapering, or excessive graft under- or over-sizing. Initial management is angioplasty with a compliant balloon, followed by extension cuff placement. Additional maneuvers include placement of a Palmaz® balloon expandable stent or endo-stapling. Conversion to open repair is not recommended unless rupture or significant, uncorrectable, device maldeployment is noted. A Type IA endoleak may occasionally resolve after reversal of heparin and no longer be evident on postoperative CT imaging. A persistent Type IA endoleak may be treated by placement of a fenestrated device, proximal cuff extension with chimney grafts to the renal arteries, external banding, embolization with coils or glue, or conversion to open surgery. Insufficient data exist to recommend a particular strategy. A Type IB endoleak is treated initially by repeat balloon
angioplasty and, as needed, by graft extension. Coil embolization of the hypogastric artery may be required when the graft is extended into the external iliac artery. A Type II endoleak is common at the time of implantation and is observed in 10 to 20% of patients at 1 month follow-up on CT imaging. A Type II endoleak is not treated at the time of implantation. A Type III endoleak is treated by angioplasty of component overlap sites or by placement of an additional conduit. A Type IV endoleak is self-limited and treatment is not required.

**Access site complications.** Early experience with EVAR using open femoral artery exposure were associated with a high rate of access site related complications (13%), including arterial dissection or perforation (1.4%), bleeding, hematoma, or false aneurysm (6.6%), arterial thrombosis (2.2%), embolization (1.1%), wound infection, skin necrosis, or lymphocele (1.4%), and amputation (0.1%). In a multicenter randomized control trial, percutaneous access was superior to open femoral artery excess with a shorter procedure time (107 vs. 141 min, \( p = 0.004 \)) and fewer access complications (6% vs. 10%, \( p = 0.005 \)) with a 96% technical success rate. A systematic review and a recent NSQIP review also demonstrated a high technical success rate, shorter operative time (135 vs. 152 min), shorter length of stay (1 vs. 2 days), and fewer wound complications (1 vs. 2.1%, \( p = 0.02 \)). Percutaneous access may not be appropriate for patients with small vessels, a high femoral artery bifurcation, or in the presence of calcification or a femoral aneurysm. In addition, a history of prior groin surgery with or without a vascular graft or patch and obesity may reduce success rates. Percutaneous access with large sheaths is improved by ultrasound guidance.

**Acute limb thrombosis.** Early graft limb thrombosis may occur in 2% of patients due to the placement of a large limb in a small vessel, iliac tortuosity with graft kinking, inadequate angioplasty or stenting, arterial dissection, or injury at the access site.

**Post-implantation syndrome.** A self-limited inflammatory state characterized by fever and elevated inflammatory markers may be observed after EVAR as a result of new thrombus formation within the excluded aneurysm sac.
**Ischemic colitis.** Colon ischemia due to occlusion of the inferior mesenteric or hypogastric arteries or embolization is rare after EVAR (< 1%).\textsuperscript{310, 340, 372, 378, 379} Circumflex femoral and circumflex iliac arteries should be preserved should hypogastric artery occlusion be planned. Suspected colonic ischemia should be assessed by endoscopy and, if confirmed, antibiotics administered and the patient maintained on intravenous fluids. Colectomy should be performed if full thickness necrosis is suspected.

**Role of elective EVAR in the high-risk and unfit patient**

The EVAR-2 trial compared EVAR to observation and found no benefit to EVAR for patients who were considered unfit for open repair due to a history of MI or cardiac revascularization, stable angina, valvular heart disease, significant arrhythmia, uncontrolled congestive heart failure, FEV\textsubscript{1} < 1 L, or serum creatinine > 2.3 mg/dL.\textsuperscript{285} However, of those randomized to EVAR only 179 of 197 (91%) underwent surgery; 14 deaths and 9 ruptures occurred prior to surgery, which occurred at a median of 55 days after randomization. Operative mortality for those randomized to EVAR was 8.4% (6.4% for elective repair). Of 207 patients randomized to observation, 70 (34%) crossed over to EVAR; 64 (31%) were repaired electively, with an operative mortality of 3%.

Among the many lessons learned from EVAR-2 and other data, it is evident there is a subgroup of patients who are not fit for open repair, which are also at high risk for EVAR. It is this subgroup that may be identified using the VQI risk model and should be considered for non-operative management. There may also be patients who while high risk for open surgery are reasonable risk for EVAR. Additionally, as noted in one third of patients randomized to observation in EVAR-2, fitness may be improved by optimization of co-morbid disease to the extent that EVAR may then be considered.
We suggest informing high-risk patients of their VQI perioperative mortality risk score in order to make an informed decision to proceed with aneurysm repair.

Level of recommendation    Weak
Quality of evidence     Low

Open surgical repair

Indications. Open surgical repair of a AAA continues to be used for patients who do not meet the anatomical requirements for endovascular repair, including short or angulated landing zones, excessive thrombus, multiple large accessory renal arteries, or small and tortuous access vessels with concomitant occlusive disease. However, fenestrated, branched and the use of chimney or snorkel grafts has expanded the range of complex aortic anatomy potentially treatable by EVAR. Open surgical repair may be required for treatment of a persistent endoleak and aneurysm sac growth after EVAR or for treatment of a mycotic aneurysm or infected graft.

Surgical approach. OSR can be performed using either a trans-peritoneal or left-flank retroperitoneal approach (Table 7). Indications for each type of approach are largely based on patient anatomy, comorbidities and surgeon preference. The trans-peritoneal approach is typically performed using a generous midline incision from the xyphoid process to the symphysis pubis. Extension of the incision alongside the xyphoid process releases rectus aponeurosis and facilitates exposure in the obese patient or in those with more proximal aortic disease. A mini-laparotomy (15 cm) has been used in select patients. A transperitoneal approach can be performed rapidly and is versatile, allowing assessment of intra-abdominal pathology and easy access to the visceral and iliac arteries. Transverse incisions just above the umbilicus also yield excellent exposure to the suprarenal aorta and bilateral iliac bifurcations. Proponents of the retroperitoneal approach claim various physiologic benefits, including significant reduction in fluid losses, cardiac stress, pulmonary complications, and ileus.
However, prospective randomized studies have generated conflicting results.\textsuperscript{380, 381} The measurable benefits attributed to retroperitoneal exposure were primarily a shorter duration of ileus and earlier resumption of oral intake. Sieunarme reported no difference in a randomized comparison of transperitoneal and retroperitoneal approaches for infrarenal AAA repair, except for higher rates of incisional pain, bulge and hernias in the retroperitoneal group.\textsuperscript{382}

The retroperitoneal approach may be preferred for repair of a suprarenal aneurysm because exposure can be facilitated by division of the left diaphragmatic crura. However, in a majority of patients repair of juxtarenal and pararenal aneurysms can be performed using a transperitoneal approach with excellent outcomes.\textsuperscript{383, 384} Although some surgeons routinely ligate or divide the left renal vein to expose the suprarenal aorta in the course of using a transperitoneal approach, others do not.\textsuperscript{383, 384} An important indication for the retroperitoneal approach is the presence of a “hostile abdomen” due to prior intra-abdominal operations, radiation, incisional hernia, stoma, enterocutaneous fistula. In addition, a retroperitoneal approach can facilitate repair of an inflammatory aneurysm or aneurysm associated with a horseshoe kidney.\textsuperscript{385, 386}

\begin{itemize}
\item We recommend a retroperitoneal approach for patients requiring open surgical repair of an inflammatory aneurysm, horseshoe kidney, or an aortic aneurysm in the presence of a hostile abdomen.
\item We suggest a retroperitoneal exposure or a transperitoneal approach with a transverse abdominal incision for patients with significant pulmonary disease requiring open surgical repair.
\end{itemize}

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Level of recommendation & Strong \\
Quality of evidence & Low \\
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Level of recommendation & Weak \\
Quality of evidence & Low \\
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Aortic clamping. Selection of the ideal clamp site and extent of reconstruction is based on analysis of cross-sectional aortic imaging including proximal aneurysm extension, iliac occlusive or aneurysmal disease, concomitant renal and mesenteric disease, anomalous venous anatomy, and presence of calcium, thrombus or atherosclerotic debris.

The location of the clamp site should take into consideration the proximal extension of the aneurysm, as well as the structural integrity of the aortic wall. Ideally, the clamp site should be relatively free of thrombus, atherosclerotic debris or calcification. Other important considerations include presence of concomitant visceral aortic disease and unusual venous anatomy, such as a retro-aortic renal vein or left-sided vena cava. The aortic clamp should be placed in the most caudal position possible to avoid unnecessary renal and visceral ischemia, while allowing a safe anastomosis into healthy aortic wall. For repair of an infrarenal aortic aneurysm, the clamp is placed immediately below the level of the lowest renal artery, while the graft is anastomosed to a rim of normal aortic wall below the level of the clamp. Performing the proximal anastomosis within healthy aorta is important to minimize the risk of aneurysmal degeneration at or above the graft.

The transperitoneal approach is typically performed using a midline incision from the xyphoid to the symphysis pubis or, in select cases, a mini-laparotomy incision. After placement of a self-retaining retractor, the transverse colon is retracted cephalad and the small bowel mesentery to the right side of the abdomen, splaying the retro-peritoneum and aortic aneurysm. The retroperitoneum is incised to the left side of the aorta, avoiding the IMA. The inferior mesenteric vein may need to be divided to avoid inadvertent traction injury or avulsion by fixed retractors used to assist in exposing the infrarenal aortic neck. The left renal vein may need to be mobilized and, if suprarenal clamp placement required, division of the gonadal, adrenal and lumbo-renal branches of the renal vein will facilitate its mobilization. Should division of the left renal vein be planned to optimize exposure of the aortic neck, the gonadal, adrenal, and lumbar branches should be preserved to provide collateral flow from the kidney. In the presence of
significant mural thrombus at the level of the aortic neck, isolation and temporary occlusion of
the renal arteries may be warranted to minimize the risk of renal artery embolization at the time
of clamp placement.

If the aneurysm extends above the renal arteries or significant aortic calcification is
present, it may be preferable to clamp the supraceliac aorta.\textsuperscript{387, 388} Patients in which an aortic
aneurysm required a suprarenal clamp have an increased risk of renal dysfunction and
morbidity, but similar 30 day mortality, as compared to those in which an infrarenal clamp site
was sufficient for repair an AAA.\textsuperscript{383, 384, 389} Visceral vessel control is not necessary as
backbleeding is minimal following supraceliac aortic crossclamp application.

The sequence of clamping should begin with the least diseased segment to avoid the
risk of distal embolization. Typically the iliac arteries are clamped first, followed by the proximal
aorta. Distal clamping is always at the level of the iliac arteries, since aneurysm disease usually
extends to the aortic bifurcation even in patients with planned reconstruction using a tube graft.
If the common iliac arteries are aneurysmal, the external iliac arteries need to be dissected and
controlled separately. The internal iliac arteries may require balloon occlusion, if external
clamping is not feasible.

Systemic anticoagulation with 100 U/kg of intravenous heparin is recommended for
elective aneurysm repair, irrespective of the location of the aortic clamp. Heparin administration
may be omitted or used in lower doses in special circumstances of a ruptured aneurysm or
other unusual situations. In these cases, the graft is vigorously flushed prior to restoration of
blood flow, or limited amounts of heparinized saline may be instilled directly into the distal
vessels after placement if the proximal aortic clamp. In patients with history of heparin-induced
thrombocytopenia, a thrombin inhibitor, such as Bivalirudin or Argatroban may be used as an
alternative.
We recommend a thrombin inhibitor, such as Bivalirudin or Argatroban as an alternative to heparin for patients with a history of heparin-induced thrombocytopenia.

Level of recommendation: Strong  
Quality of evidence: Moderate

Graft type and configuration. There is no significant difference in patency, durability, resistance to infection, or risk of degeneration or dilatation of currently used prosthetic materials. Differences in methods of fabrication of polyester grafts include knitted or woven, external or double velour, high or low porosity. Polyester grafts can be rendered impermeable by various biologic coatings, including collagen, gelatin or albumin. Graft impregnation with silver or Rifampin has been used to enhance resistance to infection. Routine use of Rifampin impregnated polyester grafts in which used gelatin-coated polyester grafts are soaked in Rifampin solution (1 mg/mL) for 15 to 30 minutes or silver impregnated grafts to limit the risk of device associated infection has not proven to be beneficial in prospective or multicenter studies. Graft configuration can be a straight tube or bifurcated. The location of the distal anastomosis is at the aortic bifurcation, iliac or femoral artery. A tube graft is preferable when feasible due to shortened operative time, reduced blood loss and need for dissection, minimizing risk of inadvertent injury to the ureter, iliac veins, and autonomic nerves. In the era prior to widespread adoption of EVAR, approximately 40% to 50% of patients could be treated with a tube graft. In the Canadian Aneurysm Trial, graft configuration was straight in 39%, aorto-bi-iliac in 31%, aortobifemoral in 24%, and aorto-iliac and femoral in 7%. Bifurcated grafts are indicated when the distal aorta and common iliac arteries are aneurysmal, which occurs in one third of patients. In the presence of iliac aneurysmal disease, the distal anastomosis should be performed immediately proximal to the iliac bifurcation to reduce the risk...
of late aneurysmal degeneration. Patients with symptomatic aortoiliac occlusive disease may benefit from distal graft anastomosis to the distal external iliac or common femoral arteries. However, graft extension to the femoral arteries increases the risk of wound infection, limb thrombosis, and anastomotic aneurysm formation. Assuming normal iliac arteries have been selected for the distal anastomosis, the risk of progressive distal aneurysmal or occlusive iliac disease is relatively low.\textsuperscript{394, 397}

We recommend straight tube grafts for open surgical repair of AAA in the absence of significant disease of the iliac arteries.

Level of recommendation: Strong
Quality of evidence: High

We recommend performing the proximal aortic anastomosis as close to the renal arteries as possible.

Level of recommendation: Strong
Quality of evidence: High

We recommend that all portions of an aortic graft 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.** Perfusion of the colon, rectum and pelvis is provided by a complex collateral network from the superior mesenteric artery (SMA) and IMA via the marginal artery of Drummond, the internal iliac arteries, and additional collaterals from the circumflex iliac, common and deep femoral arteries. Inadequate pelvic circulation can lead to sexual dysfunction, as well as hip and buttock claudication. Less frequently, colon or spinal ischemia may ensue. For example, in the Canadian Aneurysm Study, the risk of colon ischemia increased eight-fold (0.3% to 2.6%) when both internal iliac arteries were occluded compared to
when at least one of the internal iliac arteries was preserved.\textsuperscript{395, 396} Thus, all efforts should be made to preserve perfusion to at least one internal iliac artery.

Colonic ischemia following aortic repair is multifactorial in origin, but ligation of a patent IMA during reconstruction remains a risk factor.\textsuperscript{398} The IMA is occluded in 40\% to 50\% of the patients with aortic disease because of ostial atherosclerosis or mural thrombus. The value of routine re-implantation of a patent IMA has not been established, but selective re-implantation may be of value in the presence of compromised pelvic perfusion, particularly when the marginal artery is interrupted due to prior colectomy.\textsuperscript{379, 399} A prospective randomized study suggested that IMA re-implantation is beneficial in patients of advanced age and when intra-operative blood loss has been substantial.\textsuperscript{400} Likewise, re-implantation of the IMA should be considered in patients with underlying celiac and SMA occlusive disease, particularly in the presence of a large meandering artery. The IMA can be easily controlled prior to opening the aneurysm using a vessel loop and reimplantation to an aortic graft performed using a Carrel patch technique, in which a small button of aortic wall is dissected free from adherent thrombus and calcific debris. In the Canadian Aneurysm Trial, IMA re-implantation was used in 5\% of the patients, but was associated with increased risk of postoperative bleeding.\textsuperscript{395} Preservation of antegrade flow into at least one of the internal iliac arteries is recommended, whenever possible. For patients treated by open surgical repair this can be usually achieved by distal anastomosis to the iliac bifurcation, end-to-side-anastomosis at the external iliac artery with retrograde flow into the internal iliac artery, or a separate bypass graft to the internal iliac artery.

\textit{We recommend reimplantation of a patent inferior mesenteric artery (IMA) under circumstances that suggest an increased risk of colonic ischemia.}

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Level of recommendation & Strong \\
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Quality of evidence & High \\
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We recommend preserving blood flow to at least one hypogastric artery in the course of OSR.

Level of recommendation: Strong
Quality of evidence: High

Management of associated intra-abdominal vascular disease. Occlusive disease of the celiac and SMA is present in 10% of patients, whereas renal artery disease may occur in up to 40%. Since the morbidity and mortality of aortic repair is increased by concomitant renal or mesenteric reconstruction, such procedures are only indicated in the presence of symptomatic disease.

Pearce and colleagues reported a 30-day mortality of 3% among 678 patients treated for abdominal aortic aneurysms with concomitant renal artery reconstruction. However, mesenteric artery reconstruction combined with aortic reconstruction carries higher mortality rate and should be avoided unless clinically indicated. Thus, should open AAA repair be required in the presence of renal or mesenteric artery disease, a staged approach with initial stenting should be pursued.

We suggest concomitant surgical treatment of other visceral arterial disease at the time of open surgical repair in symptomatic patients who are not candidates for catheter-based intervention.

Level of recommendation: Weak
Quality of evidence: Moderate

Management of associated intra-abdominal nonvascular disease. In the occurrence of an AAA and associated intra-abdominal pathology, the most life-threatening condition should be treated first. Simultaneous repair is avoided because of added morbidity and, in the case of genitourinary or gastrointestinal procedures, the risk of bacterial contamination of the prosthesis.
Cholelithiasis is the most common abdominal pathology with a prevalence of 5% to 20%. Asymptomatic cholelithiasis should be left untreated since the risk of acute cholecystitis after elective AAA repair is <1%. In the presence of a large aneurysm, treatment of a colorectal tumor takes precedence in the presence of impending obstruction, bleeding or perforation. Otherwise, colon resection should be delayed for four to six weeks after AAA repair. Simultaneous resection of ovarian or renal tumors may be considered if a staged minimally invasive treatment is not feasible.

We suggest concomitant surgical repair of an AAA and co-existent cholecystitis or an intraabdominal tumor in patients who are not candidates for EVAR or staged intervention.

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Perioperative outcomes after open AAA repair. Factors affecting mortality of OSR include surgeon and hospital volume, urgency of the procedure, patient age, presence and severity of comorbidities and proximal aneurysm extension. Presence of symptomatic coronary artery disease, congestive heart failure, severe chronic pulmonary disease and advanced chronic kidney disease remain the most important predictors of mortality. There has been considerable variation in 30-day mortality rates in the literature, depending on the type of study reported and its design. Elective 30-day mortality for infrarenal AAA OSR in most contemporary large single-center institutional reports has ranged from 1% to 4%. Population-based studies, derived from state and national databases, indicate higher mortality rates of 4% to 8% across the entire spectrum of hospitals and health care organizations. Recent multicenter, prospective, randomized trials have demonstrated 30-day mortality of 3% to 4.7%. Analyses of outcomes in Medicare beneficiaries indicates that OSR mortality, while improved in the last decade, remains higher than that associated with
EVAR for every age category. Similarly, the morbidity of OSR is significantly higher compared to EVAR, particularly cardiac, pulmonary, renal, gastrointestinal and wound-related complications (Table 8). Finally, recent studies have shown that over 20% of the patients treated by OSR require reoperations for laparotomy-related complications within 8-years.

The impact of individual surgeon and hospital volume on outcomes of AAA OSR has been documented in several studies. A review of national Medicare claims by Birkmeyer indicated that 30-day mortality was 8% for low volume hospitals (<17/year) compared to 4.4% in high volume hospitals (>79/year). Surgeon volume and prior dedicated vascular training also affect mortality of OSR. Dimick reported that elective AAA mortality was lowest when operations were performed by vascular surgeons (2.2%), as compared to cardiac surgeons (4%) and general surgeons (5.5%) (\(p < 0.001\)). Using a risk-adjusted analysis, high hospital volume, vascular surgery specialty, and high surgeon volume were independent predictors for lower risk of in-hospital mortality after elective AAA repair. In that study, absolute risk reduction for operations performed in high-volume hospitals and high-volume surgeons was 30% and 40%, respectively. AAA repair performed by general surgeon increased risk of death by 76% compared to repair performed by vascular surgeon. A recent risk-adjusted analysis of 122,495 Medicare patients undergoing elective AAA repair between 2001 and 2008 notes that the mortality for open surgical repair is directly related to medical center volume. The odds ratio for elective perioperative mortality was lowest for centers that perform at least 18 open repairs and was less than 5%.

Accurate assessment of open surgical expertise and the applicability of outcome data acquired in the pre-endovascular era will be areas of concern as the volume of OSR continues to decline in the United States with anticipated reduction in the prevalence of cigarette smoking and expanding options for complex endovascular aneurysm repair. Care should be taken in extrapolating current outcomes for OSR from data obtained before the widespread availability of endovascular devices.
We suggest that elective OSR for AAA be performed at centers with an annual volume of at least 10 open aortic operations of any type and a documented perioperative mortality of 5% or less.

Level of recommendation    Weak
Quality of evidence     Low

The patient with a ruptured aneurysm

Preoperative management and considerations for patient transfer. A ruptured abdominal aortic aneurysm represents a true surgical emergency. Documented rupture, particularly with associated hypotension, demands immediate transfer to an adequately equipped operating room for definitive repair without delay. Should aneurysm rupture occur, more than half of patients die prior to hospitalization or without treatment.

Establishing a protocol or algorithm for urgent or emergent management of a patient with a ruptured abdominal aortic aneurysm is essential for optimizing outcomes. In the presence of a protocol, 30-day mortality was 18%, whereas in the absence of a protocol, 30-day mortality was 32%. Based on review of the literature, including existing guidelines endorsed in the United Kingdom and by the Western Vascular Society, an algorithm for the initial evaluation, diagnosis, immediate management, and triage of patients with a suspected ruptured abdominal aortic aneurysm is presented (Figure 5). An expedited evaluation consisting of the airway, breathing, and circulation (ABC) protocol, general assessment, and vital sign check should be initially performed by the emergency physician of any patient suspected of having a ruptured abdominal aortic aneurysm. Diagnosis in the emergency department is usually ascertained based on history and physical examination. Radiologic confirmation, either via bedside ultrasound imaging or a contrast-enhanced CT scan, can be obtained when an alternative diagnosis is more likely on clinical grounds. Optimization of the patients’ clinical condition in the pre-operative setting, while waiting for urgent transport to an operating room, may improve
outcomes. Intravenous access should be established with two large bore peripheral intravenous lines, as central or arterial access is not immediately necessary. Permissive hypotension, or “hypotensive hemostasis,” which refers to restricting aggressive fluid resuscitation as long as the patient remains conscious and has a systolic blood pressure between 70 to 90 mm Hg, should be implemented to limit excessive hemorrhage.\textsuperscript{424-426} Laboratory or imaging studies should only be obtained to confirm the diagnosis of ruptured abdominal aortic aneurysm. Other actions that may help improve outcomes are the immediate availability of blood and blood products, warming, and the avoidance of elective intubation.\textsuperscript{427} With the increasing utilization of endovascular methods to treat patients presenting with a ruptured abdominal aortic aneurysm, vital resources, including advanced imaging, trained staff, and robust endovascular inventory must be available. In cases where transfer is not necessary, the vascular team should be notified as soon as a ruptured abdominal aortic aneurysm is suspected. It may be prudent, however, to transfer a patient to a higher level facility when such resources are unavailable.\textsuperscript{428, 429} Patients with good functional status and without severe co-morbidity should be transferred without delay. Further, patients previously declined elective surgery should be considered for transfer and treatment. Some patients experiencing a ruptured abdominal aortic aneurysm may not be medically fit to undergo open repair, while at the same time are not anatomically suitable for endovascular repair. The urge to offer endovascular repair to patients anatomically unsuitable for such repair should be strongly resisted.\textsuperscript{430} Pre-operative predictors of death after open repair include age > 76 years, serum creatinine of > 2.0 g/dL, pH < 7.2, and blood pressure < 70 mmHg at any time. While these risk factors require more robust validation, when all four are present, open repair is uniformly fatal.\textsuperscript{431} As such, goals of care, medical comorbidities, and hemodynamics should be discussed with the receiving vascular surgeon if transfer is necessary. Ongoing cardiac arrest represents a contraindication for transfer given the unlikely survival of these patients.
Direct physician-to-physician phone handoff is necessary for all patients being transferred. It is imperative that relevant imaging is transmitted, preferably using an electronic method. Little data exist to guide best management during this critical transfer time period. Patients should receive intravenous nitroglycerin, esmolol, sodium nitroprusside, and pain medication, as needed, to avoid hypertension and minimize the risk of uncontained rupture. Permissive hypotension is appropriate with limited resuscitation and should be maintained during transfer. Blood products are preferred to treat hypotension, but transfer should not be delayed if blood products are not readily available.

**Systems of care and time goals for intervention.** Timeliness of intervention for the patient with a ruptured abdominal aortic aneurysm impacts outcomes. A goal of *door-to-intervention time of less than 90 minutes* is recommended with time zero defined as the time of first medical contact and intervention defined as initial arterial access and placement of an aortic occlusion balloon (Figure 5). Given limited studies benchmarking time to intervention for a ruptured abdominal aortic aneurysm, this goal has been proposed based on those established by the 2004 American College of Cardiology (ACC) Foundation/American Heart Association guidelines for the management of ST-segment-elevation myocardial infarction (STEMI). For the patient necessitating transfer to a regional center, the adoption of a 30-30-30-minute framework is recommended as a benchmark. The initial period denotes the time from *first medical contact* with a patient suspected of having a ruptured aortic aneurysm, including immediate management, to the point when a decision is made to transfer the patient to a regional center if so required or the initiation of emergent in-house vascular surgery evaluation. The second period represents the time required for rapid transfer to a regional center, if needed, and includes, physician-physician phone handoff, transfer of images, if available, and in-transit care. The final period includes the time from evaluation by the in-house or receiving vascular surgery team to arterial access and placement of an aortic occlusion balloon. Checklists, such as those highlighting the essential tasks needed in order to facilitate
transfer, as well as those that assist in coordinating care teams at the treating facility can be used to help meet these goals (Figures 6, 7). More importantly, and similar to STEMI management, the establishment of systems of care will be necessary. With an organized regional transfer system, operative repair can be performed in over 95% of patients with a ruptured abdominal aortic aneurysm, with 67% survival. This requires effective coordination between established sending and receiving facilities with standardized communication, a reliable transport provider, patient management guidelines during transfer, and a streamlined process for operative repair. This goal should be considered the longest times acceptable for effective management of a patient with a ruptured aortic aneurysm and systems that are able to achieve even more rapid times should be encouraged.

It should be noted that the benchmark of < 90 minute door-to-balloon time for the management of the patient with a STEMI was initially extremely challenging to meet. In 2004, the National Cardiovascular Data Registry reported that for patients requiring interhospital transfer, only 8.6% had total door-to-balloon times of less than 90 minutes with a median time of 152 minutes. Nonetheless, the establishment of an ambitious time goal promoted the development of STEMI Systems of Care to decrease time to intervention and improve overall patient survival. The challenge faced by rural centers was recognized when the 2004 AHA/ACC guidelines were issued. However, with rapid triage, transfer, and STEMI treatment programs, median total door-to-balloon times have been driven down for rural hospitals from a median of 189 minutes to 88 minutes.

We suggest a door-to-intervention time of < 90 minutes, based on a 30-30-30 minute framework, for the management of the patient with a ruptured aneurysm.

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An established protocol for the management of ruptured AAA is essential for optimal outcomes.

We recommend implementing hypotensive hemostasis with restriction of fluid resuscitation in the conscious patient.

We suggest that patients with ruptured AAA who require transfer for repair, be referred to a facility with an established rupture protocol and suitable endovascular resources.

Initial operative management. Regardless of the nature of repair, proximal control of the aorta is a crucial aspect of the initial part of the procedure. Indications for an aortic occlusion balloon include circulatory collapse, hemodynamic instability, or anatomic limitations that prevent expeditious repair. A femoral artery approach with use of a long sheath is preferred over a brachial approach. The sheath may be advanced into the supraceliac aorta to support the balloon and permit its removal after endograft placement.

In the hemodynamically unstable patient without preoperative CT imaging, evaluation of the proximal and distal sealing zones and device selection can be based on intraoperative angiography recognizing the inability to assess the extent of mural thrombus, or ideally, by intravascular ultrasound. Both bifurcated and aortouniliac endografts have been used for emergent EVAR. While used less commonly, an aortouniliac device may be helpful in the treatment of an anatomically challenging AAA. The Nellix® Endovascular Aneurysm Sealing System (EVAS, Endologix, Inc.) has also been proposed for treatment of ruptured AAA.
Role of EVAR. 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. An early randomized trial comparing EVAR and OSR for ruptured AAAs revealed that the suitability for endovascular repair was only 46%, but the rate of EVAR was lower (30%). 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 confound these analyses. The IMPROVE Trial was a multicenter randomized trial of EVAR and open repair for patients presenting with a ruptured AAA. Patients were randomized prior to obtaining CT imaging with 316 patients randomized to EVAR and 297 patients to open repair. Thirty-day mortality was similar among patients treated with EVAR (35.4%) or open repair (37.4%). Secondary analyses demonstrated shorter length of stay and a higher proportion of patients discharged to home for those treated by EVAR. A potential limitation of this study was the application of an intent-to-treat analysis, which incorporated outcomes for those participants initially randomized to EVAR but whose anatomy required open repair to the EVAR group. Recently reported one-year outcomes demonstrated that EVAR was most cost effective when compared to open repair, but no survival benefit was observed. An analysis of national trends in the United States confirm that EVAR is being used with increasing frequency for the treatment ruptured AAA, with a decrease in associated mortality. Outcomes are superior when EVAR for a ruptured aneurysm is performed in teaching hospitals and high volume centers.

If anatomically feasible, we recommend EVAR over open repair for treatment of a ruptured AAA.

Level of recommendation: Strong
Quality of evidence: Moderate
Management of postoperative complications

**Abdominal compartment syndrome.** Abdominal compartment syndrome is a well-recognized complication after both open surgical repair and EVAR for ruptured aneurysm and may occur in approximately 7% of patients. Use of an aortic occlusion balloon, coagulopathy, massive transfusion, and conversion to an aortouniliac device, are all predictors of abdominal compartment syndrome. Abdominal compartment syndrome typically occurs in the hemodynamically unstable patient with a large retroperitoneal hematoma. Diffuse visceral edema results in intra-abdominal hypertension and multiple system organ dysfunction, including oliguria, increased peak airway pressures, hypoxemia, hypercarbia, hypotension and decreased cardiac output. Early recognition and surgical decompression are necessary to improve survival.

**Ischemic colitis.** The incidence of colonic ischemia following repair of a ruptured AAA may occur in as many as one-in-five to one-in-three patients. Ischemic colitis following vascular surgery has been associated with mortality rates of 45-67% with recent reports demonstrating only modest improvement in outcomes. Delayed diagnosis with advanced ischemic colitis leading to perforation is associated with a mortality rate in excess of 90%, and a retrospective review of 222 patients revealed that ischemic colitis was the most common cause of death after open repair of a ruptured aneurysm. Colonic ischemia is much less frequent after EVAR than OSR for ruptured aneurysm (23% vs. 42%), but the risk remains. Prompt endoscopy is recommended when there is a suspicion for ischemic colitis to confirm the diagnosis and help guide management.

**Multi-system organ failure.** Given the associated hemodynamic instability and ischemia-reperfusion injury among patients presenting with a ruptured aneurysm, multisystem organ failure (MSOF) may occur in 1% to 3% of patients after EVAR or open repair. Once MSOF develops, organ dysfunction leads to a prolonged ICU stay, high resource
consumption, and a 50-70% mortality rate.\textsuperscript{463} Dedicated ICU teams and regionalized care at high-volume centers have led to reduced mortality and decreased length of stay.\textsuperscript{464, 465}

**Special considerations**

**Inflammatory aneurysm.** An inflammatory aortic aneurysm occurs in between 5 to 10% of patients.\textsuperscript{466} An inflammatory aortic aneurysm may not be readily apparent on CT imaging, but may be associated with retroperitoneal fibrosis\textsuperscript{467, 468} and displays a similar natural history to the more common degenerative aortic aneurysm.\textsuperscript{469, 470} An inflammatory aortic aneurysm is typically adherent to the duodenum, and less commonly, the ureters, renal vein and inferior vena cava.\textsuperscript{471} Should open repair be required, a retroperitoneal approach is recommended to avoid dissection of the duodenum. A systematic review of 999 patients with inflammatory aortic aneurysm confirmed that EVAR was associated with decreased mortality as compared to open repair.\textsuperscript{472-476} \textsuperscript{477}

**Horseshoe kidney.** A horseshoe kidney occurs in 0.25% of the general population and in 0.12% of patients presenting with an aortic aneurysm.\textsuperscript{478} Preoperative evaluation requires careful determination of the renal arterial anatomy, which can be highly variable with accessory renal arteries originating from the aorta, aneurysm sac and iliac arteries.\textsuperscript{479-481} If the main renal arteries are located proximal to the aneurysm, EVAR can be safely performed.\textsuperscript{482, 483} Small accessory renal arteries may be covered by EVAR.\textsuperscript{484, 485} In cases with anomalous blood supply, open surgical repair, preferably through a retroperitoneal approach, hybrid repair, or a fenestrated-branched EVAR may be considered.\textsuperscript{486, 487, 488} Multiple renal arteries can be surgically re-implanted using a Carrel patch or through an inclusion technique.\textsuperscript{489} Should open repair be required and CT imaging reveal that the horseshoe kidney is associated with a very thin fibrous isthmus, a trans-peritoneal approach may be considered with division of the “isthmus”.\textsuperscript{490-492} A hybrid approach has been described using a bifurcated Dacron graft based
off the external iliac artery to revascularize the horseshoe kidney, followed by EVAR. Likewise, repair has also been described using a fenestrated endograft and snorkel grafts.

### Aortocaval fistula
A ruptured aneurysm associated with an aortocaval fistula has been reported in 0.22% to 6% of patients. The triad of abdominal pain, a pulsatile mass, and an abdominal ‘machinery’ bruit is present in up to 80% of cases. Patients presenting with an abdominal aneurysm and high-output heart failure or a paradoxical pulmonary embolism should also be suspected of having an aortocaval fistula. Duplex imaging will reveal an arterial flow pattern in the inferior vena cava and CT imaging will demonstrate contrast in the inferior vena cava during the arterial phase. EVAR is preferred with expected resolution of preoperative heart failure and other physiologic disturbances. If open repair is required, venous bleeding should be anticipated and care taken to minimize the risk of pulmonary air embolism or embolism of thrombotic debris by placement of sponge sticks proximal and distal to the aortocaval fistula for control followed by direct suture repair of the defect.

Since 2013, an additional 53 patients presenting with an aortocaval fistula have been added to the previously reported 250 cases. The majority have been successfully treated with EVAR alone, with occasional use of an Amplatzer® plug or additional placement of a covered stent in the inferior vena cava.

### ANESTHETIC CONSIDERATIONS AND PERIOPERATIVE MANAGEMENT

#### Choice of anesthetic technique and agent
Open aneurysm repair requires general anesthesia, except in unusual circumstances, because of the required relaxation of the abdominal wall musculature and need for wide exposure of the aorta and its branches. 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. In addition, it has been postulated,
but not proven that there is decreased hemodynamic lability and cardiac ischemia.\textsuperscript{532, 533}

Nonetheless, it has been difficult to demonstrate significant benefit to either intraoperative or
postoperative epidural anesthesia and traumatic preoperative insertion of an epidural catheter
with blood tinged cerebrospinal fluid may preclude subsequent heparin administration and
require cancellation of the operative procedure. Use of epidural anesthesia with low dose
inhalation anesthesia or in the awake patient has been advocated for patients with severe
chronic obstructive pulmonary disease.\textsuperscript{534-536}

EVAR can be safely performed under general, epidural, or local anesthesia. While a
number of retrospective studies suggest that the type of anesthetic influences operative time,
length of hospital stay, and risk of morbidity, a mortality benefit has yet to be identified. In a
retrospective analysis of nearly 4,000 patients in the EUROSTAR registry, local anesthesia was
associated with shorter operative times, reduced ICU admission, shorter hospital stay, and
fewer systemic complications.\textsuperscript{537} There was a modest advantage of epidural anesthesia as
compared to general anesthesia. A recent review of the ACS NSQIP database demonstrated
that general anesthesia was associated with longer hospital stay and increased pulmonary
morbidity as compared to local or regional anesthesia.\textsuperscript{538} A meta-analysis of 13,459 patients
undergoing EVAR, revealed that patients undergoing local anesthesia were older and had more
severe cardiac and pulmonary disease, but experienced shorter operative times and hospital
stays and suffered fewer complications.\textsuperscript{539} A limitation of this review was the inability to account
for aneurysm anatomy and morphologic complexity, which may have influenced the selection of
general anesthesia for repair.

\textit{We recommend general endotracheal anesthesia for patients undergoing open aneurysm
repair.}

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Anesthetic considerations in the patient with a ruptured aneurysm. Regardless of the choice of endovascular or open surgical repair, there is evidence to support the implementation of a standardized protocol for the efficient evaluation and treatment of ruptured aneurysm, including anesthetic management.\textsuperscript{427, 446} Notably, the surgical field should be initially draped and a transfemoral aortic balloon placed, especially if general anesthesia is required due to the likelihood of vasodilatation, hypotension and cardiovascular collapse. Permissive hypotension to maintain a systolic blood pressure of 80 mmHg limits volume overload and appears sufficient to maintain critical end organ perfusion.\textsuperscript{540} The use of local anesthesia for EVAR, most often, does not provide sufficient pain control for the patient experiencing significant abdominal or back pain.

Antibiotic prophylaxis. A Cochrane review confirms that prophylactic antibiotics, administered prior to incision, reduces the risk of wound infection and early graft infection in arterial reconstructive surgery.\textsuperscript{541} However, continuing antibiotics for more than 24 hours postoperatively was without added benefit. There was no advantage among first- or second-generation cephalosporins, penicillins with lactamase inhibitors, aminoglycosides, or vancomycin. We also recommend that any potential sources of dental sepsis be eliminated at least 2 weeks before implantation an aortic prosthesis.

\textit{We recommend intravenous administration of a first generation cephalosporin or, in the event of penicillin allergy, vancomycin, within 30 minutes prior to open surgical repair or EVAR. Prophylactic antibiotics should be continued for no more than 24 hours.}

\begin{tabular}{ll}
2002 & Level of recommendation \hspace{1cm} Strong \\
2003 & Quality of evidence \hspace{1cm} High \\
2004 & \textit{We recommend that any potential sources of dental sepsis be eliminated at least 2 weeks before implantation an aortic prosthesis.} \\
2005 & Level of recommendation \hspace{1cm} Good Practice Statement \\
2009 & Quality of evidence \hspace{1cm} Ungraded \\
2010 & \\
2011 & \\
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Intraoperative fluid resuscitation and blood conservation. Allogeneic blood transfusion remains associated with immunological and infectious risks. Although preoperative autologous blood donation avoids disease transmission and transfusion reaction, as well as stimulates erythropoiesis, limitations include limited availability of blood donation, increased expense, and potential waste of non-utilized blood. Intraoperative cell salvage assists in blood conservation, has been recommended if large blood loss is anticipated, and may be helpful where concerns of the safety of banked blood exist. A prospective randomized trial of cell salvage in elective cardiac surgery did not lead to a reduction in exposure to allogeneic blood, but did reduce the number of transfused units. Various methods of cell salvage, including use of a cell saver or hemofiltration, have not been associated with meaningful differences in clinical outcomes. A Cochrane review of cell salvage used in a variety of surgeries demonstrated an overall reduction of less than 1 unit per patient with decreased likelihood of requiring an allogeneic blood transfusion, but no alteration in clinical outcome. A meta-analysis of cell salvage in open aneurysm repair, confirmed a reduced requirement for blood transfusion. In addition, one retrospective study has demonstrated that cell salvage was associated with improved survival among patients undergoing open repair of a ruptured aneurysm. Cell salvage is contraindicated in the presence of infection or malignancy.

The benefit of maintaining a predefined hematocrit level during open surgical repair of aortic aneurysm is unknown, but preemptive transfusion in the setting of rapid ongoing blood loss is well-supported. In the trauma literature, plasma, platelets, and packed red blood cells in a 1:1:1 ratio and warm fresh whole blood instead of component therapy have each been advocated. However, retrospective studies have not consistently demonstrated a survival benefit to a lower ratio of red cell transfusion to plasma transfusion for patients undergoing open repair of a rupture aneurysm. Further, withholding plasma and platelet transfusion until surgical repair is complete, is not supported by clinical evidence. Optimal blood replacement therapy during open repair has not been well defined nor indications established.
for administration of cryoprecipitate, plasma, and platelets. A retrospective study has suggested that administration of recombinant factor VIIa in the setting of intractable intraoperative and postoperative bleeding during vascular surgery has a survival benefit. A Cochrane review of perioperative administration of crystalloid and colloid fluids for open abdominal aortic surgery did not identify a superior regimen. However, a recent prospective randomized study of elective open repair of abdominal aortic aneurysm concluded that a more restrictive perioperative fluid regimen reduces complications and length of hospital stay.

We recommend using cell salvage or an ultrafiltration device if large blood loss is anticipated.

If the intraoperative hemoglobin is less than 10 gm/dL and blood loss is ongoing, we recommend transfusion of packed blood cells along with fresh frozen plasma and platelets in a ratio of 1:1:1.

Cardiovascular monitoring. Central venous pressure and arterial line monitoring are suggested for all patients undergoing open surgical repair of an aortic aneurysm. However, multiple randomized trials have shown no measureable benefit to the routine use of pulmonary artery catheters in non-selected patients. While trans-esophageal echocardiography (TEE) is useful for those patients ‘at risk’ of major hemodynamic instability or in the unstable patient to assess volume status and cardiac function, routine use does not influence clinical outcomes. Perioperative myocardial infarction is associated with adverse short- and long-term outcomes and can be prevented by early recognition of myocardial ischemia. ECG monitoring, utilizing five leads, is recommended for both OSR and EVAR. Continuous 12-lead
ECG or the monitoring of two leads instead of a single precordial lead has been shown to be a more sensitive indicator of myocardial ischemia. However, myocardial ischemia detection by TEE in the form of wall motion abnormalities precedes ST segment changes and is a more sensitive monitor for ischemia. While troponin measurement after vascular surgery has been advocated, routine measurement has not been associated with improved clinical outcomes.

We suggest using pulmonary artery catheters only if the likelihood for a major hemodynamic disturbance was high.

We recommend central venous access and arterial line monitoring in all patients undergoing open aneurysm repair.

We recommend postoperative ST-segment monitoring for all patients undergoing open aneurysm repair and for those patients undergoing EVAR who are at high cardiac risk.

We recommend postoperative troponin measurement for all patients with ECG changes or chest pain after aneurysm repair.

Maintenance of body temperature. Maintenance of body temperature above 36°C during aneurysm repair appears to be beneficial with respect to hemodynamics, laboratory measures of clotting function, and metabolic acidosis. Prospective randomized data supports the use of forced air warming blankets rather than circulating water mattresses.
addition, prospective studies support the use of low fresh flow rate anesthetic gases and the use of intravenous fluid and blood warmers to maintain normothermia.

We recommend maintaining core body temperature at or above 36°C during aneurysm repair.

Levels of recommendation: Strong
Quality of evidence: High

Role of the intensive care unit. Increasingly, the care of the postoperative patient is occurring in a step down unit or other monitored settings to best focus use of the intensive care unit on those patients in greatest need. Selective use of the intensive care unit after aneurysm surgery is most effective if preoperative criteria, such as preexisting significant coronary artery, pulmonary or renal disease, or intraoperative criteria, such as a significant arrhythmia, hemodynamic instability or requirement for postoperative mechanical ventilation are established. In a study of 230 patients undergoing aneurysm repair, 89% avoided admission to ICU by use of systematic preoperative evaluation to identify predictors of poor outcome.

Goal-directed therapy using non-invasive monitoring of cardiac output by esophageal Doppler or lithium indicator dilution and pulse power analysis (LiDCO) has been shown to improve short term outcomes. Monitoring cardiac output with a defined treatment protocol has also been shown to be cost effective in the setting of major abdominal surgery. While studies focused on the care of patients with an aortic aneurysm have not been conducted, randomized trials have included patients with vascular disease.

Fast track surgical pathways or ‘enhanced recovery’ pathways are being used increasingly to decrease length of stay and expedite discharge after abdominal surgery. Evaluation of a fast track surgery pathway in a 30 patient cohort undergoing open aneurysm repair was associated with an average length of stay of 3.6 days without readmission. The pathway included a limited retroperitoneal incision and specialized intraoperative retractors.
A recent trial confirmed benefit in 101 patients randomized to a fast track surgery care pathway, which included no bowel prep, reduced fasting, patient controlled anesthesia, as well as early mobilization and feeding. There was no difference in ICU length of stay, but time to full feeding (5 vs. 7 days, \( p < 0.001 \)) was reduced along with the incidence of postoperative complication (16% vs. 36%, \( p = 0.039 \)).

We recommend postoperative management in an intensive care unit for the patient with significant cardiac, pulmonary or renal disease, as well as for those requiring postoperative mechanical ventilation or who developed a significant arrhythmia or hemodynamic instability during operative treatment.

Level of recommendation Strong
Quality of evidence High

**Nasogastric decompression and perioperative nutrition.** Routine nasogastric decompression is not recommended after aortic surgery. A Cochrane review examined 37 studies involving 5,711 patients randomized to routine or selective nasogastric decompression after emergency or elective abdominal surgery.\(^{583}\) Selective decompression was associated with a decreased risk of pulmonary complications without untoward adverse effects.\(^{583}\) Although postoperative malnutrition is uncommon after EVAR, given the anticipated short length of hospital stay,\(^{584}\) a risk for malnutrition exists for patients who undergo open aneurysm repair, particularly those with preexisting renal insufficiency.\(^{585}\) Early feeding reduces the likelihood of malnutrition, as demonstrated in a randomized trial of 128 patients undergoing colorectal and abdominal vascular surgery.\(^{586}\)

We recommend optimization of preoperative nutritional status prior to elective open aneurysm repair, if repair will not be unduly delayed.

Level of recommendation Strong
Quality of evidence High
We recommend using nasogastric decompression intraoperatively for all patients undergoing open aneurysm repair, but postoperatively, only for those patients with nausea and abdominal distention.

Level of recommendation: Strong
Quality of evidence: High

We recommend parenteral nutrition if a patient is unable to tolerate enteral support seven days after aneurysm repair.

Level of recommendation: Strong
Quality of evidence: High

Prophylaxis for deep vein thrombosis. Early mobilization and shorter length of stay has reduced the incidence of venous thromboembolism after aortic surgery relative to earlier eras of open aortic repair. Two studies using the NSQIP database determined that the 30-day incidence of venous thromboembolism after open aneurysm repair and EVAR were less than 2% and 1%, respectively. The risk of postoperative deep venous thrombosis (DVT) prophylaxis after open aneurysm repair was first highlighted by Olin et al. who performed postoperative venography in 50 consecutive patients. While most patients were asymptomatic, 21% had evidence of an acute DVT, predominantly within the calf veins.

While venous thromboembolism risk stratification can be performed using the Caprini or similar scoring scheme, most patients undergoing aneurysm repair will be classified as moderate (Caprini Risk Score: 3-4) or high (Caprini Risk Score: >5) risk. For example, the majority of patients undergoing aneurysm repair will be 61 years of age or older (Caprini score of 2 points) with planned surgery of greater than 45 minutes (Caprini score of 2 points). These two factors alone yield a Caprini Risk Score of 4. Nonetheless, recommendations for thromboprophylaxis after aneurysm surgery are not well defined, given the lack of evidence for safety, particularly among patients undergoing open surgical repair, or effectiveness.
We recommend thromboprophylaxis that includes intermittent pneumatic compression and early ambulation for all patients undergoing OSR or EVAR.

Level of recommendation: Strong
Quality of Evidence: High

We suggest thromboprophylaxis with unfractionated or low molecular weight heparin for patients undergoing aneurysm repair at moderate to high risk for venous thromboembolism and low risk for bleeding.

Level of recommendation: Weak
Quality of evidence: Low

Postoperative blood transfusion. A threshold for blood transfusion after OSR or EVAR has not been established. A number of studies suggest that anemia or a low hemoglobin level is associated with increased mortality after open AAA repair. In a review of a statewide database, transfusion after major vascular procedures occurred in 25% of patients at a median hemoglobin level of 7.7 g/dL. Perioperative transfusion was independently associated with death, myocardial infarction, and pneumonia. A hemoglobin concentration of less than 7 g/dL has been recommended as a transfusion threshold for a number of high risk conditions in both critical and ambulatory care. Given the prevalence of coronary artery disease among patients undergoing vascular surgery, blood transfusion for a hemoglobin concentration of less than 10 g/dL has been a common practice. However, a meta-analysis comparing transfusion thresholds of 7 to 8 g/dL and 9 to 10 g/dL did not discern a difference in outcome for patients undergoing either cardiac or vascular surgery. In this regard, motivated by a desire to reduce the established risks of blood transfusion, decrease blood utilization, and lower costs, a recent Cochrane analysis supports more restrictive guidelines for all patients, including those with cardiovascular disease. Therefore, on the basis of currently available evidence, in the absence of ongoing blood loss, transfusion during or after OSR or EVAR is recommended only if the hemoglobin concentration is at or below 7 g/dL.
In the absence of ongoing blood loss, we suggest a threshold for blood transfusion during or after aneurysm repair at a hemoglobin concentration of 7 gm/dL or below.

Level of recommendation    Weak
Quality of evidence     Low

Perioperative pain management. Central regional opioids, systemic opioid patient-controlled analgesia and peripheral regional techniques are recommended for pain management, including multimodal techniques such as central regional blockage with local anesthetics.\(^6^0^3^\) The geriatric population warrants special consideration and incorporation of acetaminophen is recommended in the postoperative pain plan.\(^6^0^3^\)

A Cochrane analysis reviewed 1,498 patients enrolled in 15 trials who were treated with either epidural or systemic opioid analgesia, most often after open aortic surgery.\(^6^0^4^\) The method of pain control had no impact on 30 day mortality, but initial pain scores, the duration of ventilation, postoperative respiratory failure, gastrointestinal bleeding, ICU length of stay, and the incidence of MI were all reduced among patients treated with epidural analgesia.\(^6^0^4^, 6^0^5^\)

Epidural anesthesia may also be beneficial for patients with COPD.\(^6^0^6^\) Complications after the placement of an epidural catheter are uncommon, but include epidural abscess and hematoma.\(^6^0^7^\)

There is limited evidence that a pre-incisional transversus abdominis plane (TAP) block decreases the use of pain medication after major abdominal surgery.\(^6^0^8^, 6^0^9^\) A Cochrane review of 8 studies with 358 participants found TAP reduced opioid consumption in a subset of studies and had no impact on nausea, vomiting or sedation scores.\(^6^1^0^\)

We recommend multimodality treatment, including epidural analgesia, for postoperative pain control after open surgical repair of an abdominal aortic aneurysm.

Level of recommendation    Strong
Quality of evidence     High
POSTOPERATIVE AND LONG-TERM MANAGEMENT

Late outcomes

Endoleak. Endoleak is defined as persistent blood flow in the aneurysm sac after EVAR. Endoleaks at the time of repair may be present in up to 25%. Although an endoleak may often resolve without intervention, some require immediate or delayed treatment to prevent aneurysm rupture. Additionally, some endoleaks develop months or years after EVAR. Thus, lifelong surveillance after EVAR is required. An endoleak may be identified by CT imaging or duplex ultrasound. There are four main types of endoleak. Management depends on endoleak type and the associated risk of sac rupture.

Type I endoleak. A type I endoleak occurs when there is an incomplete seal at the proximal aortic attachment site (type IA) or at the distal iliac attachment site (IB). A type IA endoleak most often occurs in the presence of a short or severely angulated neck, a reverse tapered neck, or when the attachment site contains considerable thrombus or calcification. A type I endoleak is associated with elevated sac pressure and an ongoing risk of rupture. While a small endoleak may seal and can be observed, it is preferable that when identified at the time of repair, every attempt should be made to treat a type I endoleak before the conclusion of the procedure. Balloon molding of the proximal seal zone, placement of a proximal cuff, and endostaples have all been used with varying degrees of success. Endostaples may reduce the risk of endograft migration and a type IA endoleak, but long-term data are limited. Other options for type IA endoleak treatment include embolization with coils or glue, proximal extension with a chimney approach, or conversion to a fenestrated endograft. A type IB endoleak is treated with distal extension of the iliac limb, if repeat angioplasty fails to eliminate the endoleak. It may be necessary to extend the endograft to the external iliac artery.
with coil occlusion of hypogastric artery. Conversion to open repair should be considered in the
presence of a persistent type IA endoleak.624

**Type II endoleak.** Persistent filling of the aneurysm sac from patent lumbar arteries or
the inferior mesenteric artery (IMA) constitutes a type II endoleak.611-613 Type II endoleaks are
the most common endoleak, present at the time of repair in up to one-fourth of patients. When
identified at the time of the procedure, treatment is not indicated, as at least 50% will
spontaneously resolve.617, 622, 623, 625 The incidence of type II endoleak at 6 months is 10% to
15%.626-628 Factors that increase the risk of a persistent type II endoleak include a patent
inferior mesenteric artery, number and diameter of patent lumbar arteries, especially L3 and L4
lumbar arteries, and ongoing anticoagulation.629-632

The fate of persistent type II endoleaks is variable. Aneurysm sac size may decrease in
up to 25%,626, 633 remain stable in 50%-70%,634 or increase in up to 25% of patients. The
delayed onset of a type II endoleak may also be noted 6 months or later after EVAR. A delayed
type II endoleak may be associated with aneurysm sac expansion;635 however, expansion in sac
diameter greater than 10 mm is uncommon.636

Treatment of a type II endoleak includes embolization of the inferior mesenteric or lumbar
arteries with coils or glue,623 direct trans-lumbar injection of the aneurysm sac,616 transcaval
embolization,637, 638 or laparoscopic ligation of the inferior mesenteric and lumbar arteries,639 all
with variable success rates. Up to 60% of treated aneurysms continue to expand requiring
multiple procedures and in some cases explant with conversion to open repair.640, 641 Stent graft
preservation with oversewing of the inferior mesenteric and lumbar arteries from within the sac
has been reported.642-644 Type II endoleak remains a challenge to treat effectively.645

Rupture from a type II endoleak is rare and more often related to an unrecognized type I
endoleak. The decision to treat is based on the size and expansion (≥ 5 mm) of the aneurysm,
type and size of patent inflow and outflow vessels, and the presence of symptoms.646, 647
Selective intervention appears both safe and cost-effective.636, 648
**Type III endoleak.** A type III endoleak occurs when there is incomplete seal between components or component separation and less frequently due to fabric erosion. The aneurysm sac becomes re-pressurized with an increased risk of rupture. All type III endoleaks should be treated. When the endoleak is present at the contralateral gate or between an iliac limb and an iliac extension, treatment entails bridging the gap with an appropriately sized limb.617,618

**Type IV endoleak.** A type IV endoleak is due to fabric porosity, which may be present at the time of repair. All type IV endoleaks seal spontaneously and do not require treatment.

**Endotension.** Endotension is defined as sac enlargement without a discernable endoleak. It may be caused by blood flow that is undetectable at the limits of the imaging modality, pressure transmission through fabric,649,650 or accumulation of a serous ultra-filtrate across a microporous fabric.612 Endotension is less common with the newer generation grafts. Management should be individualized, and may include observation, relining of low-porosity endografts, or rarely explantation and conversion to open repair.

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*We recommend treatment of Type I endoleaks.*

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*We suggest treatment of Type II endoleaks associated with aneurysm expansion.*

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*We recommend surveillance of Type II endoleaks not associated with aneurysm expansion.*

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*We recommend treatment of Type III endoleaks.*

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We suggest no treatment of Type IV endoleaks.

Level of recommendation: Weak
Quality of Evidence: Low

We recommend open repair if endovascular intervention fails to treat a Type I or III endoleak with ongoing aneurysm enlargement.

Level of recommendation: Strong
Quality of Evidence: Moderate

We suggest open repair if endovascular intervention fails to treat a Type II endoleak with ongoing aneurysm enlargement.

Level of recommendation: Weak
Quality of Evidence: Low

We suggest treatment for ongoing aneurysm expansion, even in the absence of a visible endoleak.

Level of recommendation: Weak
Quality of Evidence: Low

Device migration. Device migration most commonly presents as caudal migration of the proximal endograft. A delayed type IA endoleak will occur if the endograft migrates into the aneurysm sac. Rarely aortic remodeling will create forces that cause cranial migration of the distal landing zone and a resultant type IB endoleak. Device migration is a late event, typically occurring two or more years after implantation. Factors that predispose to migration include hostile neck anatomy, inadequate device fixation, and progressive aortic dilatation and elongation. Treatment for caudal device migration depends on anatomic considerations, including the quality of the aortic seal zone, as well as the distance between the renal arteries and the flow divider of the original endograft. Options include conversion to an aorto-unilateral iliac bypass with crossover femoral-femoral bypass and iliac occlusion or placement of an aortic extension.
cuff. However, the former approach has a lower risk of recurrent endoleak and rupture since treatment with an aortic cuff is often associated with continued risk of device migration. \(^657\) Alternatives include proximal extension with a branched or fenestrated endograft. \(^351\) FEVAR for EVAR failure is technically complex because the existing endograft may interfere with rotational torque and visualization of the radio-opaque markers.

**Limb occlusion.** Nearly 25% of all arterial re-interventions after open repair are due to limb occlusion, and are most common in patients with associated occlusive disease. \(^658\) 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, a patient may present before complete occlusion of the graft.

Endografts are at a higher risk for limb thrombosis than bifurcated surgical grafts, as observed in the EVAR-1 trial. \(^659\) Endograft limbs can be narrowed by a calcified small aortic bifurcation or by tortuous, angulated, and diseased iliac arteries. Although device-dependent, the incidence of limb occlusion after EVAR is approximately 4%, with the majority of occlusions presenting within two months and nearly all within the first year after EVAR. \(^370, 660, 661\) Non-supported limbs are at especially high risk of limb occlusion. \(^662\) However, stented limbs may also occlude due to fabric in-folding and kinking between stents. \(^660, 663\) While the causes of endograft limb occlusion may be related to a number of factors, one of the most common reasons is compromised outflow. Occlusion of the internal iliac artery with or without extension of the endograft limb to the external iliac artery or unrecognized distal dissection may increase the risk of limb thrombosis. \(^664, 665\) Acute endograft limb occlusion often presents with worsening claudication rather than critical ischemia, provided the limb has been deployed proximal to the internal iliac artery, which facilitates collateral perfusion to the lower extremity.

A stenotic limb, noted on Duplex or by a reduction in ankle-brachial index, can be treated by stent placement. An occluded limb after EVAR or open repair includes, can be treated by
thrombectomy, pharmacolytic therapy with secondary endovascular or local surgical intervention, or by femoral-femoral or axillo-femoral bypass. Mechanical balloon thrombectomy is less likely to be successful for treatment of an endograft limb thrombosis due to difficulty advancing the catheter beyond the occluded segment, concerns related to dislodging or disrupting the sealing zones, and the presence of stents, which may interfere with balloon thrombectomy. The underlying cause of the thrombosis must be identified and treated and if a mechanical cause for thrombosis cannot be determined, femoral-femoral bypass should be considered. Simple thrombectomy or thrombolysis will often lead to recurrent early thrombosis. Five year patency for a femoral-femoral bypass graft, when placed in the treatment of aneurysmal disease exceeds 80%. We recommend that follow-up of patients after aneurysm repair include a thorough lower extremity pulse exam or ABI.

We recommend a prompt evaluation for possible graft limb occlusion if patients develop new onset lower extremity claudication, ischemia, or a reduction in ABI after aneurysm repair.

Graft infection. All implanted aortic prostheses are at risk for infection either at implantation or later by hematogenous seeding. Although graft infection is rare with an incidence of 0.3%, historically, it has been the indication for intervention in up to 25% of redo aortic surgery. Although controversial, the risk of graft infection after EVAR may be lower than after open repair, perhaps due to delivery of the endoprosthesis through a completely enclosed system. However, the EVAR-1 trial had a comparable incidence of device infection between EVAR and open repair over a four year follow-up period. Similarly, in a
recent analysis of more than 45,000 Medicare beneficiaries, graft infections or aortoenteric fistulae four years after EVAR or open repair was comparable for both groups (~0.3%). Likewise, Vogel reported a nearly identical two-year incidence of graft infection (< 0.2%) for OSR or EVAR in a review of 14,000 patients undergoing aneurysm repair. Graft infection after EVAR or open repair may present in isolation or with an aortoenteric fistula.

While aortic graft infection presents on average three years or later after open repair, endograft infection often manifests earlier for reasons that remain unclear. Femoral artery extension of a prosthesis increases the incidence of graft infection from 1% to 3%. Other predisposing factors for graft infection include the need for surgical revision and emergent indication for initial surgery. Generalized sepsis, groin drainage, pseudoaneurysm formation, or ill-defined pain may be presenting symptoms and staphylococcal organisms are the most frequent isolates. CT imaging may provide an initial estimate of the extent of infection, determine if a pseudoaneurysm exists at the proximal anastomosis, and assist in operative planning for effective revascularization of the lower extremities.

Conventional treatment for graft infection includes staged excision of all infected graft material with extra-anatomic reconstruction, particularly in the presence of extensive contamination and gross purulence. In situ reconstruction using femoral vein, a silver or antibiotic impregnated graft, or cryopreserved allograft represent additional surgical options, and particularly appropriate in the presence of minimal contamination. Placement of a silver or antibiotic prosthetic or PTFE graft in a grossly contaminated field is reserved for the unstable patient.

Treatment of an endograft infection poses unique challenges. A dense inflammatory reaction can completely obliterate natural tissue planes and endograft hooks or suprarenal fixation may dictate the need for supraceliac cross clamping to explant the device. Further, hooks and the suprarenal segment may be embedded into the aortic wall or covered with a pseudo-intima requiring careful removal to avoid injury to the operator, aortic wall, as well as
preservation of the aortic neck for closure or in situ reconstruction. Despite the introduction of endograft re-sheathing techniques, attendant renal ischemia is an established risk. Percutaneous drainage and antibiotic therapy has been suggested for patients unfit to undergo open repair.\textsuperscript{673, 687-689}

An aortoenteric fistula can complicate a graft infection in 1\% to 2\% of patients.\textsuperscript{671, 676} Although the duodenum is most frequently affected, all viscera, including small and large bowel, have been implicated.\textsuperscript{672, 676} A common presentation is upper gastrointestinal bleeding, as a herald bleed, which may progress to exsanguinating hemorrhage.\textsuperscript{690} Any upper gastrointestinal bleeding in a patient with an aortic graft should raise the suspicion of an aortoenteric fistula. The diagnosis may occasionally be confirmed by endoscopy or CT imaging.\textsuperscript{691-693} Bleeding is more common when the anastomosis erodes into the gastrointestinal tract, while sepsis and abscess formation are 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 defect.\textsuperscript{694} While endovascular repair of an aortoenteric fistula is uniformly unsuccessful, severe hemorrhage may necessitate the use of an endograft to temporarily control bleeding, as a "bridge" to definitive surgical repair.\textsuperscript{695, 696}

**Prevention of an aortic graft infection.** Recommendations for antibiotic prophylaxis after placement of an aortic prosthesis following open surgical repair or EVAR have historically followed guidelines for the prevention of infective endocarditis after a prosthetic heart valve. Recent guidelines have sought to reduce the indications of antibiotic prophylaxis, particularly with the publication of the National Institute for Health and Clinical Excellence (NICE) guidance in 2008, which recommended against antibiotic prophylaxis for infective endocarditis, regardless of the dental, genitourinary, or gastrointestinal procedure, or predisposing cardiac condition, including the presence of a prosthetic valve.\textsuperscript{697} A recent report has observed a small but statistically significant increase in infective endocarditis cases in the UK since the implementation of the NICE recommendations.\textsuperscript{698} Although limitations exist in this study and
causation has not been established, concerns have been raised. Several investigations note a relationship between dental procedures and infective endocarditis in high risk patients. Current European Society of Cardiology, American College of Cardiology, and American Heart Association guidelines recommend ongoing use of antibiotic prophylaxis for patients with a prosthetic valve undergoing high risk procedures. High risk procedures, as defined in both guidelines, included dental procedures involving the manipulation of the gingival or periapical region of teeth or perforation of the oral mucosa, including scaling and root canal procedures. Both guidelines noted that there was no compelling evidence that bacteremia resulted from respiratory tract procedures, gastrointestinal or genitorurinary procedures, dermatological or musculoskeletal procedures and prophylaxis was not recommended for patients undergoing these procedures unless the procedures were performed in the presence of an infection. It was also strongly recommended that any potential sources of dental sepsis be eliminated at least 2 weeks before implantation of a prosthetic valve or other intracardiac or intravascular foreign material, unless the procedure was deemed urgent. Others have raised concerns that patients undergoing colonoscopy or urological procedures, especially the elderly and those with cancer or who are immunocompromised require antibiotic prophylaxis. Both the European and AHA/ACC guidelines noted that their recommendations were not based on strong evidence and further prospective evaluation was recommended.

For those patients with an aortic prosthesis, whether placed by open surgical repair or EVAR, we suggest antibiotic prophylaxis to prevent graft infection prior to any dental procedure involving the manipulation of the gingival or periapical region of teeth or perforation of the oral mucosa, including scaling and root canal procedures. We also suggest antibiotic prophylaxis prior to respiratory tract procedures, gastrointestinal or genitorurinary procedures, dermatological or musculoskeletal procedures, if the potential for infection exists or the patient is immunocompromised.
We recommend antibiotic prophylaxis to prevent graft infection prior to any dental procedure involving the manipulation of the gingival or periapical region of teeth or perforation of the oral mucosa, including scaling and root canal procedures, for any patient with an aortic prosthesis, whether placed by open surgical repair or EVAR.

Level of recommendation: Strong
Quality of evidence: Moderate

We suggest antibiotic prophylaxis prior to respiratory tract procedures, gastrointestinal or genitorurinary procedures, dermatological or musculoskeletal procedures, for any patient with an aortic prosthesis, if the potential for infection exists or the patient is immunocompromised.

Level of recommendation: Good practice statement
Quality of evidence: Ungraded

After aneurysm repair, we recommend prompt evaluation for possible graft infection if a patient presents with generalized sepsis, groin drainage, pseudoaneurysm formation, or ill-defined pain.

Level of recommendation: Strong
Quality of evidence: High

We recommend prompt evaluation for possible aortoenteric fistula in a patient presenting with gastrointestinal bleeding after aneurysm repair.

Level of recommendation: Strong
Quality of evidence: High

In patients presenting with an infected graft in the presence of extensive contamination with gross purulence, we recommend extra-anatomic reconstruction followed by excision of all graft material along with aortic stump closure covered by an omental flap.

Level of recommendation: Strong
Quality of evidence: Moderate

In patients presenting with an infected graft with minimal contamination, we suggest in situ reconstruction with cryopreserved allograft.

Level of recommendation: Weak
Quality of evidence: Moderate

In a stable patient presenting with an infected graft, we suggest in situ reconstruction with femoral vein after graft excision and debridement.

Level of recommendation: Weak
Quality of evidence: Moderate
In unstable patients with infected graft, we recommend in situ reconstruction with a silver or antibiotic impregnated graft, cryopreserved allograft, or a PTFE graft.

Level of recommendation    Strong
Quality of evidence     Moderate

Incisional hernia. Retroperitoneal incisions for aortic aneurysm repair may lead to denervation of the 11th intercostal nerve, which has been associated with numbness in the region of the incision in up to one-third of patients, as well as bulging of the lateral abdominal wall with musculature atrophy in 7 to 15%. Transperitoneal repair is associated with a higher incidence of late small bowel obstruction and approximately 10% of patients may develop a ventral hernia within the first 6 years after repair, particularly among those who are obese. Should a midline incision be used, continuous suturing technique and avoidance of rapidly absorbable sutures is recommended. Wound infections of the abdominal incisions are rare (0.4%).

Para-anastomotic aneurysm. Para-anastomotic aneurysms after aortic aneurysm repair include both false aneurysms resulting from a disruption of the anastomosis and true aneurysms that develop adjacent to the anastomosis. True metachronous aneurysms occur at a greater frequency than anastomotic pseudoaneurysms. However, the incidence of para-anastomotic aneurysms is not well defined. Predisposing factors include hypertension, COPD, and tobacco use. In the era prior to CT imaging, Szilagyi analyzed a 15-year experience in which anastomoses at the femoral artery were at highest risk (3%), followed by the iliac artery (1.2%) and infrarenal aorta (0.2%). Subsequent studies have reported an incidence after open repair of between 4% and 10% at 10-year follow-up. In one study of 511 patients, Kaplan-Meier analysis revealed a probability of para-anastomotic aneurysm of 0.8% at five years, 6.2% at 10 years, and 35.8% at 15 years. This observation has been confirmed by others, particularly the risk of femoral pseudoaneurysm formation among patients treated with
an aortobifemoral graft.\textsuperscript{709, 712} 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 open repair carries a significant risk of major morbidity and mortality and endovascular repair, where anatomically feasible, provides a minimally invasive option.\textsuperscript{713, 714} Infrarenal and fenestrated endografts have been used with chimney, as well as snorkeling techniques.\textsuperscript{715-718}

**Recommendation for postoperative surveillance**

Systematic reviews by the SVS showed a significant incidence of postoperative endoleaks up to 5 years after EVAR, which provides rationale for surveillance. The evidence was insufficient to recommend an optimal frequency of surveillance. MRI was more sensitive than CTA and contrast CT although the difference was small. Duplex ultrasound was inferior to CT and contrast enhanced ultrasound in terms of detection rate, although leaks missed on ultrasound did not require intervention or were not considered to be clinically significant.

The goal of postoperative surveillance is to prevent late rupture and aneurysm-related death. After open surgical repair, an anastomotic aneurysm or aneurysmal dilatation in the adjacent visceral aorta or iliac arteries may occur in 1\%, 5\%, and 20\% of patients at 5, 10 and 15 years.\textsuperscript{709, 712} Thus, abdominal and pelvic CT imaging is recommend every 5 years after open surgical repair.

Surveillance after EVAR is performed to identify sac growth, endoleak, device migration, or device failure. A comprehensive analysis of contemporary Medicare patients revealed that the incidence of late rupture 8 years after EVAR is over 5\%.\textsuperscript{340} Most ruptures developed from type I or type III endoleaks with sac enlargement, predisposed by unfavorable anatomy for endovascular repair.\textsuperscript{720-722}
**Surveillance imaging modality.** Initially recommended surveillance protocols were consistent with those used by FDA-sponsored pivotal trials with CT imaging at 1, 6 and 12 months and yearly thereafter. The 6-month CT scan can be eliminated from routine surveillance if the one-month scan shows no concerning endoleak or sac enlargement. Color duplex ultrasonography, contrast-enhanced color duplex ultrasonography, and three-dimensional contrast-enhanced ultrasonography have all shown to be accurate in detecting type I and type III endoleaks, as well as sac enlargement. Ultrasonography eliminates radiation exposure, reduces cost, and avoids use of a nephrotoxic contrast agent. Further surveillance with ultrasound is safe if CT imaging one year after EVAR demonstrates no endoleak and stable sac size or for those patients with a type II endoleak and a stable aneurysm size. A new endoleak, graft migration or aneurysm sac growth greater than 5 mm -10 mm should prompt further evaluation with a CT scan.

**Surveillance outcomes.** Surveillance non-compliance rates approach 60% with gaps observed 3 to 4 years after EVAR, particularly among patients of advanced age, Medicaid eligibility, or after treatment at a low volume center. Although the risk of late device related complications and aneurysm rupture are well documented, population studies have not demonstrated that annual EVAR surveillance confers a survival benefit or decreases aneurysm-related mortality. Not all late ruptures are preceded by endoleak or sac enlargement, which suggests that not all late ruptures can be prevented by vigilant surveillance.

**Summary.** Current recommendations for surveillance after EVAR include a CT scan at one month. Concerning findings should prompt surveillance at 6 months. In the absence of a type I or type III endoleak and sac enlargement surveillance can be performed with CT or color duplex ultrasound. Annual duplex is most likely sufficient for routine surveillance in the absence of new endoleak or sac enlargement. New findings should prompt CT imaging to evaluate for I or type III endoleaks. Abdominal and pelvic CT imaging should be performed every 5 years after open surgical repair or EVAR.
We recommend surveillance during the first year after EVAR using contrast enhanced CT at one month and in the absence of an endoleak or sac enlargement, contrast enhanced CT or color duplex sonographic imaging at 12 months.

If a Type II endoleak is observed one month after EVAR, we suggest postoperative surveillance with contrast enhanced CT and color duplex sonographic imaging at six months.

If neither endoleak nor AAA enlargement is observed one year after EVAR, we suggest color Duplex ultrasonography when feasible, or CT imaging if ultrasound is not possible, for annual surveillance.

If a Type II endoleak is associated with an aneurysm sac that is shrinking or stable in size, we suggest color Duplex ultrasonography for continued surveillance at 6 month intervals for 24 months and then annually thereafter.

If a new endoleak is detected, we suggest evaluation for a Type I or Type III endoleak.

We suggest non-contrast CT imaging of the entire aorta at five-year intervals after open repair or EVAR.
The complexity and intensity associated with aortic aneurysm treatment results in significant costs and resource utilization. These initial costs include imaging, preoperative risk management, the operating room, personnel, implants, and recovery. Long-term follow-up and imaging has costs, as well as the treatment of any complications from the initial procedure or failure of the graft itself. Besides these provider costs, patients and their families also bear cost from lost work productivity and out-of-pocket expenditures.

Contemporary estimates show that in-hospital costs of treatment for open repair or EVAR are approximately $40,000 in the US, with lower cost estimates in Canada ($16,000 USD) and other countries. Implants are a significant portion of EVAR costs (34-52%) but these costs are offset by the higher costs after open repair from longer hospitalization. While open repair is slightly more expensive during the initial hospitalization, there are no significant differences seen in long-term follow-up due to need for surveillance imaging and re-interventions after EVAR.

Calculating cost of care does not shed light on the benefit of care. The major benefit of treatment for patients with an aortic aneurysm is increased survival. However, patients endure a decreased quality of life after surgery, which may be prolonged should a complication occur. Because EVAR confers a lower complication rate and smaller incisions compared to open repair, patients undergoing EVAR generally have better health-related quality of life within the first 12 months, though there is no significant difference beyond the first year.

When cost and effectiveness are combined, cost-effectiveness analysis can reveal the value of different treatment options, and also allow comparison to other treatments in other fields. Early Markov decision analysis models show that EVAR was cost-effective compared to open repair, with an incremental cost effectiveness ratio of $22,826. However, contemporary Markov models using data from the DREAM, EVAR-1, OVER, and ACE randomized trials showed EVAR to be cost-effective based on the OVER trial data, but no difference in
lifetime cost-effectiveness was derived from data generated by the European trials, suggesting that results may not be generalizable among different countries. EVAR also does not appear to be cost-effective for treatment of complicated aneurysms. Cost comparisons for fenestrated or branched EVAR graft demonstrated higher costs in comparison to open repair (€38,212 vs. €16,497) without significant differences in 30-day mortality.

Most of the data for cost-effectiveness pertains to elective cases, where expected morbidity and mortality can be managed through patient selection and preparation. In the urgent and emergent situation, morbidity and mortality risk is higher, leading to higher costs and lower quality of life. Nevertheless, evidence suggests that EVAR in the acute setting is favorable.

A single screening ultrasound for abdominal aortic aneurysm in asymptomatic men over 65 has shown to be cost-effective in the UK and through Markov modeling. In the UK, the cost per life year saved with screening was $1,173, which is less costly than screening programs for breast, cervical, and colorectal cancer. The cost-effectiveness of screening for younger cohorts, women, and reimaging intervals for small aneurysms remains uncertain. Early treatment of a small aneurysm, less than 5 cm in diameter, is not cost-effective in comparison to serial imaging.

Because of the lower peri-operative complication rates with EVAR, patients who could not undergo open repair are being offered EVAR or hybrid procedures. In the setting of constrained costs and capitated care, expensive procedures for asymptomatic elderly patients with significant comorbidities who will not derive a meaningful survival benefit are not cost-effective.

EVAR implants are a major component of costs of treatment. As additional devices have been introduced to the market, it had been speculated that competition would lower price and incentivize further innovation. However, a decrease in device cost has not been observed. Several institutions have reported that EVAR confers a negative operating margin in Medicare
While cost-effectiveness results can vary among different patient populations, healthcare systems, and over time, the factors that influence cost and outcomes remain consistent. In a future of rising costs and constrained resources, cost-effectiveness analysis will provide a basis to guide healthcare policy that sustains healthcare coverage for all.

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

Advances in biotechnology, drug discovery, and in the engineering sciences hold significant promise for the development of new diagnostic tests, bioactive compounds, as well as intraoperative tools and devices that will enhance the care of the patient with an aortic aneurysm. Research is needed to: (1) ascertain genetic or other biological factors that accurately measure the life-long risk of developing an aortic aneurysm; (2) discover pharmacological agents to limit aneurysm enlargement; (3) characterize biomarkers or imaging derived determinants of rupture risk; (4) design prostheses that resist infection and thrombosis; (5) develop tools, intraoperative imaging or robotic systems, as well as improved endovascular grafts that facilitate repair in the presence of challenging anatomy and improve the safety and accuracy of device deployment; and (6) identify approaches that reliably treat type I and II endoleaks. All within the framework of enhancing cost effective care.

A number of areas of uncertainty also exist in the care of patients with an abdominal aortic aneurysm in the application of existing technology that would benefit from further investigation. Further, given the role of sex differences in the pathophysiology and outcomes of AAA, investigations in cells, animals, and humans should be designed to assess for gender and should clearly state related study population details so that results can be interpreted appropriately. While the following list is not meant to be comprehensive, future research efforts should consider addressing the following topics:

- What is the most cost effective and clinically effective surveillance protocol for the patient with a small aneurysm?
Should the aortic size index replace aortic diameter as a determinant for recommending aneurysm repair?

Do female patients benefit from a refined metric, such as the aortic size index, or size threshold for recommending repair?

Which quality and volume metrics best identify centers that should engage in either EVAR or the open surgical repair of an aortic aneurysm?

Does use of a perioperative mortality risk scoring scheme provide benefit in patient and family communication and mutual decision making?

Does a perioperative mortality risk scoring scheme provide utility to surgeons, patients, and families in guiding recommendations for repair in the high risk patient?

Can perioperative mortality risk scoring schemes be further refined to enhance their predictive ability?

Does a frailty assessment enhance our ability to identify those patients who will not benefit from aneurysm repair?

Can a single risk-benefit scoring scheme be developed, which incorporate risk of repair, risk of aneurysm rupture, and anticipated life expectancy?

Would a risk-benefit scoring scheme that incorporate risk of repair, risk of aneurysm rupture, and anticipated life expectancy assist mutual decision making between the surgeon, patient, and their family?

Will a defined system of care and associated time benchmark from first medical contact to intervention improve outcomes for the patient with a ruptured aneurysm?

Which factors are most important in optimizing patient outcomes within a system of care for the treatment of a ruptured aneurysm?

Is prophylaxis for deep vein thrombosis needed for the patient undergoing EVAR?
• Does the patient undergoing open surgical repair and at low or moderate risk for deep vein thrombosis benefit from heparin prophylaxis?

• What is the optimal hemoglobin level that necessitates transfusion in the stable postoperative patient without ongoing blood loss?

• What is the optimal interval, imaging modality, and duration for postoperative surveillance after aneurysm repair?

• What is the most cost effective and clinically effective surveillance protocol for the patient after EVAR?
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Table Legends

Table 1. Evidence profiles derived from evidence synthesis reports (systematic reviews and meta-analysis) that were identified through an umbrella systematic review.


Figure 1. The annual adult per capita cigarette consumption and age-adjusted abdominal aortic aneurysm (AAA) deaths per 100,000 white men by year in the United States. AAA: abdominal aortic aneurysm. From Lederle FA. The rise and fall of abdominal aortic aneurysm. Circulation. 2011;124(10):1097-9.


Figure 3. Meta-regression of abdominal aortic aneurysm growth rate estimates by aneurysm diameter (please see primary source for individual study citations). The solid line represents the overall regression, the dotted line connects estimates from the same study, and circles have diameters that represent amount of information. Adapted from Thompson SG, Brown LC, Sweeting MJ, Bown MJ, Kim LG, Glover MJ, et al. Systematic review and meta-analysis of the growth and rupture rates of

Figure 4. Estimated time to have a 10% probability of attaining an aortic diameter of 5.5 cm in male patients (please see primary source for individual study citations). Black diamonds represent 95% confidence intervals and errors represent 95% prediction intervals. MASS: Multicenter Aneurysm Screening Study; PIVOTAL: Positive Impact of endovascular Options for Treating Aneurysm earLy; UKSAT: United Kingdom Small Aneurysm Trial. Adapted from Thompson SG, Brown LC, Sweeting MJ, Bown MJ, Kim LG, Glover MJ, et al. Systematic review and meta-analysis of the growth and rupture rates of small abdominal aortic aneurysms: implications for surveillance intervals and their cost-effectiveness. Health Technol Assess. 2013;17(41):1-118.

Figure 5. Algorithm for management of the patient with a suspected or confirmed ruptured aneurysm.

Figure 6. Referring hospital checklist for the patient with a suspected or confirmed ruptured aneurysm.

Figure 7. Receiving hospital personnel alert checklist for management of the patient with a suspected or confirmed ruptured aneurysm.
Table 1: Evidence profiles derived from evidence synthesis reports (systematic reviews and meta-analysis) that were identified through an umbrella systematic review:

<table>
<thead>
<tr>
<th>Systematic reviews</th>
<th>Question/comparison</th>
<th>Findings (Quality of evidence)</th>
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<tbody>
<tr>
<td><strong>Screening, diagnosis and preoperative surveillance</strong></td>
<td></td>
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<tr>
<td>Guirguis-Blake, 2014(^{752})</td>
<td>Effectiveness of screening for AAA</td>
<td>• Screening (primarily in men &gt;65) was associated with reduction in AAA-mortality (High). Absolute reduction (4 per 1,000). Number needed to screen (238).</td>
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<td>Cosford, 2011(^{753})</td>
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<td>Takaji, 2010(^{754})</td>
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<td>Alamoudi, 2015(^{755})</td>
<td>Diagnostic accuracy of imaging for AAA compared to digital subtraction angiography</td>
<td>• AAA &lt; 2.5 cm, the mean reported sensitivities and specificities: -DUS: 81% and 91.1% -CTA: 84.3% and 98.4% -MRA: 95.8% and 95.8% • Non-radiologist performed ultrasound achieved acceptable sensitivity and specificity for both detection and measurement of AAA.</td>
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<td>Concannon, 2014(^{756})</td>
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<td><strong>Factors affecting growth and rupture of small AAA</strong></td>
<td>Rupture was higher in women, smokers and elevated blood pressure (moderate)</td>
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<tr>
<td>Sweeting, 2012(^{757})</td>
<td>Factors affecting growth and rupture of small AAA</td>
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<tr>
<td><strong>Surveillance intervals for small AAA</strong></td>
<td>For each 0.5-cm increase in AAA diameter, growth rates increased on average by 0.59 mm per year and rupture rates increased by a factor of 1.91 (moderate).</td>
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<td>RESCAN Collaborators, 2013(^{758})</td>
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<td><strong>Treatment</strong></td>
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<td>Stather, 2013(^{759})</td>
<td>Open vs endovascular repair</td>
<td>• EVAR had lower 30-day or in-hospital mortality rate (high) • Reduction in quality of life at 3 months more pronounced with open repair • At 2 and 4 years, no difference in mortality (low) • EVAR required more reinterventions and rupture (high).</td>
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<td>Coughlin, 2013(^{760})</td>
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<tr>
<td>Biancari, 2011(^{761})</td>
<td>Open vs endovascular repair (age &gt;80)</td>
<td>• Elective EVAR was associated with lower immediate postoperative mortality and morbidity (low, observational data)</td>
</tr>
<tr>
<td>Kontopodis, 2015(^{762})</td>
<td>Open vs endovascular repair (age &lt;70)</td>
<td>• EVAR was associated with a decreased risk of 30-day mortality</td>
</tr>
<tr>
<td>Study</td>
<td>Intervention</td>
<td>Findings</td>
</tr>
<tr>
<td>-------</td>
<td>--------------</td>
<td>----------</td>
</tr>
<tr>
<td>Saedon, 2015&lt;sup&gt;763&lt;/sup&gt;</td>
<td>Open vs endovascular repair (obese patients)</td>
<td>• EVAR had fewer 30-day postoperative mortality and early postoperative complications (myocardial infarction, chest infection, renal failure, wound infection). Risk of postoperative bowel ischemia and stroke were similar (Low).</td>
</tr>
<tr>
<td>Rayet, 2008&lt;sup&gt;764&lt;/sup&gt;</td>
<td>Open vs endovascular repair (ruptured AAA)</td>
<td>• EVAR had lower mortality in 31 studies that was insignificant in pooled analysis of 3 recent trials or in an adjusted analysis (Low).</td>
</tr>
<tr>
<td>Sweeting 2015&lt;sup&gt;765&lt;/sup&gt;</td>
<td>Transperitoneal vs retroperitoneal approach for elective open AAA repair</td>
<td>• No difference in mortality (low). • Retroperitoneal approach may reduce blood loss, hospital stay and ICU stay (Low). • No differences in aortic cross-clamp time and operating time.</td>
</tr>
<tr>
<td>Ma, 2016&lt;sup&gt;771&lt;/sup&gt;</td>
<td>Retroperitoneal vs transperitoneal approach to the infrarenal abdominal aorta</td>
<td>• Retroperitoneal approach is associated with lower rates of postoperative ileus and pneumonia (moderate)</td>
</tr>
<tr>
<td>Jackson, 2014&lt;sup&gt;773&lt;/sup&gt;</td>
<td>Totally percutaneous versus standard femoral artery access for elective bifurcated abdominal endovascular aneurysm repair.</td>
<td>• One small highly imprecise study (low)</td>
</tr>
<tr>
<td>BaniHani, 2011&lt;sup&gt;774&lt;/sup&gt;</td>
<td>Interventions for preventing venous thromboembolism following abdominal aortic surgery.</td>
<td>• The body of direct evidence is insufficient (2 small studies with methodological limitations) • Extrapolation from indirect evidence is required.</td>
</tr>
<tr>
<td>Twine, 2011&lt;sup&gt;775&lt;/sup&gt;</td>
<td>Effects of statins on AAA</td>
<td>• Reduction in mortality (moderate) • No change in expansion (low)</td>
</tr>
<tr>
<td>Reference</td>
<td>Topic</td>
<td>Findings</td>
</tr>
<tr>
<td>------------------------</td>
<td>----------------------------------------------------------------------</td>
<td>--------------------------------------------------------------------------</td>
</tr>
<tr>
<td>Bergqvist, 2011(^{776})</td>
<td>Pharmacological interventions to attenuate the expansion of AAA</td>
<td>• No consistent pattern of pharmacological influence on expansion rate (low)</td>
</tr>
<tr>
<td>Pieper, 2013(^{777})</td>
<td>Surgical outcomes and hospital volume</td>
<td>• Lower mortality for elective and ruptured AAA repair in high volume hospitals (Low)</td>
</tr>
<tr>
<td>Habets, 2013(^{778})</td>
<td>Magnetic resonance imaging vs computed tomography angiography for the detection of endoleaks after EVAR for AAA.</td>
<td>• Magnetic resonance imaging was more sensitive for endoleaks Type II (moderate).</td>
</tr>
<tr>
<td>Karthikesalinga, 2012(^{779})</td>
<td>Diagnostic accuracy of duplex ultrasonography and contrast-enhanced ultrasonography for types 1 and 3 endoleak</td>
<td>• Both, duplex ultrasonography and contrast-enhanced ultrasonography were highly specific for types 1 and 3 endoleaks (moderate) • Sensitivity estimate were likely similar but less reliable</td>
</tr>
<tr>
<td>Antoniou, 2015(^{780})</td>
<td>Late rupture of AAA after EVAR</td>
<td>• Graft-related endoleaks were the predominant cause of late aneurysm rupture.</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Variable</th>
<th>Estimate</th>
<th>P</th>
<th>OR</th>
<th>95% CI</th>
<th>Score</th>
</tr>
</thead>
<tbody>
<tr>
<td>Male (vs female)</td>
<td>1.74</td>
<td>&lt;.0001</td>
<td>5.71</td>
<td>5.57-5.85</td>
<td>18</td>
</tr>
<tr>
<td>Age (vs &lt;55)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>55-59</td>
<td>1.01</td>
<td>&lt;.0001</td>
<td>2.76</td>
<td>2.55-3.00</td>
<td>11</td>
</tr>
<tr>
<td>60-64</td>
<td>1.68</td>
<td>&lt;.0001</td>
<td>5.35</td>
<td>4.97-5.76</td>
<td>17</td>
</tr>
<tr>
<td>65-69</td>
<td>2.24</td>
<td>&lt;.0001</td>
<td>9.41</td>
<td>8.76-10.12</td>
<td>23</td>
</tr>
<tr>
<td>70-74</td>
<td>2.67</td>
<td>&lt;.0001</td>
<td>14.46</td>
<td>13.45-15.55</td>
<td>28</td>
</tr>
<tr>
<td>75-79</td>
<td>3.02</td>
<td>&lt;.0001</td>
<td>20.43</td>
<td>18.99-21.99</td>
<td>31</td>
</tr>
<tr>
<td>80-84</td>
<td>3.35</td>
<td>&lt;.0001</td>
<td>28.37</td>
<td>26.31-30.59</td>
<td>35</td>
</tr>
<tr>
<td>Race/ethnicity (vs white)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Hispanic</td>
<td>-0.37</td>
<td>&lt;.0001</td>
<td>0.69</td>
<td>0.62-0.77</td>
<td>-4</td>
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<tr>
<td>African American</td>
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<td>&lt;.0001</td>
<td>0.72</td>
<td>0.66-0.78</td>
<td>-3</td>
</tr>
<tr>
<td>Asian</td>
<td>-0.41</td>
<td>&lt;.0001</td>
<td>0.72</td>
<td>0.59-0.75</td>
<td>-4</td>
</tr>
<tr>
<td>High blood pressure</td>
<td>0.22</td>
<td>&lt;.0001</td>
<td>1.25</td>
<td>1.21-1.28</td>
<td>2</td>
</tr>
<tr>
<td>Coronary artery disease</td>
<td>0.54</td>
<td>&lt;.0001</td>
<td>1.72</td>
<td>1.69-1.76</td>
<td>6</td>
</tr>
<tr>
<td>Family history of AAA</td>
<td>1.34</td>
<td>&lt;.0001</td>
<td>3.80</td>
<td>3.66-3.95</td>
<td>14</td>
</tr>
<tr>
<td>High cholesterol</td>
<td>0.29</td>
<td>&lt;.0001</td>
<td>1.34</td>
<td>1.31-1.37</td>
<td>3</td>
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<tr>
<td>Diabetes</td>
<td>-0.29</td>
<td>&lt;.0001</td>
<td>0.73</td>
<td>0.70-0.77</td>
<td>-3</td>
</tr>
<tr>
<td>Peripheral arterial disease</td>
<td>0.47</td>
<td>&lt;.0001</td>
<td>1.59</td>
<td>1.54-1.65</td>
<td>5</td>
</tr>
<tr>
<td>Cerebrovascular disease</td>
<td>0.41</td>
<td>&lt;.0001</td>
<td>1.51</td>
<td>1.48-1.56</td>
<td>4</td>
</tr>
<tr>
<td>Smoking, packs/day</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>≤10 yrs</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>&lt;0.5</td>
<td>0.96</td>
<td>&lt;.0001</td>
<td>2.61</td>
<td>2.47-2.74</td>
<td>10</td>
</tr>
<tr>
<td>0.5-1</td>
<td>1.16</td>
<td>&lt;.0001</td>
<td>3.19</td>
<td>2.95-3.46</td>
<td>12</td>
</tr>
<tr>
<td>&gt;1</td>
<td>1.16</td>
<td>&lt;.0001</td>
<td>3.20</td>
<td>2.88-3.56</td>
<td>12</td>
</tr>
<tr>
<td>11-20 yrs</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>&lt;0.5</td>
<td>1.58</td>
<td>&lt;.0001</td>
<td>4.87</td>
<td>4.63-5.12</td>
<td>16</td>
</tr>
<tr>
<td>0.5-1</td>
<td>1.76</td>
<td>&lt;.0001</td>
<td>5.79</td>
<td>5.48-6.12</td>
<td>18</td>
</tr>
<tr>
<td>&gt;1</td>
<td>1.79</td>
<td>&lt;.0001</td>
<td>6.00</td>
<td>5.66-6.35</td>
<td>19</td>
</tr>
<tr>
<td>21-35 yrs</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>&lt;0.5</td>
<td>1.99</td>
<td>&lt;.0001</td>
<td>7.29</td>
<td>6.97-7.64</td>
<td>21</td>
</tr>
<tr>
<td>0.5-1</td>
<td>2.08</td>
<td>&lt;.0001</td>
<td>7.99</td>
<td>7.62-8.38</td>
<td>22</td>
</tr>
<tr>
<td>&gt;1</td>
<td>2.13</td>
<td>&lt;.0001</td>
<td>8.41</td>
<td>8.07-9.36</td>
<td>22</td>
</tr>
<tr>
<td>&gt;35 yrs</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>&lt;0.5</td>
<td>2.19</td>
<td>&lt;.0001</td>
<td>8.96</td>
<td>8.57-9.36</td>
<td>23</td>
</tr>
<tr>
<td>0.5-1</td>
<td>2.42</td>
<td>&lt;.0001</td>
<td>11.19</td>
<td>10.76-11.64</td>
<td>25</td>
</tr>
<tr>
<td>&gt;1</td>
<td>2.50</td>
<td>&lt;.0001</td>
<td>12.13</td>
<td>11.66-12.61</td>
<td>26</td>
</tr>
<tr>
<td>Quit smoking</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>&lt;5 yrs ago</td>
<td>-0.14</td>
<td>&lt;.0001</td>
<td>0.87</td>
<td>0.84-0.912</td>
<td>-1</td>
</tr>
<tr>
<td>5-10 yrs ago</td>
<td>-0.39</td>
<td>&lt;.0001</td>
<td>0.68</td>
<td>0.65-0.71</td>
<td>-4</td>
</tr>
<tr>
<td>&gt;10 yrs ago</td>
<td>-0.87</td>
<td>&lt;.0001</td>
<td>0.42</td>
<td>0.41-0.43</td>
<td>-9</td>
</tr>
<tr>
<td>Fruit &amp; veg, ≥3 times/wk</td>
<td>-0.10</td>
<td>&lt;.0001</td>
<td>0.91</td>
<td>0.88-0.92</td>
<td>-1</td>
</tr>
<tr>
<td>Nuts, &gt;3 times/wk</td>
<td>-0.11</td>
<td>&lt;.0001</td>
<td>0.90</td>
<td>0.89-0.93</td>
<td>-1</td>
</tr>
<tr>
<td>Exercise, ≥1 time/wk</td>
<td>-0.15</td>
<td>&lt;.0001</td>
<td>0.86</td>
<td>0.85-0.88</td>
<td>-2</td>
</tr>
<tr>
<td>BMI ≥25 kg/m²</td>
<td>0.18</td>
<td>&lt;.0001</td>
<td>1.20</td>
<td>1.17-1.22</td>
<td>2</td>
</tr>
</tbody>
</table>

RMI, Body mass index; CI, confidence interval; OR, odds ratio.
The model was developed on 50% of the Life Line Screening cohort and validated on the other 50%. The area under the receiver operator curve (ROC) of the model (C statistic) was 0.893. From this model a scoring system was derived as described in materials and methods. The overall accuracy of the scoring system as measured by the C statistic was 0.842.

<table>
<thead>
<tr>
<th>1. Is there an active cardiac condition?</th>
</tr>
</thead>
<tbody>
<tr>
<td>• Unstable coronary syndrome</td>
</tr>
<tr>
<td>• Unstable or severe angina</td>
</tr>
<tr>
<td>• Recent MI (&lt;1 month)</td>
</tr>
<tr>
<td>• Decompensated CHF</td>
</tr>
<tr>
<td>• Significant arrhythmias</td>
</tr>
<tr>
<td>• Severe valvular disease</td>
</tr>
<tr>
<td>Presence cancels or delays aneurysm repair until conditions treated. Implement medical management and consider coronary angiography.</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>2. Does the patient have good functional capacity without symptoms?</th>
</tr>
</thead>
<tbody>
<tr>
<td>• MET ≥4 (see Table III)</td>
</tr>
<tr>
<td>Clinical risk factors:</td>
</tr>
<tr>
<td>• Mild angina pectoris</td>
</tr>
<tr>
<td>• Prior MI</td>
</tr>
<tr>
<td>• Compensated or prior CHF</td>
</tr>
<tr>
<td>• Diabetes mellitus</td>
</tr>
<tr>
<td>• Renal insufficiency</td>
</tr>
<tr>
<td>May proceed with aneurysm repair. In patients with known cardiovascular disease or at least one clinical risk factor, beta blockade is appropriate.</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>3. Is functional capacity poor or unknown?</th>
</tr>
</thead>
<tbody>
<tr>
<td>• MET &lt;4 (see Table III)</td>
</tr>
<tr>
<td>Clinical risk factors:</td>
</tr>
<tr>
<td>• Mild angina pectoris</td>
</tr>
<tr>
<td>• Prior MI</td>
</tr>
<tr>
<td>• Compensated or prior CHF</td>
</tr>
<tr>
<td>• Diabetes mellitus</td>
</tr>
<tr>
<td>• Renal insufficiency</td>
</tr>
<tr>
<td>In patients with three or more clinical risk factors, preoperative non-invasive testing is appropriate if it will change management.</td>
</tr>
</tbody>
</table>

CHF, Congestive heart failure; MET, metabolic equivalent unit; MI, myocardial infarction.

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

*MET,* Metabolic equivalent unit (1 MET = 3.5 mL kg⁻¹ min⁻¹ oxygen uptake).
Table 5. Genetic loci implicated in the pathogenesis of an abdominal aortic aneurysm.


<table>
<thead>
<tr>
<th>Genetic locus</th>
<th>Nearest gene (gene symbol)</th>
<th>SNP rs#</th>
<th>RAF</th>
<th>OR (95% CI)</th>
<th>P-value</th>
<th>Other diseases the locus has been associated with</th>
</tr>
</thead>
<tbody>
<tr>
<td>3p12.3 [40]</td>
<td>Contactin 3 (CNTN3)</td>
<td>rs7635818</td>
<td>0.42</td>
<td>1.33 (1.10–1.21)</td>
<td>0.0028</td>
<td>Numerous; including CHD, IA, cancers and Alzheimer’s disease</td>
</tr>
<tr>
<td>9p21.3q [32]</td>
<td>Cdkn2b antisense RNA 1 (CDKN2BAS1)</td>
<td>rs10757278</td>
<td>0.45</td>
<td>1.31 (1.22–1.42)</td>
<td>1.2 × 10⁻¹²</td>
<td>CHD, pulmonary embolus, PAD</td>
</tr>
<tr>
<td>9p33.1q [41]</td>
<td>Dab2 interacting protein (DAB2IP)</td>
<td>rs7025486</td>
<td>0.25</td>
<td>1.21 (1.14–1.28)</td>
<td>4.6 × 10⁻¹⁰</td>
<td>CHD, pulmonary embolus, PAD</td>
</tr>
<tr>
<td>12q13.3q [42]</td>
<td>Low-density lipoprotein receptor-related protein 1 (LRP1)</td>
<td>rs1466535</td>
<td>0.68</td>
<td>1.15 (1.10–1.21)</td>
<td>4.5 × 10⁻¹⁰</td>
<td></td>
</tr>
</tbody>
</table>

CHD, coronary heart disease; 95% CI, 95% confidence interval; IA, intracranial aneurysm; OR, odds ratio; PAD, peripheral artery disease; RAF, risk allele frequency in population; SNP, single nucleotide polymorphism.

*Replicated in multiple populations.

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Points</th>
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<tbody>
<tr>
<td>Treatment</td>
<td>0</td>
</tr>
<tr>
<td>EVAR</td>
<td>0</td>
</tr>
<tr>
<td>OAR (infra-renal)</td>
<td>2</td>
</tr>
<tr>
<td>OAR (supra-renal)</td>
<td>4</td>
</tr>
<tr>
<td>Aneurysm size, mm</td>
<td>0</td>
</tr>
<tr>
<td>&lt; 65</td>
<td>2</td>
</tr>
<tr>
<td>&gt; 65</td>
<td>0</td>
</tr>
<tr>
<td>Age, years</td>
<td>0</td>
</tr>
<tr>
<td>&lt; 75</td>
<td>0</td>
</tr>
<tr>
<td>&gt; 75</td>
<td>1</td>
</tr>
<tr>
<td>Gender</td>
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<td>Male</td>
<td>0</td>
</tr>
<tr>
<td>Female</td>
<td>1</td>
</tr>
<tr>
<td>Comorbidities</td>
<td>1</td>
</tr>
<tr>
<td>Myocardial disease</td>
<td>1</td>
</tr>
<tr>
<td>Cerebrovascular disease</td>
<td>1</td>
</tr>
<tr>
<td>Chronic obstructive pulmonary disease</td>
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<tr>
<td>Laboratory value</td>
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<tr>
<td>Creatinine, mg/dL</td>
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<tr>
<td>&lt; 1.5</td>
<td>0</td>
</tr>
<tr>
<td>1.5 to &lt; 2</td>
<td>2</td>
</tr>
<tr>
<td>≥ 2</td>
<td>2</td>
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</table>

EVAR, Endovascular aneurysm repair; OAR, open aneurysm repair.

<table>
<thead>
<tr>
<th>Points</th>
<th>Probability of mortality, %</th>
<th>Proposed risk designation</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>0.12</td>
<td>Low-risk group</td>
</tr>
<tr>
<td>1</td>
<td>0.20</td>
<td></td>
</tr>
<tr>
<td>2</td>
<td>0.34</td>
<td></td>
</tr>
<tr>
<td>3</td>
<td>0.59</td>
<td></td>
</tr>
<tr>
<td>4</td>
<td>1.00</td>
<td></td>
</tr>
<tr>
<td>5</td>
<td>1.71</td>
<td>Medium-risk group</td>
</tr>
<tr>
<td>6</td>
<td>2.91</td>
<td></td>
</tr>
<tr>
<td>7</td>
<td>4.90</td>
<td>High-risk group</td>
</tr>
<tr>
<td>8</td>
<td>8.14</td>
<td></td>
</tr>
<tr>
<td>9</td>
<td>13.2%</td>
<td>Prohibitive high-risk group</td>
</tr>
<tr>
<td>10</td>
<td>20.75</td>
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<tr>
<td>11</td>
<td>31.05</td>
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</tr>
<tr>
<td>12</td>
<td>43.63</td>
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<td>13</td>
<td>57.10</td>
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<tr>
<td>14</td>
<td>69.59</td>
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</table>

<table>
<thead>
<tr>
<th>Advantages</th>
<th>Transperitoneal</th>
<th>Retroperitoneal</th>
</tr>
</thead>
<tbody>
<tr>
<td>Most rapid, greatest versatility</td>
<td>• Provides widest access</td>
<td>• Avoids hostile abdomen</td>
</tr>
<tr>
<td>Provides widest access</td>
<td>• Enables evaluation and treatment of concomitant intra-abdominal pathology</td>
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<td>Disadvantages</td>
<td>• Longer postoperative ileus</td>
<td>• Poor access to right renal and iliac arteries</td>
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<td>• Potential for greater fluids loses</td>
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<td>• Difficulty with exposure and control for suprarenal aneurysms</td>
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<td>• Higher incidence of incisional hernia</td>
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<td>Ureteral injury</td>
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Figure 1. The annual adult per capita cigarette consumption and age-adjusted abdominal aortic aneurysm (AAA) deaths per 100,000 white men by year in the United States. AAA: abdominal aortic aneurysm. From Lederle FA. The rise and fall of abdominal aortic aneurysm. Circulation. 2011;124(10):1097-9.
**Figure 3.** Meta-regression of abdominal aortic aneurysm growth rate estimates by aneurysm diameter (please see primary source for individual study citations). The solid line represents the overall regression, the dotted line connects estimates from the same study, and circles have diameters that represent amount of information. Adapted from Thompson SG, Brown LC, Sweeting MJ, Bown MJ, Kim LG, Glover MJ, et al. Systematic review and meta-analysis of the growth and rupture rates of small abdominal aortic aneurysms: implications for surveillance intervals and their cost-effectiveness. Health Technol Assess. 2013;17(41):1-118.
**Figure 4.** Estimated time to have a 10% probability of attaining an aortic diameter of 5.5 cm in male patients (please see primary source for individual study citations). Black diamonds represent 95% confidence intervals and errors represent 95% prediction intervals. MASS: Multicenter Aneurysm Screening Study; PIVOTAL: Positive Impact of endovascular Options for Treating Aneurysm earLy; UKSAT: United Kingdom Small Aneurysm Trial. Adapted from Thompson SG, Brown LC, Sweeting MJ, Bown MJ, Kim LG, Glover MJ, et al. Systematic review and meta-analysis of the growth and rupture rates of small abdominal aortic aneurysms: implications for surveillance intervals and their cost-effectiveness. Health Technol Assess. 2013;17(41):1-118.
Figure 5. Algorithm for management of the patient with a suspected or confirmed ruptured aneurysm.
Figure 6. Referring hospital checklist for the patient with a suspected or confirmed ruptured aneurysm.

- Physician-to-physician phone handoff
- Intravenous peripheral access
- Continuous vital sign monitoring
- Permissive hypotension (to maintain a mental status and target systolic pressure of 70-90 mmHg)
- Transfer of obtained images (either by upload or CD/DVD)
Figure 7. Receiving hospital personnel alert checklist for management of the patient with a suspected or confirmed ruptured aneurysm.
APPENDIX: SEARCH STRATEGY

**Ovid**

**Database(s):** Embase 1988 to 2016 Week 38, Ovid MEDLINE(R) In-Process & Other Non-Indexed Citations and Ovid MEDLINE(R) 1946 to Present, EBM Reviews - Cochrane Central Register of Controlled Trials August 2016, EBM Reviews - Cochrane Database of Systematic Reviews 2005 to September 15, 2016

**Search Strategy:**

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3 TITLE-ABS-KEY((meta W/1 analys*) OR (randomized W/3 study) OR (randomized W/3 trial) OR (randomised W/3 study) OR (randomised W/3 trial) OR "pragmatic clinical trial" OR (doubl* W/1 blind*) OR (doubl* W/1 mask*) OR (singl* W/1 blind*) OR (singl* W/1 mask*) OR (tripl* W/1 blind*) OR (tripl* W/1 mask*) OR (trebl* W/1 blind*) OR (trebl* W/1 mask*) OR "latin square" OR placebo* OR nocebo*)

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The Care of Patients with an Abdominal Aortic Aneurysm: The Society for Vascular Surgery Practice Guidelines

The SVS Document Oversight Committee is requesting your comments on the proposed SVS Clinical Practice Guidelines on “The Care of Patients with an Abdominal Aortic Aneurysm.”

Once you have completed review of the guideline, please use the following link to provide your comments. The open comment period ends on Monday, July 31, 2017.

Click this Link to Provide Your Comments