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

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ABSTRACT

Background: Decision-making related to the care of patients with an abdominal aortic aneurysm (AAA) is complex. Aneurysms present with varying risks of rupture, and patient-specific factors influence anticipated life expectancy, operative risk, and need to intervene. Careful attention to the choice of operative strategy along with optimal treatment of medical comorbidities is critical to achieving excellent outcomes. Moreover, appropriate postoperative surveillance is necessary to minimize subsequent aneurysm-related death or morbidity.

Methods: The committee made specific practice recommendations using the Grading of Recommendations Assessment, Development, and Evaluation system. Three systematic reviews were conducted to support this guideline. Two focused on evaluating the best modalities and optimal frequency for surveillance after endovascular aneurysm repair (EVAR). A third focused on identifying the best available evidence on the diagnosis and management of AAA. Specific areas of focus included (1) general approach to the patient, (2) treatment of the patient with an AAA, (3) anesthetic considerations and perioperative management, (4) postoperative and long-term management, and (5) cost and economic considerations.

Results: Along with providing guidance regarding the management of patients throughout the continuum of care, we have revised a number of prior recommendations and addressed a number of new areas of significance. New guidelines are provided for the surveillance of patients with an AAA, including recommended surveillance imaging at 12-month intervals for patients with an AAA of 4.0 to 4.9 cm in diameter. We recommend endovascular repair as the preferred method of treatment for ruptured aneurysms. Incorporating knowledge gained through the Vascular Quality Initiative and other regional quality collaboratives, we suggest that the Vascular Quality Initiative mortality risk score be used for mutual decision-making with patients considering aneurysm repair. We also suggest that elective EVAR be limited to hospitals with a documented mortality and conversion rate to open surgical repair of 2% or less and that perform at least 10 EVAR cases each year. We also suggest that elective open aneurysm repair be limited to hospitals with a documented mortality of 5% or less and that perform at least 10 open aortic operations of any type each year. To encourage the development of effective systems of care that would lead to improved outcomes for those patients undergoing emergent repair, we suggest a door-to-intervention time of <90 minutes, based on a framework of 30-30-30 minutes, for the management of the patient with a ruptured aneurysm. We recommend treatment of type I and III endoleaks as well as of type II endoleaks with aneurysm expansion but recommend continued surveillance of type II endoleaks not associated with aneurysm expansion. Whereas antibiotic prophylaxis is recommended for patients with an aortic prosthesis before any dental procedure involving the manipulation of the gingival or periapical region of teeth or perforation of the oral mucosa, antibiotic prophylaxis is not recommended before respiratory tract procedures, gastrointestinal or genitourinary procedures, and dermatologic or musculoskeletal procedures unless the potential for infection exists or the patient is immunocompromised. Increased utilization of color duplex ultrasound is suggested for postoperative surveillance after EVAR in the absence of endoleak or aneurysm expansion.

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Conclusions: Important new recommendations are provided for the care of patients with an AAA, including suggestions to improve mutual decision-making between the treating physician and the patients and their families as well as a number of new strategies to enhance perioperative outcomes for patients undergoing elective and emergent repair. Areas of uncertainty are highlighted that would benefit from further investigation in addition to existing limitations in diagnostic tests, pharmacologic agents, intraoperative tools, and devices. (J Vasc Surg 2018;67:2-77.)

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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 (AAA), 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 AAA.

Level of recommendation	1 (Strong)
Quality of evidence	A (High)

Assessment of medical comorbidities. In patients with active cardiac conditions, including unstable angina, decompensated heart failure, severe valvular disease, and significant arrhythmia, we recommend cardiology consultation before endovascular aneurysm repair (EVAR) or open surgical repair (OSR).

Level of recommendation	1 (Strong)
Quality of evidence	B (Moderate)

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

Level of recommendation	2 (Weak)
Quality of evidence	B (Moderate)

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

Level of recommendation	1 (Strong)
Quality of evidence	B (Moderate)

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

Level of recommendation	1 (Strong)
Quality of evidence	A (High)

We suggest coronary revascularization before aneurysm repair in patients with acute ST-segment or non-ST-segment elevation myocardial infarction (MI), unstable angina, or stable angina with left main coronary artery or three-vessel disease.

Level of recommendation	2 (Weak)
Quality of evidence	B (Moderate)

We suggest coronary revascularization before aneurysm repair in patients with stable angina and two-vessel disease that includes the proximal left descending artery and either ischemia on noninvasive stress testing or reduced left ventricular function (ejection fraction $< 50\%$).

Level of recommendation	2 (Weak)
Quality of evidence	B (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 4 to 6 weeks of dual antiplatelet therapy.

Level of recommendation	2 (Weak)
Quality of evidence	B (Moderate)

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	2 (Weak)
Quality of evidence	B (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	2 (Weak)
Quality of evidence	B (Moderate)

In patients with a drug-eluting coronary stent requiring open aneurysm repair, we recommend discontinuation of $P2Y_{12}$ platelet receptor inhibitor therapy 10 days

preoperatively with continuation of aspirin. The $P2Y_{12}$ 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	1 (Strong)
Quality of evidence	B (Moderate)

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

Level of recommendation	2 (Weak)
Quality of evidence	B (Moderate)

If a decision was made to start beta blocker therapy (because of 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	2 (Weak)
Quality of evidence	B (Moderate)

We suggest preoperative pulmonary function studies, including room air arterial blood gas determinations, in patients with a history of symptomatic chronic obstructive pulmonary disease (COPD), long-standing tobacco use, or inability to climb one flight of stairs.

Level of recommendation	2 (Weak)
Quality of evidence	C (Low)

We recommend smoking cessation for at least 2 weeks before aneurysm repair.

Level of recommendation	1 (Strong)
Quality of evidence	C (Low)

We suggest administration of pulmonary bronchodilators for at least 2 weeks before aneurysm repair in patients with a history of COPD or abnormal results of pulmonary function testing.

Level of recommendation	2 (Weak)
Quality of evidence	C (Low)

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

Level of recommendation	2 (Weak)
Quality of evidence	C (Low)

We recommend preoperative hydration in nondialysis-dependent patients with renal insufficiency before aneurysm repair.

Level of recommendation	1 (Strong)
Quality of evidence	A (High)

We recommend preprocedure and postprocedure hydration with normal saline or 5% dextrose/sodium bicarbonate for patients at increased risk of contrast-induced nephropathy (CIN) undergoing EVAR.

Level of recommendation	1 (Strong)
Quality of evidence	A (High)

We recommend holding metformin at the time of administration of contrast material among patients with an estimated glomerular filtration rate (eGFR) of <60 mL/min or up to 48 hours before administration of contrast material if the eGFR is <45 mL/min.

Level of recommendation	1 (Strong)
Quality of evidence	C (Low)

We recommend restarting metformin no sooner than 48 hours after administration of contrast material as long as renal function has remained stable (<25% increase in creatinine concentration above baseline).

Level of recommendation	1 (Strong)
Quality of evidence	C (Low)

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

Level of recommendation	1 (Strong)
Quality of evidence	B (Moderate)

We suggest hematologic assessment if the preoperative platelet count is <150,000/μL.

Level of recommendation	2 (Weak)
Quality of evidence	C (Low)

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

Level of recommendation	1 (Strong)
Quality of evidence	A (High)

We suggest that the maximum aneurysm diameter derived from computed tomography (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 AAAs in men or women 65 to 75 years of age with a history of tobacco use.

Level of recommendation	1 (Strong)
Quality of evidence	A (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 and 75 years of age or in those older than 75 years and in good health.

Level of recommendation	2 (Weak)
Quality of evidence	C (Low)

We suggest a one-time ultrasound screening for AAAs in men or women older than 75 years with a history of tobacco use and in otherwise good health who have not previously received a screening ultrasound examination.

Level of recommendation	2 (Weak)
Quality of evidence	C (Low)

If initial ultrasound screening identified an aortic diameter >2.5 cm but <3 cm, we suggest rescreening after 10 years.

Level of recommendation	2 (Weak)
Quality of evidence	C (Low)

We suggest surveillance imaging at 3-year intervals for patients with an AAA between 3.0 and 3.9 cm.

Level of recommendation	2 (Weak)
Quality of evidence	C (Low)

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

Level of recommendation	2 (Weak)
Quality of evidence	C (Low)

We suggest surveillance imaging at 6-month intervals for patients with an AAA between 5.0 and 5.4 cm in diameter.

Level of recommendation	2 (Weak)
Quality of evidence	C (Low)

We recommend a CT scan to evaluate patients thought 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	1 (Strong)
Quality of evidence	B (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 to be attributed to the aneurysm.

Level of recommendation	1 (Strong)
Quality of evidence	C (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	1 (Strong)
Quality of evidence	A (High)

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

Level of recommendation	2 (Weak)
Quality of evidence	C (Low)

We suggest repair in women with AAA between 5.0 cm and 5.4 cm in maximum diameter.

Level of recommendation	2 (Weak)
Quality of evidence	B (Moderate)

In patients with a small aneurysm (4.0-5.4 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	2 (Weak)
Quality of evidence	C (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	1 (Strong)
Quality of evidence	B (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	2 (Weak)
Quality of evidence	C (Low)

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

Level of recommendation	1 (Strong)
Quality of evidence	B (Moderate)

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

Level of recommendation	1 (Strong)
Quality of evidence	A (High)

Should repair of a symptomatic AAA be delayed to optimize coexisting medical conditions, we recommend that the patient be monitored in an intensive care unit (ICU) setting with blood products available.

Level of recommendation	1 (Strong)
Quality of evidence	C (Low)

Assessment of operative risk and life expectancy. We suggest informing patients contemplating open repair or EVAR of their Vascular Quality Initiative (VQI) perioperative mortality risk score.

Level of recommendation	2 (Weak)
Quality of evidence	C (Low)

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

Level of recommendation	1 (Strong)
Quality of evidence	A (High)

We recommend using Food and Drug Administration (FDA)-approved branch endograft devices in anatomically suitable patients to maintain perfusion to at least one internal iliac artery.

Level of recommendation	1 (Strong)
Quality of evidence	A (High)

We recommend staging bilateral internal iliac artery occlusion by at least 1 to 2 weeks if required for EVAR.

Level of recommendation	1 (Strong)
Quality of evidence	A (High)

We suggest renal artery or superior mesenteric artery (SMA) angioplasty and stenting for selected patients with symptomatic disease before EVAR or OSR.

Level of recommendation	2 (Weak)
Quality of evidence	C (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 inferior mesenteric artery (IMA), which will be sacrificed during the course of treatment.

Level of recommendation	2 (Weak)
Quality of evidence	C (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	2 (Weak)
Quality of evidence	C (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	2 (Weak)
Quality of evidence	C (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 for them to make an informed decision to proceed with aneurysm repair.

Level of recommendation	2 (Weak)
Quality of evidence	C (Low)

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

Level of recommendation	1 (Strong)
Quality of evidence	C (Low)

We suggest a retroperitoneal exposure or a transperitoneal approach with a transverse abdominal incision for patients with significant pulmonary disease requiring OSR.

Level of recommendation	2 (Weak)
Quality of evidence	C (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	1 (Strong)
Quality of evidence	B (Moderate)

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

Level of recommendation	1 (Strong)
Quality of evidence	A (High)

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

Level of recommendation	1 (Strong)
Quality of evidence	A (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	1 (Strong)
Quality of evidence	A (High)

We recommend reimplantation of a patent IMA under circumstances that suggest an increased risk of colonic ischemia.

Level of recommendation	1 (Strong)
Quality of evidence	A (High)

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

Level of recommendation	1 (Strong)
Quality of evidence	A (High)

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

Level of recommendation	2 (Weak)
Quality of evidence	B (Moderate)

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

Level of recommendation	2 (Weak)
Quality of evidence	C (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	2 (Weak)
Quality of evidence	C (Low)

The patient with a ruptured aneurysm. We suggest a door-to-intervention time of <90 minutes, based on a framework of 30-30-30 minutes, 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	1 (Strong)
Quality of evidence	B (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 it is anatomically feasible, we recommend EVAR over open repair for treatment of a ruptured AAA.

Level of recommendation	1 (Strong)
Quality of evidence	C (Low)

Choice of anesthetic technique and agent. We recommend general endotracheal anesthesia for patients undergoing open aneurysm repair.

Level of recommendation	1 (Strong)
Quality of evidence	A (High)

Antibiotic prophylaxis. We recommend intravenous administration of a first-generation cephalosporin or, in the event of penicillin allergy, vancomycin within 30 minutes before OSR or EVAR. Prophylactic antibiotics should be continued for no more than 24 hours.

Level of recommendation	1 (Strong)
Quality of evidence	A (High)

We recommend that any potential sources of dental sepsis be eliminated at least 2 weeks before implantation of 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	1 (Strong)
Quality of evidence	B (Moderate)

If the intraoperative hemoglobin level is <10 g/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	1 (Strong)
Quality of evidence	B (Moderate)

Cardiovascular monitoring. We suggest using pulmonary artery catheters only if the likelihood of a major hemodynamic disturbance is high.

Level of recommendation	1 (Strong)
Quality of evidence	B (Moderate)

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

Level of recommendation	1 (Strong)
Quality of evidence	B (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	1 (Strong)
Quality of evidence	B (Moderate)

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

Levels of recommendation	1 (Strong)
Quality of evidence	A (High)

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

Levels of recommendation	1 (Strong)
Quality of evidence	A (High)

Role of the ICU. We recommend postoperative management in an ICU 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	1 (Strong)
Quality of evidence	A (High)

Nasogastric decompression and perioperative nutrition. We recommend optimization of preoperative nutritional status before elective open aneurysm repair if repair will not be unduly delayed.

Level of recommendation	1 (Strong)
Quality of evidence	A (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	1 (Strong)
Quality of evidence	A (High)

We recommend parenteral nutrition if a patient is unable to tolerate enteral support 7 days after aneurysm repair.

Level of recommendation	1 (Strong)
Quality of evidence	A (High)

Prophylaxis for deep venous thrombosis. We recommend thromboprophylaxis that includes intermittent pneumatic compression and early ambulation for all patients undergoing OSR or EVAR.

Level of recommendation	1 (Strong)
Quality of evidence	A (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	2 (Weak)
Quality of evidence	C (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 g/dL or below.

Level of recommendation	2 (Weak)
Quality of evidence	C (Low)

Perioperative pain management. We recommend multimodality treatment, including epidural analgesia, for postoperative pain control after OSR of an AAA.

Level of recommendation	1 (Strong)
Quality of evidence	A (High)

Late outcomes. We recommend treatment of type I endoleaks.

Level of recommendation	1 (Strong)
Quality of evidence	B (Moderate)

We suggest treatment of type II endoleaks associated with aneurysm expansion.

Level of recommendation	2 (Weak)
Quality of evidence	C (Low)

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

Level of recommendation	1 (Strong)
Quality of evidence	B (Moderate)

We recommend treatment of type III endoleaks.

Level of recommendation	1 (Strong)
Quality of evidence	B (Moderate)

We suggest no treatment of type IV endoleaks.

Level of recommendation	2 (Weak)
Quality of evidence	C (Low)

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

Level of recommendation	1 (Strong)
Quality of evidence	B (Moderate)

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

Level of recommendation	2 (Weak)
Quality of evidence	C (Low)

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

Level of recommendation	2 (Weak)
Quality of evidence	C (Low)

We recommend that follow-up of patients after aneurysm repair include a thorough lower extremity pulse examination or ankle-brachial index (ABI).

Level of recommendation	1 (Strong)
Quality of evidence	B (Moderate)

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

Level of recommendation	1 (Strong)
Quality of evidence	A (High)

We recommend antibiotic prophylaxis to prevent graft infection before 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 OSR or EVAR.

Level of recommendation	1 (Strong)
Quality of evidence	B (Moderate)

We suggest antibiotic prophylaxis before respiratory tract procedures, gastrointestinal or genitourinary

procedures, and dermatologic or musculoskeletal procedures for any patient with an aortic prosthesis if the potential for infection exists or the patient is immunocompromised.

Level of recommendation	2 (Weak)
Quality of evidence	C (Low)

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	1 (Strong)
Quality of evidence	A (High)

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

Level of recommendation	1 (Strong)
Quality of evidence	A (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	1 (Strong)
Quality of evidence	B (Moderate)

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

Level of recommendation	2 (Weak)
Quality of evidence	B (Moderate)

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

Level of recommendation	2 (Weak)
Quality of evidence	B (Moderate)

In unstable patients with infected graft, we recommend in situ reconstruction with a silver- or antibiotic-impregnated graft, cryopreserved allograft, or polytetrafluoroethylene (PTFE) graft.

Level of recommendation	1 (Strong)
Quality of evidence	B (Moderate)

Recommendation for postoperative surveillance. We recommend baseline imaging in the first month after EVAR with contrast-enhanced CT and color duplex ultrasound imaging. In the absence of an endoleak or sac enlargement, imaging should be repeated in 12 months using contrast-enhanced CT or color duplex ultrasound imaging.

Level of recommendation	1 (Strong)
Quality of evidence	B (Moderate)

If a type II endoleak is observed 1 month after EVAR, we suggest postoperative surveillance with contrast-enhanced CT and color duplex ultrasound imaging at 6 months.

Level of recommendation	2 (Weak)
Quality of evidence	B (Moderate)

If neither endoleak nor AAA enlargement is observed 1 year after EVAR, we suggest color duplex ultrasound when feasible, or CT imaging if ultrasound is not possible, for annual surveillance.

Level of recommendation	2 (Weak)
Quality of evidence	C (Low)

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

Level of recommendation	2 (Weak)
Quality of evidence	C (Low)

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

Level of recommendation	2 (Weak)
Quality of evidence	C (Low)

We suggest noncontrast-enhanced CT imaging of the entire aorta at 5-year intervals after open repair or EVAR.

Level of recommendation	2 (Weak)
Quality of evidence	C (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[1] and subsequently updated in 2009,[2] for surgeons and physicians who are involved in the preoperative, operative, and postoperative care of patients with AAAs. This document provides recommendations for evaluating the patient (including risk of aneurysm rupture and associated medical comorbidities), 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 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 comorbidities is critical to achieving excellent outcomes. Moreover, appropriate postoperative surveillance of the patient and timely intervention in the case of a late complication are 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 to generate a concise set of recommendations. The strength of any given recommendation and the quality of evidence were graded on the basis of 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 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, because of low-quality evidence or because high-quality evidence suggests that benefits and risks are closely balanced, a **weak recommendation** was recorded. Guideline developers used the term *we recommend* to denote strong recommendations, whereas for weak recommendations, they used the less definitive wording *we suggest*. Thus, **quality of evidence was rated high** when additional research is considered very unlikely to change confidence in the estimate of effect, **moderate** when further research is likely to have an important impact on 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 only by 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 surveillance after EVAR. 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 19, 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 AAA. The actual strategy is available in the Appendix (online only). This search yielded 1206 references, from which 29 evidence synthesis reports (systematic reviews and meta-analyses) were used to grade the quality of evidence (Table I) in various topics that relate to AAA, such as screening, diagnosis, surveillance, and treatment.

GENERAL APPROACH TO THE PATIENT

History and risk factors for AAA

The risk of an AAA as well as of aneurysm enlargement and rupture for each patient and related family members can largely be 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 >3 million men and women, from diverse racial, ethnic, and socioeconomic backgrounds.[33] 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 AAA, with a significant increase in risk for the ages of 65 to 69 years (odds ratio [OR], 5.4) and 75 to 79 years (OR, 14.5).

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

Systematic reviews	Question/comparison	Findings (quality of evidence)
Screening, diagnosis, and preoperative surveillance		
Guirguis-Blake, 2014[4] Cosford, 2011[5] Takaji, 2010[6]	Effectiveness of screening for AAA	• Screening (primarily in men >65 years) was associated with reduction in AAA mortality (high); absolute reduction: 4 per 1000; number needed to screen: 238

Alamoudi, 2015[7]
Concannon, 2014[8]
	Diagnostic accuracy of imaging for AAA compared with digital subtraction angiography	• The mean reported sensitivities and specificities were as follows:
		  • 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

Sweeting, 2012[9]	Factors affecting growth and rupture of small AAA	• Rupture was higher in women, in smokers, and with elevated blood pressure (moderate)
RESCAN Collaborators, 2013[10]	Surveillance intervals for small AAA	• For each 0.5-cm increase in AAA diameter, growth rates increased on average by 0.59 mm/y and rupture rates increased by a factor of 1.91 (moderate)
Treatment		
Stather, 2013[11] Coughlin, 2013[12]	Open vs endovascular repair	• EVAR had lower 30-day or in-hospital mortality rate (high) • Reduction in quality of life at 3 months was more pronounced with open repair • At 2 and 4 years, no difference in mortality (low) • EVAR required more reinterventions and was associated with increased late rupture (high)
Biancari, 2011[13]	Open vs endovascular repair (age >80 years)	• Elective EVAR was associated with lower immediate postoperative mortality and morbidity (low, observational data)
Kontopodis, 2015[14]	Open vs endovascular repair (age <70 years)	• EVAR was associated with a decreased risk of 30-day mortality and 30-day morbidity and shorter length of hospitalization (moderate); long-term survival and the need for reintervention were similar (low)
Saedon, 2015[15]	Open vs endovascular repair (obese patients)	• EVAR had lower 30-day postoperative mortality and fewer early postoperative complications (MI, chest infection, renal failure, wound infection); risks of postoperative bowel ischemia and stroke were similar (low)
Rayet, 2008[16] Sweeting, 2015[17] Antoniou, 2013[18] Antoniou, 2015[19] Badger, 2016[20] Li, 2016[21] Luebke, 2015[22]	Open vs endovascular repair (ruptured AAA)	• EVAR had lower mortality in 31 studies that was insignificant in pooled analysis of three recent trials or in an adjusted analysis (low)
Ma, 2016[23]	Transperitoneal vs retroperitoneal approach for elective open AAA repair	• 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

(Continued on next page)

Table I. Continued.

Systematic reviews	Question/comparison	Findings (quality of evidence)
Twine, 2013[24]	Retroperitoneal vs transperitoneal approach to the infrarenal abdominal aorta	• Retroperitoneal approach is associated with lower rates of postoperative ileus and pneumonia (moderate)
Jackson, 2014[25]	Totally percutaneous vs standard femoral artery access for elective bifurcated abdominal EVAR	• One small, highly imprecise study (low)
BaniHani, 2011[26]	Interventions for preventing venous thromboembolism after abdominal aortic surgery	• The body of direct evidence is insufficient (two small studies with methodologic limitations) • Extrapolation from indirect evidence is required
Twine, 2011[27]	Effects of statins on AAA	• Reduction in mortality (moderate) • No change in expansion (low)
Bergqvist, 2011[28]	Pharmacologic interventions to attenuate the expansion of AAA	• No consistent pattern of pharmacologic influence on expansion rate (low)
Pieper, 2013[29]	Surgical outcomes and hospital volume	• Lower mortality for elective and ruptured AAA repair in high-volume hospitals (low)
Postoperative surveillance		
Habets, 2013[30]	Magnetic resonance imaging vs CTA for the detection of endoleaks after EVAR for AAA	• Magnetic resonance imaging was more sensitive for type II endoleaks (moderate)
Karthikesalingam, 2012[31]	Diagnostic accuracy of DUS and contrast-enhanced ultrasound for types I and III endoleak	• Both DUS and contrast-enhanced ultrasound were highly specific for types I and III endoleaks (moderate) • Sensitivity estimates were likely similar but less reliable
Antoniou, 2015[32]	Late rupture of AAA after EVAR	• Graft-related endoleaks were the predominant cause of late aneurysm rupture

AAA, Abdominal aortic aneurysm; *CTA*, computed tomography angiography; *DUS*, duplex ultrasound; *EVAR*, endovascular aneurysm repair; *ICU*, intensive care unit; *MI*, myocardial infarction; *MRA*, magnetic resonance angiography.

Consistent with prior estimates,[34-38] an AAA was more likely among men (OR, 5.7) and less common in Hispanics (OR, 0.7), African Americans (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 AAA (OR, 2.6), which increased in a dose-dependent manner such that smoking more than one pack per day for >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 three 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.[39-41] Increasing risk has also been noted with increased salt intake,[42] high blood pressure, concomitant peripheral arterial disease and cerebrovascular disease, and family history of AAA (Table II).[33]

Given the prevalence of AAA-related risk factors in the United States, the prevalence of AAA, as defined by an aortic diameter >3 cm, was estimated at 1.4% among those between 50 and 84 years old, or 1.1 million adults. Importantly, these findings largely concur with and expand on prior prevalence and association estimates derived from more homogeneous populations, such as male military veterans.[43] First-degree relatives of patients with an AAA have an approximately 20% likelihood for development of an AAA.[44,45]

The association between cigarette smoking and AAA disease deserves special emphasis.[46] More than 90% of patients with AAA 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.[47] For patients with early aneurysmal disease, a recent meta-analysis concluded that smoking increased the rate of aneurysm enlargement by 35%.[9] Current smokers are more than 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 for development of an aneurysm by 4%.[48] The decades-long decline in per

Table II. Risk factors for the development of an abdominal aortic aneurysm (*AAA*)

Variable	Estimate	*P*	OR	95% CI	Score
Male (vs female)	1.74	<.0001	5.71	5.57-5.85	18
Age, years (vs <55 years)					
55-59	1.01	<.0001	2.76	2.55-3.00	11
60-64	1.68	<.0001	5.35	4.97-5.76	17
65-69	2.24	<.0001	9.41	8.76-10.12	23
70-74	2.67	<.0001	14.46	13.45-15.55	*28*
75-79	3.02	<.0001	20.43	18.99-21.99	31
80-84	3.35	<.0001	28.37	26.31-30.59	35
Race/ethnicity (vs white)					
Hispanic	−0.37	<.0001	0.69	0.62-0.77	−4
African American	−0.33	<.0001	0.72	0.66-0.78	−3
Asian	−0.41	<.0001	0.72	0.59-0.75	−4
High blood pressure	0.22	<.0001	1.25	1.21-1.28	2
Coronary artery disease	0.54	<.0001	1.72	1.69-1.76	6
Family history of AAA	1.34	<.0001	3.80	3.66-3.95	14
High cholesterol	0.29	<.0001	1.34	1.31-1.37	3
Diabetes	−0.29	<.0001	0.75	0.73-0.77	−3
Peripheral arterial disease	0.47	<.0001	1.59	1.54-1.65	5
Carotid disease	0.41	<.0001	1.51	1.46-1.56	4
Cerebrovascular history	0.16	<.0001	1.18	1.14-1.21	2
Smoking, packs/day					
≤10 years					
<0.5	0.96	<.0001	2.61	2.47-2.74	10
0.5-1	1.16	<.0001	3.19	2.93-3.46	12
>1	1.16	<.0001	3.20	2.88-3.56	12
11-20 years					
<0.5	1.58	<.0001	4.87	4.63-5.12	16
0.5-1	1.76	<.0001	5.79	5.48-6.12	18
>1	1.79	<.0001	6.00	5.66-6.35	19
21-35 years					
<0.5	1.99	<.0001	7.29	6.97-7.64	21
0.5-1	2.08	<.0001	7.99	7.62-8.38	22
>1	2.13	<.0001	8.41	8.57-9.36	22
>35 years					
<0.5	2.19	<.0001	8.96	8.57-9.36	23
0.5-1	2.42	<.0001	11.19	10.76-11.64	25
>1	2.50	<.0001	12.13	11.66-12.61	26
Quit smoking					
<5 years ago	−0.14	<.0001	0.87	0.84-0.912	−1
5-10 years ago	−0.39	<.0001	0.68	0.65-0.71	−4
> 10 years ago	−0.87	<.0001	0.42	0.41-0.43	−9
Fruits and vegetables, >3 times/week	−0.10	<.0001	0.91	0.88-0.92	−1
Nuts, >3 times/week	−0.11	<.0001	0.90	0.89-0.93	−1
Exercise, ≥1 time/week	−0.15	<0001	0.86	0.85-0.88	−2
BMI ≥25 kg/m^2	0.18	<0001	1.20	1.17-1.22	2

BMI, 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 operating characteristic curve of the model (C statistic) was 0.893. From this model, a scoring system was derived. The overall accuracy of the scoring system as measured by the C statistic was 0.842.
From Kent KC, Zwolak RM, Egorova NN, Riles TS, Manganaro A, Moskowitz AJ, et al. Analysis of risk factors for abdominal aortic aneurysm in a cohort of more than 3 million individuals. J Vasc Surg 2010;52:539-48.

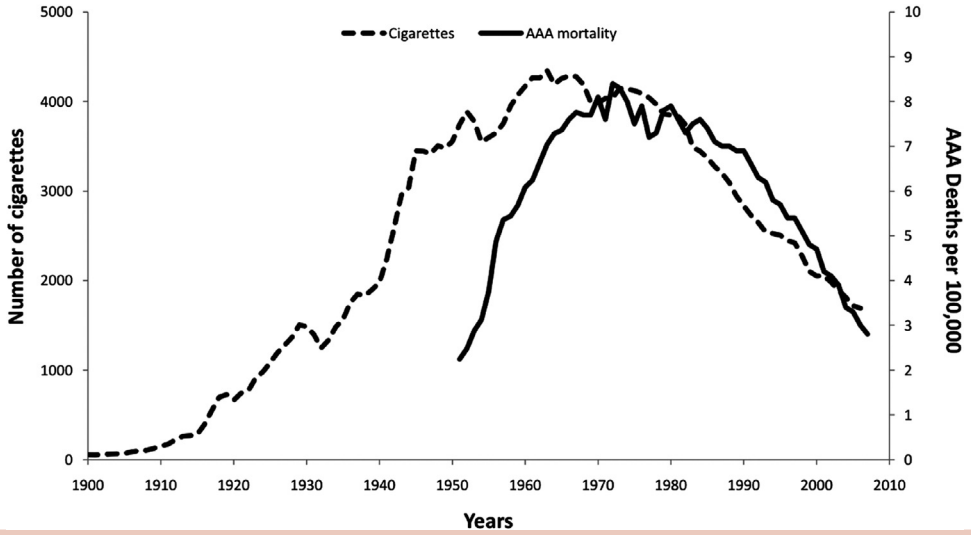

Fig 1. The annual adult per capita cigarette consumption and age-adjusted abdominal aortic aneurysm (*AAA*) deaths per 100,000 white men by year in the United States. (From Lederle FA. The rise and fall of abdominal aortic aneurysm. Circulation 2011;124:1097-9.).

capita cigarette consumption in American adults has been paralleled by a similar decline in deaths from ruptured AAA (Fig 1).[49]

Estimates of the incidence of death from ruptured AAA have declined by >50% in the last 20 years, probably because of 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.[50-52] However, in countries where cigarette consumption remains high or is increasing, aneurysm-related mortality continues to increase.[53] 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 AAA.[54-56]

Risk factors for rupture are also relevant in evaluating and managing patients with a known or suspected AAA. In the UK Small Aneurysm Trial (UKSAT), 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 1 second, current smoking history, and elevated mean blood pressure.[57,58] Multiple studies have suggested that women are at greater risk for rupture,[59,60] as are patients receiving immunomodulatory therapy after major organ transplantation.[61-63] Women who smoke are at high risk for an AAA. In a recent Swedish population study, women with a history of smoking of >20 pack-years were nearly twice as likely to develop AAA as men with a similar smoking history.[64] However, the risk of AAA after smoking cessation declines more rapidly in women than in men.[64] Increased aortic mural calcification has also been suggested as a risk factor for rupture.[65]

Rupture risk for those unfit for repair in the Aneurysm Detection and Management (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 ≥7.0 cm.[66] 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 may be as low as 5.3% per year for AAAs between 5.5 and 7.0 cm in diameter and 6.3% per year for AAAs >7.0 cm. 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.[67]

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 after prior OSR of an AAA, the presence of an anastomotic pseudoaneurysm,[68] iliac artery aneurysm,[69] or suprarenal aortic aneurysm[70] should be considered. Abdominal or back pain after EVAR should also prompt evaluation of potential aneurysm expansion or rupture.[71-73]

Physical examination

An AAA 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 twelfth thoracic vertebra, and lies in the retroperitoneum just anterior to 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. At the level of the umbilicus and fourth lumbar vertebra, the aorta bifurcates into the right and left common iliac arteries. The focused examination 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.[74] 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.[75] Palpation does not precipitate rupture, and the concern for a symptomatic aneurysm should not preclude thorough examination. An abdominal aneurysm is common (37%-40%) in patients with popliteal artery aneurysms,[76-78] as are concurrent distal arterial aneurysms in patients with an AAA.[79-81]

In patients with a suspected or known AAA, 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 AAA.	
Level of recommendation	1 (Strong)
Quality of evidence	A (High)

Assessment of medical comorbidities

Preoperative evaluation of cardiac risk. Despite improvements in cardiovascular risk factor management, 5-year survival after successful aneurysm repair remains below 70%.[82-84] Cardiovascular and pulmonary disease remain the leading causes of early and late death after OSR or EVAR.[85] EVAR is associated with a threefold reduction in perioperative mortality compared with propensity-matched patients undergoing elective OSR,[86] including even younger patients with fewer comorbidities.[87,88] For patients with advanced chronic renal insufficiency[89] and oxygen-dependent COPD,[90] EVAR outcomes are superior to those achieved with contemporary OSR, particularly when it is performed under local or regional anesthesia. However, despite the reduced risk compared with OSR, EVAR remains an intermediate- to high-risk procedure for cardiovascular complication.

Given the risk associated with either OSR or EVAR, it is essential to evaluate the overall operative risk associated with either method of repair. The first step should be to determine whether an active cardiovascular condition exists (Table III), which would mandate further assessment and management before planned aneurysm repair. In the absence of an active 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 IV). Patients capable of moderate physical activities, such as climbing two flights of stairs or running a short distance (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.[91] 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 of early and late death after aortic aneurysm repair.[92-94]

All patients should be evaluated with a 12-lead ECG within 30 days of planned repair. Although resting left ventricular 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, and significant arrhythmia, we recommend cardiology consultation before EVAR or OSR.	
Level of recommendation	1 (Strong)
Quality of evidence	B (Moderate)
In patients with significant clinical risk factors, such as coronary artery disease, congestive heart failure, cerebrovascular disease, diabetes mellitus, chronic renal insufficiency, and unknown or poor functional capacity (MET < 4), who are to undergo OSR or EVAR, we suggest noninvasive stress testing.	
Level of recommendation	2 (Weak)
Quality of evidence	B (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	1 (Strong)
Quality of evidence	B (Moderate)
We recommend echocardiography before planned operative repair in patients with dyspnea of unknown origin or worsening dyspnea.	
Level of recommendation	1 (Strong)
Quality of evidence	A (High)

Table III. Preoperative cardiac evaluation for the patient undergoing aneurysm repair

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

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

CHF, Congestive heart failure; *MET,* metabolic equivalent; *MI,* myocardial infarction.
From Chaikof EL, Brewster DC, Dalman RL, Makaroun MS, Illig KA, Sicard GA, et al. The care of patients with an abdominal aortic aneurysm: the Society for Vascular Surgery practice guidelines. J Vasc Surg 2009;50(Suppl):S2-49; originally adapted from Fleisher LA, Beckman JA, Brown KA, Calkins H, Chaikof E, Fleischmann KE, et al. ACC/AHA 2007 guidelines on perioperative cardiovascular evaluation and care for noncardiac surgery: executive summary. Circulation 2007;116:1971-96.

Table IV. Functional capacity estimation from an assessment of physical activity

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

METs, Metabolic equivalents (1 MET = 3.5 mL kg^{-1} min^{-1} oxygen uptake).
From Chaikof EL, Brewster DC, Dalman RL, Makaroun MS, Illig KA, Sicard GA, et al. The care of patients with an abdominal aortic aneurysm: the Society for Vascular Surgery practice guidelines. J Vasc Surg 2009;50(Suppl):S2-49; originally adapted from Hlatky MA, Boineau RE, Higginbotham MB, Lee KL, Mark DB, Califf RM, et al. A brief self-administered questionnaire to determine functional capacity (the Duke Activity Status Index). Am J Cardiol 1989;64:651-4.

Preoperative coronary revascularization. A meta-analysis of 22 studies examining >13,000 patients with coronary artery disease identified an AAA in 8.4% of patients.[95] 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.[96,97] Coronary revascularization is indicated for acute coronary syndrome with or without ST-segment

elevation, unstable angina, and 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 noninvasive testing or reduced left ventricular function.

The risk for perioperative stent thrombosis for both bare-metal stents and drug-eluting stents 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 bare-metal stent if dual antiplatelet therapy cannot be continued through the perioperative period. Likewise, OSR should not be performed within 6 months after implantation of a drug-eluting stent if cessation of dual antiplatelet therapy is required.[98] 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 with use of 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.[91] Whereas simultaneous open aneurysm repair and coronary artery bypass grafting has been reported for select symptomatic patients with critical coronary artery disease,[99-102] if it is anatomically feasible, EVAR under local anesthesia would be a preferred option.

We suggest coronary revascularization before aneurysm repair in patients with acute ST-segment or non-ST-segment elevation MI, unstable angina, or stable angina with left main coronary artery or three-vessel disease.	
Level of recommendation	2 (Weak)
Quality of evidence	B (Moderate)
We suggest coronary revascularization before aneurysm repair in patients with stable angina and two-vessel disease that includes the proximal left descending artery and either ischemia on noninvasive stress testing or reduced left ventricular function (ejection fraction $< 50\%$).	
Level of recommendation	2 (Weak)
Quality of evidence	B (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 4 to 6 weeks of dual antiplatelet therapy.	
Level of recommendation	2 (Weak)
Quality of evidence	B (Moderate)
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	2 (Weak)
Quality of evidence	B (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	2 (Weak)
Quality of evidence	B (Moderate)
In patients with a drug-eluting coronary stent requiring open aneurysm repair, we recommend discontinuation of $P2Y_{12}$ platelet receptor inhibitor therapy 10 days preoperatively with continuation of aspirin. The $P2Y_{12}$ 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	1 (Strong)
Quality of evidence	B (Moderate)

Perioperative medical management of coronary artery disease. The initiation of beta blockade before noncardiac surgery has been associated with an increased risk of stroke and all-cause mortality.[103-105] The use of an α_2 agonist is no longer recommended to prevent cardiac events, nor is that of calcium channel blockers, such as diltiazem and verapamil, given their potential to impair myocardial function in patients with reduced left ventricular function. Continuation of ACE inhibitors and angiotensin receptor blockers is based on individual clinical circumstances.[91]

Aspirin reduces adverse cardiovascular events among patients with coronary artery disease and can be continued during the perioperative period.[106] Both warfarin and the new oral anticoagulants (nonvitamin K antagonist oral anticoagulants) should be discontinued at least 5 days and 2 days, respectively, before major surgery.[107] 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 it is part of an established medical regimen.	
Level of recommendation	2 (Weak)
Quality of evidence	B (Moderate)
If a decision was made to start beta blocker therapy (because of 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	2 (Weak)
Quality of evidence	B (Moderate)

Pulmonary disease. Between 7% and 11% of patients with COPD have an AAA.[57] The prevalence of COPD in patients presenting with ruptured AAA has largely been attributed to cigarette smoking as a common risk

factor.[59] Common genetic, inflammatory, and remodeling pathways that predispose patients to both conditions may also be present.[108] Several studies have reported that COPD is an independent predictor of mortality after open repair,[34,109] with the severity of pulmonary disease and the capacity to optimize preoperative respiratory function influencing outcome.[110] EVAR is better tolerated than OSR, particularly if EVAR is performed under local anesthesia.[111,112] However, patients with severe COPD exhibit increased in-hospital mortality, pulmonary complications, major adverse events, and decreased 5-year survival whether they are treated with open repair or EVAR.[90]

If COPD is suspected or present, room air arterial blood gas determinations and standard pulmonary function testing should be performed before surgery. In the setting of oxygen-dependent COPD, pulmonary consultation should be obtained for assessment of prognosis and optimization of medical therapy. Smoking cessation before aneurysm repair and administration of pulmonary bronchodilators for at least 2 weeks are recommended. The diagnosis of an aortic aneurysm can be a strong motivator for smoking cessation,[113] and efforts to begin smoking cessation before surgery can result in long-term benefits.[114] Nicotine replacement[115] and use of nortriptyline and bupropion, alone or in combination, along with inpatient and outpatient counseling have proven beneficial for smoking cessation.[116]

We suggest preoperative pulmonary function studies, including room air arterial blood gas determinations, in patients with a history of symptomatic COPD, long-standing tobacco use, or inability to climb one flight of stairs.	
Level of recommendation	2 (Weak)
Quality of evidence	C (Low)
We recommend smoking cessation for at least two weeks prior to aneurysm repair.	
Level of recommendation	1 (Strong)
Quality of evidence	C (Low)
We suggest administration of pulmonary bronchodilators for at least 2 weeks before aneurysm repair in patients with a history of COPD or abnormal results of pulmonary function testing.	
Level of recommendation	2 (Weak)
Quality of evidence	C (Low)

Renal insufficiency. Preoperative renal insufficiency is an established risk factor for poor outcome after aneurysm repair. Among patients with moderate renal dysfunction (eGFR of 30-60 mL/min), mortality and cardiovascular events are more likely for patients treated by OSR than by EVAR.[89] 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 3 years.[117] Median survival was 2 years.[117]

Significant declines in renal mass and eGFR have been documented after OSR and EVAR, even in the setting of age-adjusted normal renal function before surgery.[118] For example, acute kidney injury and chronic kidney injury have been noted after complex EVAR with snorkel or renal stent placement, with an increased risk among women.[119] Even transient postoperative renal dysfunction is associated with an increase in mortality, morbidity, and the need for additional ICU support.[120]

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.[121] Similarly, given the association of ACE 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 euvolemic.[122,123] Whereas the administration of many agents has been evaluated, none have proved of value in limiting renal injury after AAA repair. Antioxidants, such as mannitol, before or during OSR have demonstrated no benefit.[124] Likewise, fenoldopam, dopamine, atrial natriuretic peptide, diuretics, and antiplatelet and anti-inflammatory agents are of no value in the prevention or treatment of acute kidney injury.[125] Last, remote ischemic preconditioning has been studied as a strategy for reducing the risk of renal dysfunction. However, systematic review and meta-analysis of the current literature do not confirm the efficacy of this technique in patients undergoing major vascular surgery.[126-129]

CIN is defined as a 25% increase in serum creatinine concentration or an absolute increase of 0.5 mg/dL 2 to 7 days after administration of contrast material. Patients with renal disease (eGFR $\leq$ 45 mL/min/1.73 m^2), diabetes, congestive heart failure, ejection fraction $<$40%, hypertension, anemia, advanced age, proteinuria, and gout are at increased risk for CIN.[130] Gadolinium is not a safe alternative to iodinated contrast agents, given the risk for nephrogenic systemic fibrosis in patients with a GFR of $<$30 mL/min/1.73 m^2.

There is a linear relationship between the volume of contrast material administered and the onset and severity of CIN. For every 100 mL of contrast material infused during coronary artery interventions, there is a 12% increase in the risk for CIN.[131] Preprocedural hydration may be beneficial, but fenoldopam, dopamine, atrial natriuretic peptide, theophylline, and calcium channel blockers are not.[132] Preprocedural 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.[133] Recent evidence suggests that statin therapy may be of benefit in preventing CIN. For example, two studies

suggest that initiating high-dose statin therapy 2 days before exposure to contrast material and continuing for 3 days afterward may reduce the risk for CIN in patients undergoing coronary interventions.[134,135] 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 compared with the use of *N*-acetylcysteine and saline alone.[136]

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 iodixanol (Visipaque, an iso-osmolar agent; 290 mOsm/kg).[137] However, rates of CIN for iopamidol (Isovue-370, 796 mOsm/kg, nonionic) are similar to those for iodixanol, which suggests that other physiochemical properties, apart from osmolarity, are important determinants of CIN.[138] Likewise, several randomized trials of ionic and nonionic contrast agents have demonstrated no difference in CIN.[139]

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 administration of contrast material. Patients at increased risk for CIN should be hydrated before and after EVAR. Normal saline at 1 mL/kg/h can be administered for 6 to 12 hours before and after the procedure. Alternatively, 5% dextrose/sodium bicarbonate can be administered at 3 mL/kg/h for 1 hour before EVAR and at 1 mL/kg/h for 6 hours afterward. Periprocedural *N*-acetylcysteine may be of benefit to reduce CIN. Additional strategies to limit contrast agent load include use of carbon dioxide,[140] intravascular or duplex ultrasound,[141] and fusion imaging.[142]

We suggest holding ACE inhibitors and angiotensin receptor antagonists on the morning of surgery and restarting these agents after the procedure, once euvolemia has been achieved.	
Level of recommendation	2 (Weak)
Quality of evidence	C (Low)
We recommend preoperative hydration in nondialysis-dependent patients with renal insufficiency before aneurysm repair.	
Level of recommendation	1 (Strong)
Quality of evidence	A (High)
We recommend preprocedure and postprocedure hydration with normal saline or 5% dextrose/sodium bicarbonate for patients at increased risk of CIN undergoing EVAR.	
Level of recommendation	1 (Strong)
Quality of evidence	A (High)

Diabetes mellitus. Diabetic patients have increased operative mortality after AAA repair, with reduced survival 2 to 5 years after surgery, consistent with an increased burden of cardiovascular disease.[143] However, whether diabetes is a distinct risk factor for major adverse events or death after OSR or EVAR is not well defined.[144-148]

Metformin is a first-line medication for the treatment of type 2 diabetes and prescribed for >100 million patients worldwide. Metformin is contraindicated if the eGFR is below 30 mL/min/1.73 m^2 because of the risk of lactic acidosis, which carries a mortality of up to 50%.[149] 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 <60 mL/min should prompt the cessation of metformin either at the time of administration of contrast material or up to 48 hours before if eGFR is <45 mL/min.[150] Metformin should be restarted no sooner than 48 hours after administration of contrast material as long as renal function has remained stable (<25% increase in creatinine concentration above baseline).[150]

We recommend holding metformin at the time of administration of contrast material among patients with an eGFR of <60 mL/min or up to 48 hours before administration of contrast material if the eGFR is <45 mL/min.	
Level of recommendation	1 (Strong)
Quality of evidence	C (Low)
We recommend restarting metformin no sooner than 48 hours after administration of contrast material as long as renal function has remained stable (<25% increase in creatinine concentration above baseline).	
Level of recommendation	1 (Strong)
Quality of evidence	C (Low)

Hematologic disorders. The presence of an aortic aneurysm influences both platelet count and function. Low platelet counts and high glycocalicin levels have been observed in patients with an AAA, which has been attributed to increased platelet destruction within the aneurysm sac.[151] Whereas 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 <150,000 platelets/μL. After elective OSR, a platelet count of <130,000/μL is associated with an increased risk of bleeding.[152] 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.[153] Matsumura and colleagues suggested that a lower preoperative platelet count is an independent predictor of 2-year mortality among patients undergoing both EVAR and OSR.[154]

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.[155-157] Anticardiolipin antibodies, 5,10 methylenetetrahydrofolate reductase 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 <7 g/dL.	
Level of recommendation	1 (Strong)
Quality of evidence	B (Moderate)
We suggest hematologic assessment if the preoperative platelet count is <150,000/μL.	
Level of recommendation	2 (Weak)
Quality of evidence	C (Low)

Biomarkers and heritable risks for an AAA

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 of the response to medical or surgical therapy. Among biomarkers evaluated to date, fibrinogen, D-dimer, and interleukin 6 have been consistently associated with the presence of AAA in multiple cross-sectional, case-controlled studies.[158] A meta-analysis has reported that fibrinogen, D-dimer, and thrombin-antithrombin III complex levels are increased in patients with AAA.[159] Matrix metalloproteinase 9, tissue inhibitor of matrix metalloproteinase 1, interleukin 6, C-reactive protein, α_1-antitrypsin, triglycerides, lipoprotein(a), apolipoprotein A, and high-density lipoprotein are also differentially expressed in patients with AAA. Whereas a linear correlation has been noted between C-reactive protein and aortic diameter,[160] this observation is at odds with at least one prior report.[161] A number of microRNAs related to smooth muscle cell function and collagen formation have also been suggested as possible AAA biomarkers.[56,162-165] At the current time, none of these candidates has the sensitivity, specificity, or rigorous clinical validation to be relied on as a diagnostic or prognostic indicator for rupture risk.[166,167]

Genetic markers identifying risk of aortic aneurysm. Genetic influences in AAA disease, first suggested by Clifton,[168] have been demonstrated by twin studies[169] and by formal segregation analyses.[170] 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.[171] Most genomic studies have investigated single-nucleotide polymorphisms in genes related to AAA pathogenesis. Potential AAA-related single-nucleotide polymorphisms have been identified in genes encoding ACE, 5,10 methylenetetrahydrofolate reductase, angiotensin II type 1 receptor, interleukin 10, matrix metalloproteinase 3, and transforming growth factor β receptor II.[172]

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.[172] Allelic variation at the q31 locus on chromosome 4 may influence endothelin signaling and respiratory epithelial response to injury, such as cigarette smoking.[173,174] Several genome-wide association studies have also been conducted for various cardiovascular diseases,[175] at least three of which have focused on AAA.[176] A number of genetic loci have been implicated in AAA pathogenesis (Table V).[172] The noncoding region of chromosome 9p21 has been identified as an important source of heritable risk for coronary and peripheral arterial disease as well as for AAA, independent of smoking, hypertension, and hyperlipidemia. The at-risk allele may mediate this effect by downregulating the cell cycle regulatory gene *CDKN2B*.[177] 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.[178,179]

Some monogenic diseases increase the risk of AAA, including mutations within the *COL3AI* gene, associated with an autosomal dominant defect in type III collagen synthesis present in patients with the Ehlers-Danlos phenotype[180,181] or mutations in fibrillin 1, responsible for Marfan syndrome.[182] 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 AAA accurately, reproducibly, and efficiently. Sensitivity and specificity approach 100%, but in 1% to 3% of patients, the aorta cannot be visualized because of bowel gas or obesity.[183,184] Transabdominal ultrasound is ideal for screening and surveillance[185] but insufficiently precise for procedural planning or more complex morphologic analyses.[186-188]

CT imaging is more reproducible than ultrasound, with >90% of measurements within 2 mm of the initial reading.[189] Both techniques suffer from a lack of standardization in terms of determining the degree and rate of disease progression.[190] An aneurysm measured by standard axial CT is generally >2 mm larger in diameter than when it is 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 overestimation of aneurysm size. There is also significant

Table V. Genetic loci implicated in the pathogenesis of an abdominal aortic aneurysm (AAA)

Genetic locus	Nearest gene (gene symbol)	SNP rs#	RAF	OR (95% CI)	P[a]	Other diseases with which the locus has been associated
3p12.3 [40]	Contactin 3 (*CNTN3*)	rs7635818	0.42	1.33 (1.10-1.21)	.0028	
9p21.3[b] [32]	CDKN2B antisense RNA 1 (*CDKN2BAS1*)	rs10757278	0.45	1.31 (1.22-1.42)	1.2×10^{-12}	Numerous; including CHD, IA, cancers, and Alzheimer disease
9q33.1[b] [41]	DAB2 interacting protein (*DAB2IP*)	rs7025486	0.25	1.21 (1.14-1.28)	4.6×10^{-10}	CHD, pulmonary embolus, PAD
12q13.3[b] [42]	Low-density lipoprotein receptor-related protein 1 (*LRP1*)	rs1466535	0.68	1.15 (1.10-1.21)	4.5×10^{-10}	

CHD, Coronary heart disease; *CI,* confidence interval; *IA,* intracranial aneurysm; *OR,* odds ratio; *PAD,* peripheral artery disease; *RAF,* risk allele frequency in population; *rs#,* reference SNP ID number; *SNP,* single-nucleotide polymorphism.
[a]*P* values are taken from the first report demonstrating association with AAA.
[b]Replicated in multiple populations.
From Golledge J, Kuivaniemi H. Genetics of abdominal aortic aneurysm. Curr Opin Cardiol 2013;28:290-6.

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.[191] 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.[192]

The increasingly small size and low cost of portable ultrasound units and absence of radiation or administration of nephrotoxic contrast material with ultrasound have made it the preferred technique for aneurysm screening and surveillance.[193-195] However, aneurysm diameter measurements, whether through the use of CT or ultrasound imaging, are most reliable and reproducible when standardized measurement techniques are used and the maximum transverse dimension measured orthogonal to the vessel axis is reported. Moreover, it is essential that ultrasound examination be performed by a qualified individual skilled in vascular imaging. For operative planning, CT remains an essential tool, given its precision, reproducibility, resolution, capacity for complete anatomic examination, and data conversion into numerous reformatting and measurement programs. 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 is used with increasing accuracy and facility in the differential diagnosis of abdominal pain. High sensitivity and specificity have been reported in detecting nonruptured aneurysms,[195,196] and use of bedside ultrasound significantly reduces time to diagnosis and treatment.[197,198] However, regions of the retroperitoneum may not be well visualized in nonfasting patients or those with ileus or excessive intestinal gas.[199]

The accuracy of CT imaging for diagnosis of symptomatic and ruptured AAA has also improved because of advances in coordinated timing and appropriate dosage of contrast material as well as through the use of multidetector arrays and image postprocessing. Timed boluses of contrast material greatly increase the sensitivity and specificity of CT imaging.[200] With modern equipment and imaging techniques, false-positive CT interpretation is low,[201] and radiographic findings of rupture are well characterized.[202]

We recommend using ultrasound, when feasible, as the preferred imaging modality for aneurysm screening and surveillance.	
Level of recommendation	1 (Strong)
Quality of evidence	A (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 criterion 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 during 23 years at a single institution.[203] Of the 473 nonresected AAAs identified in this series, 118 were ruptured. Approximately 40% of AAAs >5 cm in diameter were ruptured. However, 40% of AAAs between 7 and 10 cm were not ruptured,

whereas 13% of AAAs <5 cm were ruptured.[203] Thus, a variety of potentially more sensitive predictors of rupture risk have been proposed, including AAA expansion rate,[58,66,204,205] wall stiffness,[206-208] wall tension,[209] and peak AAA wall stress.[210-212]

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.[209] Several other investigators subsequently demonstrated that wall stress is highly dependent on AAA shape rather than diameter alone.[210-216] With ultrasound-based assessment, determination of peak wall stress may soon be translatable to real-time assessment of the patient.[217] Modeling by computational fluid dynamics has suggested that intraluminal hemodynamic conditions also influence AAA growth, remodeling, and risk of rupture.[218-222]

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.[223-225] Further analyses have also incorporated the influence of intraluminal hemodynamic conditions on wall stress and strength indices through fluid structure interaction simulations.[226,227] The value of CT-determined intraluminal thrombus volume[228,229] as well as positron emission tomography-CT imaging in predicting rupture risk remains uncertain.[230,231] The utility of positron emission tomography may require the development of AAA-specific radiotracer agents.[232]

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.[233] Molecular imaging of pathologic processes characteristic of aortic degeneration, including angiogenesis,[234] matrix disruption,[235] activated macrophage localization,[236] and proteolysis,[237] is being translated to the clinic[238-241] and may help 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.[242] 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 = aneurysm diameter [cm]/body surface area [m^2]) may represent a superior predictor of rupture risk for women.[243]

Recommendations for aneurysm screening. Aneurysm screening has been motivated by a desire to reduce AAA-related mortality and to prolong life expectancy. Overall, the probability of AAA in the general population is low but significantly increased when certain risk factors are present.[33] Four randomized clinical trials that included 127,891 men and 9342 women between 65 and 79 years of age provided evidence that ultrasound screening is effective in reducing aneurysm-related mortality.[244-249] This benefit begins within 3 years of testing and persists for up to 15 years.[250] In addition, screening is associated with a reduced risk for AAA rupture and emergency surgery.[250]

The Multicenter Aneurysm Screening Study (MASS) group, the largest of the four randomized clinical trials, reported that during 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 during 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.[251]

Screening for AAA in women is more controversial. Because few women were included in these trials, a decrease in AAA-related mortality or incidence of rupture could not be identified.[5] 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.[252]

In 2005, the U.S. Preventive Services Task Force (USPSTF) recommended a one-time screening by abdominal ultrasound for men aged 65 to 75 years with a history of smoking.[253] In 2014, the USPSTF updated their 2005 recommendations to include one-time ultrasound screening for men aged 65 to 75 years 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 years who have never smoked (grade D), and evidence was insufficient to recommend for or against screening in women aged 65 to 75 years who had a smoking history.[254] The USPSTF recommendations are based on the assumption that 6% to 7% of men with a smoking history in the 65- to 75-year 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 <0.60%.[254]

In 2007, following passage of the Screening Abdominal Aortic Aneurysms Very Efficiently (SAAAVE) amendment in 2006, the U.S. Centers for Medicare and Medicaid Services began offering one-time screening by ultrasound for men aged 65 to 75 years if they had smoked ≥100 cigarettes in their lifetime and for 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 be ordered only by the primary care physician within 6 months of the activation of Medicare benefits.[253] However, by 2012, it was

apparent that <3% of abdominal ultrasound claims were for SAAAVE-specific AAA screening, and although abdominal ultrasound examinations in the affected age groups had increased, there was no discernible effect on AAA rupture or all-cause mortality.[255] Subsequent Medicare eligibility guidelines have been modified to allow additional physician specialists to order the tests and to 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.[256] A significant portion of patients present with rupture despite known AAA status.[256] In an analysis of a cohort of >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, nonsmokers, and other groups traditionally considered at lower risk.[33]

Effective use of the electronic health record to improve screening of target populations has been demonstrated by Kaiser Permanente of Southern California.[257] "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 preceding 10 years (>54,000) were excluded from the alert system.[257]

In addition to the United States, government-sponsored screening programs have been implemented in the United Kingdom 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.[258] Similarly, in the first 3 years of the U.K. National Health Service Abdominal Aortic Aneurysm Screening Program, AAA detection rate was only 1.6%, less than 50% of that expected on the basis of the results of the prior MASS cohort.[259] However, cost-effectiveness calculations derived from MASS (£7600/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%.[260-262]

Other challenges to efficient implementation of screening include the identification and exclusion of patients who have had prior abdominal cross-sectional imaging studies, accounting for increasing longevity, potential needs for late rescreening,[263] and selective screening to optimize yield at minimal cost.[264] Reducing all-cause mortality and enhancing yield might be increased by integrating AAA screening with concurrent echocardiography[265] or blood pressure and peripheral vascular disease testing.[266] Ensuring full participation for all groups of patients at risk remains an ever-present challenge.[267,268]

We recommend a one-time ultrasound screening for AAAs in men or women 65 to 75 years of age with a history of tobacco use.	
Level of recommendation	1 (Strong)
Quality of evidence	A (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 and 75 years of age or in those older than 75 years and in good health.	
Level of recommendation	2 (Weak)
Quality of evidence	C (Low)
We suggest a one-time ultrasound screening for AAAs in men or women older than 75 years with a history of tobacco use and in otherwise good health who have not previously received a screening ultrasound.	
Level of recommendation	2 (Weak)
Quality of evidence	C (Low)
If initial ultrasound screening identified an aortic diameter >2.5 cm but <3 cm, we suggest rescreening after 10 years.	
Level of recommendation	2 (Weak)
Quality of evidence	C (Low)

Recommendations for aneurysm surveillance. Surveillance imaging should use ultrasound examinations unless other imaging modalities are specifically indicated. The optimal frequency for surveillance after recognition of early-stage AAA disease has not been defined by randomized clinical study. Some authors have suggested that there is no need to observe patients with an initial aortic diameter <3 cm, given their low risk for rupture.[248,269] However, in a 12-year analysis of 1121 men 65 years of age 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 3 years; and of those with a diameter between 3.5 and 3.9 cm, 10.5% exceeded 5.5 cm or required surgery within 2 years. Rupture occurred in 1.4%.

Two randomized controlled trials, the UKSAT[270] and the U.S. Veterans Affairs ADAM trial,[271] as well as a follow-up study of patients detected in the U.K. MASS trial[246] demonstrated that a policy of surveillance until aneurysm diameter exceeds 5.5 cm is safe and associated with a rupture rate of 1% per year. Whereas aortic size was defined by the maximum external aortic diameter, the surveillance frequency differed among these studies.

In an analysis of expansion rates of 1743 participants in the UKSAT, AAA growth rate increased with

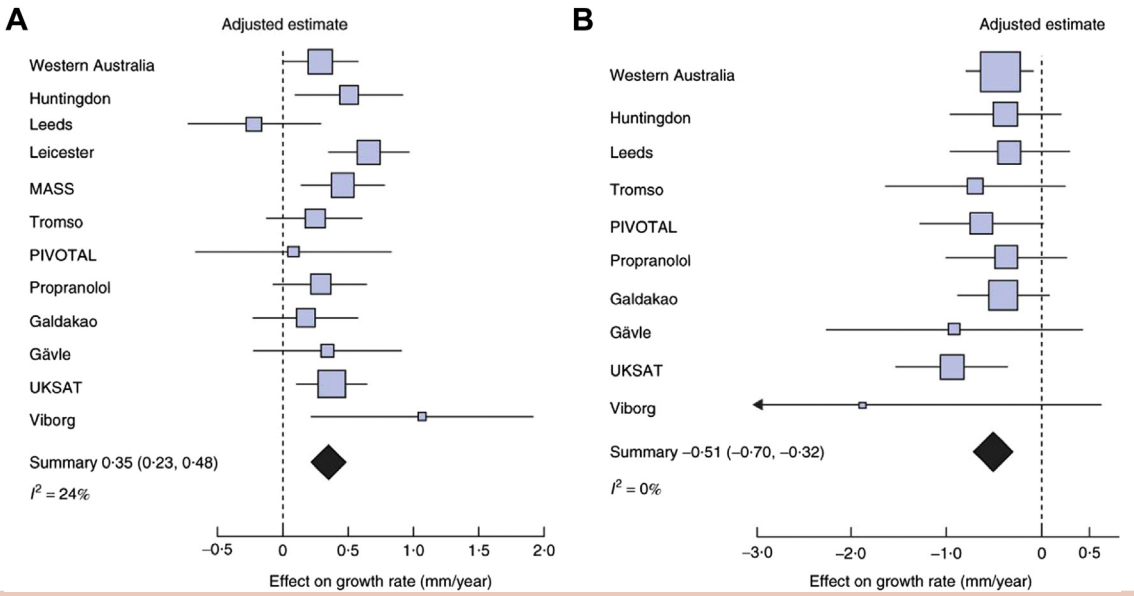

Fig 2. A, Influence of smoking (current vs ex/never) on aneurysm enlargement in individual studies and meta-analysis (see primary source for individual study citations). **B,** Influence of diabetes on aneurysm enlargement in individual studies and meta-analysis (see primary source for individual study citations). *MASS,* Multicenter Aneurysm Screening Study; *PIVOTAL,* Positive Impact of Endovascular Options for Treating Aneurysm Early; *UKSAT,* UK Small Aneurysm Trial. (From Sweeting MJ, Thompson SG, Brown LC, Powell JT. Meta-analysis of individual patient data to examine factors affecting growth and rupture of small abdominal aortic aneurysms. Br J Surg 2012;99:655-65.).

aneurysm size and among current smokers was lower in those with low ABI and diabetes and was unaffected by lipids and blood pressure.[272] 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/y, with no significant difference between men and women. When estimates were pooled using random-effects meta-analysis, following further adjustment for all demographics and medical and drug history, rates of aneurysm enlargement were significantly increased in smokers and decreased in those with diabetes (Fig 2). Pooled meta-analysis failed to identify the effects of any class of drug on aneurysm expansion.[9] After adjustment for initial aneurysm diameter, medical history, and demographics, a strong association was noted between smoking and rupture (hazard ratio, 2.02; 95% confidence interval [CI], 1.33-3.06; $P = .001$) and a far higher risk for women than for men (hazard ratio, 3.76; 95% CI, 2.58-5.47; $P < .001$). Rupture risk was increased in older participants, those enrolled in earlier studies, those with lower body mass index, and those with higher mean arterial or pulse pressure. The effect of any class of drug was difficult to evaluate because of the low incidence of rupture.[9]

Thompson and associates performed a meta-regression of growth estimates based on aneurysm diameter (Fig 3) and time to a 10% probability of attaining an aortic diameter of 5.5 cm (Fig 4).[273] 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, whereas an interval of 1 year was recommended for AAAs between 4.0 and 4.9 cm and 6 months for those between 5.0 and 5.4 cm. However, the presence of diabetes, female sex, and current smoking history were not accounted for by the model or considered within this set of recommendations.[273] 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 3-year intervals for patients with an AAA between 3.0 and 3.9 cm.	
Level of recommendation	2 (Weak)
Quality of evidence	C (Low)
We suggest surveillance imaging at 12-month intervals for patients with an AAA of 4.0 to 4.9 cm in diameter.	
Level of recommendation	2 (Weak)
Quality of evidence	C (Low)
We suggest surveillance imaging at 6-month intervals for patients with an AAA between 5.0 and 5.4 cm in diameter.	
Level of recommendation	2 (Weak)
Quality of evidence	C (Low)

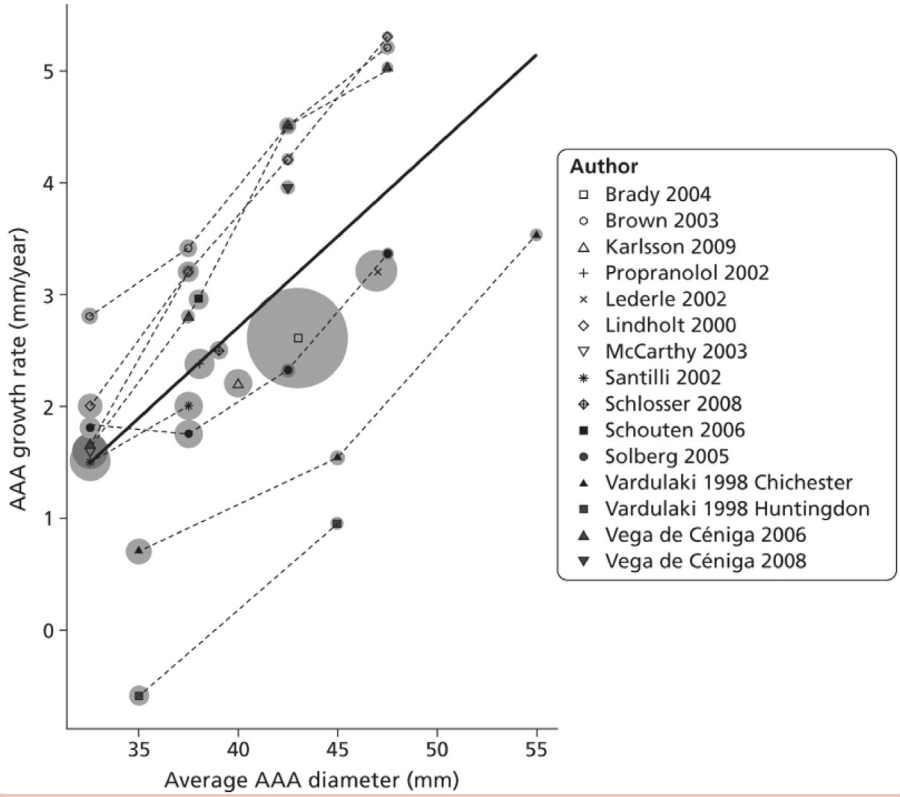

Fig 3. Meta-regression of abdominal aortic aneurysm (*AAA*) growth rate estimates by aneurysm diameter (see primary source for individual study citations). The *solid line* represents the overall regression, the *dotted line* connects estimates from the same study, and *circles* have diameters that represent 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;17:1-118.).

Recommendations for imaging of the symptomatic patient. In patients with abdominal or back pain, ultrasound imaging is recommended to determine whether 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 CT aortography with timed intravenous injection of contrast material, if it is not contraindicated, to exclude rupture and to 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 or symptom evolution or whether 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 thought 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	1 (Strong)
Quality of evidence	B (Moderate)

TREATMENT OF THE PATIENT WITH AN AAA

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 rest primarily on the maximum transverse diameter as measured on ultrasound, CT, 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, <4.0 cm in maximum diameter, are at low risk of rupture and should be monitored, whereas an aneurysm >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 although size guidelines are currently lacking because of their

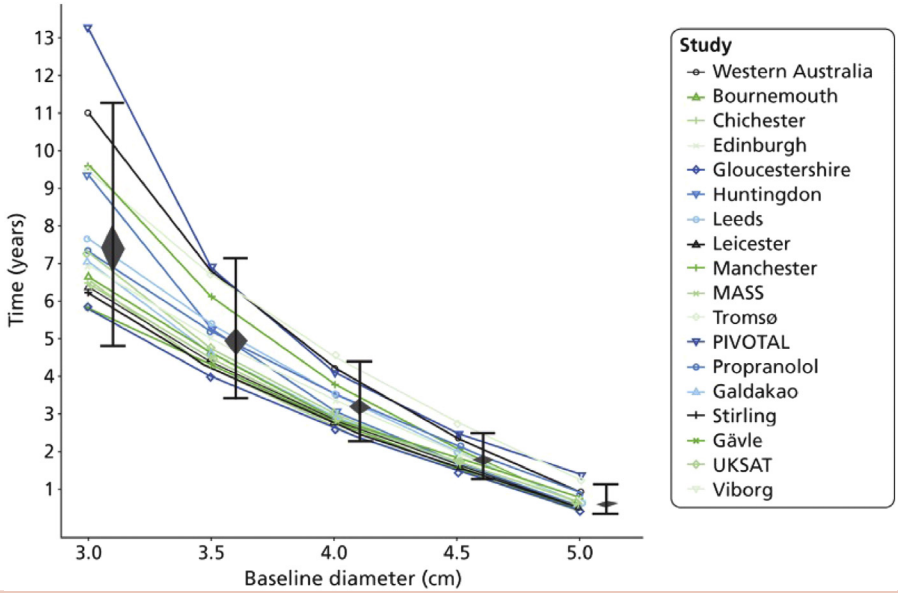

Fig 4. Estimated time to have a 10% probability of attaining an aortic diameter of 5.5 cm in male patients (see primary source for individual study citations). The *black diamonds* represent 95% confidence intervals and *error bars* represent 95% prediction intervals. *MASS,* Multicenter Aneurysm Screening Study; *PIVOTAL,* Positive Impact of Endovascular Options for Treating Aneurysm Early; *UKSAT,* UK 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;17:1-118.).

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 UKSAT[274] and the ADAM trial,[275] 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 OSR and surveillance groups. Currently, nearly 80% of all AAAs are treated by EVAR in the United States.[276,277] 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)[278] and Positive Impact of Endovascular Options for Treating Aneurysms Early (PIVOTAL)[279] trials compared immediate EVAR with surveillance for AAAs 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 review[280] of these four studies demonstrated no advantage to immediate repair by open surgery or EVAR for small AAAs (4.0-5.5 cm).

Patients with an asymptomatic fusiform AAA >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 >5.4 cm but who are of advanced age or have significant comorbid 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.[9,273,281,282]

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 to be attributed to the aneurysm.	
Level of recommendation	1 (Strong)
Quality of evidence	C (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	1 (Strong)
Quality of evidence	A (High)
We suggest elective repair for the patient who presents with a saccular aneurysm.	
Level of recommendation	2 (Weak)
Quality of evidence	C (Low)
We suggest repair in women with AAA between 5.0 cm and 5.4 cm in maximum diameter.	
Level of recommendation	2 (Weak)

Quality of evidence	B (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	2 (Weak)
Quality of evidence	C (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.[47] Smoking cessation is the most important intervention for a patient with an aneurysm.[9,272,273,283,284] Hemodynamic control with propranolol has not been shown to inhibit aneurysm expansion.[285,286] Despite the benefits of statins in cardiovascular disease, their ability to limit aneurysm expansion is lacking.[287-290] ACE inhibitors and losartan, an angiotensin receptor antagonist, decrease the rate of AAA expansion in mice, but clinical investigations have reported conflicting results.[291,292] Clinical trials of β-adrenergic receptor blockade demonstrate no effect on the rate of aneurysm progression.[285,286] 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 discontinued ACE inhibitors.[293]

Some have suggested that serologic evidence of *Chlamydophila pneumoniae* infection may be associated with AAA expansion,[294] but a prospective randomized trial demonstrated that azithromycin had no effect on aneurysm enlargement.[295] Doxycycline can inhibit matrix metalloproteinases in plasma and aneurysm tissue and thus has been proposed as an agent to limit AAA growth.[296,297] However, a randomized trial of low-dose doxycycline (100 mg once daily) demonstrated no reduction in aneurysm growth during an 18-month period.[298] An ongoing National Institutes of Health 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.[41,299]

We recommend smoking cessation to reduce the risk of AAA growth and rupture.	
Level of recommendation	1 (Strong)
Quality of evidence	B (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	2 (Weak)
Quality of evidence	C (Low)
We suggest not administering beta blocker therapy for the sole purpose of reducing the risk of AAA expansion and rupture.	
Level of recommendation	1 (Strong)
Quality of evidence	B (Moderate)

Timing of intervention

A patient with a known AAA or pulsatile mass on abdominal examination who presents without hemodynamic instability and acute onset of back or abdominal pain should undergo an immediate CT scan to determine whether rupture has occurred. Whereas a ruptured AAA represents a surgical emergency, the timing of aneurysm repair for patients with a symptomatic but nonruptured 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 ICU.

A more frequent concern is the timing for treatment of an asymptomatic, large AAA, >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 in diameter but >33% per year when aneurysms were 7 cm or larger in diameter.[66] 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 >7 cm in diameter.[67] 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 to optimize outcomes, especially for patients with associated comorbid conditions. Given the risk of rupture, both patient and family need to understand and to 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.[99,300] 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,[301] nor has this notion been supported by a prospective clinical study.[302] 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 before elective AAA repair.

In summary, the optimal timing of AAA repair is based on clinical presentation and aneurysm status: a ruptured AAA requires emergent repair; a symptomatic, nonruptured aneurysm is best treated urgently; and an asymptomatic AAA can be treated electively after completion of preoperative assessment. Delay in the treatment of an asymptomatic, large AAA should be minimized.

We recommend immediate repair for patients who present with a ruptured aneurysm.	
Level of recommendation	1 (Strong)
Quality of evidence	A (High)
Should repair of a symptomatic AAA be delayed to optimize coexisting medical conditions, we recommend that the patient be monitored in an ICU setting with blood products available.	
Level of recommendation	1 (Strong)
Quality of evidence	C (Low)

Assessment of operative risk and life expectancy

Several prediction models developed to estimate operative risk for open AAA repair and EVAR 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 OSR.[303] The most well known of this first generation of risk model were the Glasgow Aneurysm Score (GAS), the Leiden Score, and the Hardman Index. As one example, the GAS was developed from a cohort of 268 open AAA repairs, of which 41% of patients presented with ruptured aneurysms and the overall mortality was 20%. The risk score accounted for age, presence of shock, renal disease, and history of myocardial or cerebrovascular disease.[304] The European Collaborators on Stent/graft Techniques for aortic Aneurysm Repair (EUROSTAR) suggested that the GAS could be used to estimate mortality for EVAR with 30-day mortality of 1.1% for GAS <74, 2.1% for GAS of 74 to 84, and 5.3% for GAS >84.[305]

During 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 to specifically account for the mortality risk associated with EVAR. Egorova and coworkers used Medicare data to identify 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, congestive heart failure, peripheral artery disease, and liver disease.[306] 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, Giles and colleagues 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).[307] For the first time, a single scoring scheme was developed that could be applied to patients to assess risk for either EVAR or OSR. Recently, Eslami and collaborators used the Vascular Study Group of New England database to develop a new risk model that 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; Table VI, *A* and *B*).[308] This model has since been validated using the 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 5-year mortality compared with age-matched controls.[84] Bahia and colleagues recently conducted a systematic review of long-term survival after aneurysm repair.[84] Patients with large aneurysms, at greatest risk of rupture, also had significantly worse 5-year survival. As one would anticipate, many of the same risk factors for perioperative death also have an impact on 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 3-year survival after initiation of 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. Whereas the study has been criticized for its trial design, EVAR did not affect overall survival. Two-year survival was 60% and 5-year survival was 35%.[309] De Martino and coworkers assessed survival after EVAR within the Vascular Study Group of New England population using the EVAR 2 trial criteria.[310] Five-year survival for patients with aneurysms smaller than 6.5 cm was 46%; and for patients with aneurysms

Table VI, A. Mortality risk scoring scheme for patients undergoing repair of an abdominal aortic aneurysm (AAA)

Parameter	Points
Treatment	
EVAR	0
OAR (infrarenal)	2
OAR (suprarenal)	4
Aneurysm size, mm	
<65	0
≥65	2
Age, years	
≤75	0
>75	1
Gender	
Male	0
Female	1
Comorbidities	
Myocardial disease	1
Cerebrovascular disease	1
Chronic obstructive pulmonary disease	2
Laboratory value	
Creatinine, mg/dL	
<1.5	0
1.5 to <2	2
≥2	2

EVAR, Endovascular aneurysm repair; *OAR*, open aneurysm repair.
From Eslami MH, Rybin D, Doros G, Kalish JA, Farber A; Vascular Study Group of New England. Comparison of a Vascular Study Group of New England risk prediction model with established risk prediction models of in-hospital mortality after elective abdominal aortic aneurysm repair. J Vasc Surg 2015;62:1125-33.e2.

Table VI, B. Risk categorization based on mortality risk scoring scheme (Table VI, *A*) for patients undergoing repair of an abdominal aortic aneurysm (AAA)

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

From Eslami MH, Rybin D, Doros G, Kalish JA, Farber A; Vascular Study Group of New England. Comparison of a Vascular Study Group of New England risk prediction model with established risk prediction models of in-hospital mortality after elective abdominal aortic aneurysm repair. J Vasc Surg 2015;62:1125-33.e2.

larger than 6.5 cm, 5-year survival was 28%. In a recent study, those patients declined for AAA repair had a 2-year survival of 35%.[311] Thus, for a patient with high operative risk and shortened life expectancy, rupture risk must be high for benefit to be obtained from EVAR.

We suggest informing patients contemplating open repair or EVAR of their VQI perioperative mortality risk score.	
Level of recommendation	2 (Weak)
Quality of evidence	C (Low)

EVAR

EVAR has rapidly expanded as the preferred approach for treatment of AAA since the first report >25 years ago.[312,313] 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.[51]

Considerations for percutaneous repair. EVAR has evolved since its inception with the development of 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, these have reduced the requirement for open surgical exposure of the femoral artery. A randomized study comparing open exposure and the percutaneous "pre-close" technique using the Perclose ProGlide (Abbott Vascular, Santa Clara, Calif) device demonstrated both safety and effectiveness.[314] After femoral artery access, systemic anticoagulation with 100 units/kg of intravenous heparin is recommended with a target activated clotting time ≥300 seconds.

Infrarenal fixation. EVAR requires nonaneurysmal proximal and distal attachment sites or sealing zones as dictated by device-specific instructions for use. Most endografts that are dependent on infrarenal fixation have required a proximal sealing zone of at least 15 mm in length, a neck diameter <32 mm, and a neck angulation of <60 degrees. Several devices now report efficacy with shorter neck lengths and more severe levels of angulations. Use of devices outside recommended parameters increases 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, and extensive neck calcification. Whereas concerns have been raised about the risks of renal or mesenteric embolization, occlusion, and end-organ ischemia, observational studies have documented the efficacy and safety of suprarenal fixation.[315-319] The rate of renal dysfunction appears to be equivalent for endografts that use nitinol or stainless steel transrenal stents and not significantly different from that observed with infrarenal fixation.[318] Although suprarenal fixation may produce a higher incidence of small renal infarcts, these do not appear to be clinically significant in most patients. Renal dysfunction after EVAR with suprarenal fixation is likely to be multifactorial and transient in most patients.[320] Nonetheless, renal artery occlusion and infarctions have been reported in patients with pre-existing renal artery occlusive disease, and although infrequent, visceral dysfunction and celiac or mesenteric artery occlusion may occur secondary to suprarenal fixation.[321-324] One report showed no difference in renal function between the two device types, whereas another study demonstrated a reduction in renal function after the use of a suprarenal fixation device.[325,326] A recent meta-analysis examining the renal complications after standard EVAR with suprarenal and infrarenal fixation demonstrated no difference in renal complications.[327]

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.[328-332] 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.[329,330] 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.[333] 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.[334] 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 (St. Jude Medical, St. Paul, Minn) to occlude only the main trunk of the HA.[335] A more recent report demonstrated no clinical difference between coils and plug embolization.[336] Despite concerns about prolonged procedural time and increased amount of contrast material, concomitant unilateral HA embolization during EVAR has been shown to be safe and effective compared with a staged approach.[337]

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 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 have suggested that such devastating complications are exceedingly rare.[329,338-340] The risks associated with bilateral HA occlusion are restricted to more severe, persistent, and frequent buttock claudication and erectile dysfunction.[341]

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.[329,340] An alternative consideration to avoid bilateral HA embolization during EVAR is open or endovascular revascularization of at least one internal iliac artery.[342,343] FDA-approved iliac branch graft devices to maintain ipsilateral internal iliac perfusion have been developed or are under review.[344-346] These devices have displayed satisfactory early outcomes and should be considered before 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 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.[338] 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 comorbidities present in the older patient demographic at risk for AAA disease.[347] Several endovascular techniques have been described to preserve internal iliac artery flow, including the development of commercially available aortoiliac endografts that incorporate an iliac branch.[348-351]

We recommend preservation of flow to at least one internal iliac artery.	
Level of recommendation	1 (Strong)
Quality of evidence	A (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	1 (Strong)
Quality of evidence	A (High)
We recommend staging bilateral internal iliac artery occlusion by at least 1 to 2 weeks if required for EVAR.	
Level of recommendation	1 (Strong)
Quality of evidence	A (High)

Management of associated vascular disease. Coexistence of other vascular disease with an AAA is common. Several series reporting observations of aortography have documented >50% stenosis in 20% to 40% of renal arteries, 10% to 15% of celiac or superior mesenteric branches, and 20% to 30% of iliac vessels.[352,353]

The decision to intervene is based on 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.[354,355] The exception may be the patient presenting with high-grade stenosis of the SMA and a meandering mesenteric artery that is based off of a large IMA, which will be sacrificed during the course of treatment. A decision to repair each lesion should be based on 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 disease 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.[352] Whether an accessory renal artery requires preservation depends on the size of the artery, its contribution to the renal parenchyma, and the presence of coexisting kidney disease. Renal infarction after occlusion of an accessory renal artery is common, occurring in 84% of kidneys, but it is well tolerated in most patients, without significant impact on long-term glomerular filtration rate.[356] Nonetheless, preservation should be considered for large accessory renal arteries (≥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.[357,358]

We suggest renal artery or SMA angioplasty and stenting for selected patients with symptomatic disease before EVAR or OSR.	
Level of recommendation	2 (Weak)
Quality of evidence	C (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	2 (Weak)
Quality of evidence	C (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	2 (Weak)
Quality of evidence	C (Low)

Perioperative outcomes of elective EVAR

Incidence of 30-day and in-hospital mortality. The UK EVAR 1, Dutch Randomized Endovascular Aneurysm Management (DREAM), U.S. Veterans Affairs Open Versus Endovascular Repair (OVER), and French Anevrysme de l'aorte abdominale: Chirurgie versus Endoprothese (ACE) multicenter randomized trials collectively randomized 2790 patients to EVAR or open repair.[359-362] 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% compared with 4.2% for open surgery (OR, 0.3; 95% CI, 0.22-0.50; $P < .0001$).[363] 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).[364] Outcomes after AAA repair are related to experience. Whereas earlier studies[365,366] 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.[367] The OR for elective perioperative mortality adjusted for the surgeon's 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	2 (Weak)
Quality of evidence	C (Low)

Perioperative morbidity. Estimated blood loss is significantly lower with EVAR than with open repair.[368] Whereas 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%; $P < .001$), acute renal failure (4.3% vs 11.3%; $P < .001$), MI (2.5% vs 5.2%; $P < .001$), and bowel ischemia (0.6% vs 2.1%; $P < .001$). EVAR patients were also more likely to be discharged to home rather than to a skilled nursing facility (95% vs 83%; $P < .001$). 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 compared with 7 days after open repair ($P < .001$).

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 undersizing or oversizing.[369-371] Initial management is angioplasty with a compliant balloon, followed by extension cuff placement. Additional maneuvers include placement of a Palmaz (Cordis, Bridgewater, NJ) balloon-expandable stent or endostapling.[369,372] 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.[373] A persistent type IA endoleak may be treated by placement of a fenestrated device,[374,375] proximal cuff extension with chimney grafts to the renal arteries,[376,377] 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 repeated balloon angioplasty and, as needed, by graft extension. Coil embolization of the HA 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.[378,379] 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.[380] A type IV endoleak is self-limited, and treatment is not required.

Access site complications. Early experience with EVAR using open femoral artery exposure was associated with a high rate of access site-related complications (13%). These included 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%).[381] In a multicenter randomized controlled trial, percutaneous access was superior to open femoral artery access with a shorter procedure time (107 vs 141 minutes; $P = .004$) and fewer access complications (6% vs 10%; $P = .005$), with a 96% technical success rate.[314] A systematic review and a recent National Surgical Quality Improvement Program (NSQIP) review also demonstrated a high technical success rate, shorter operative time (135 vs 152 minutes), shorter length of stay (1 vs 2 days), and fewer wound complications (1% vs 2.1%; $P = .02$).[382-385] Percutaneous access may not be appropriate for patients with small vessels, for patients with 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.[386-390] Percutaneous access with large sheaths is improved by ultrasound guidance.[386,391-393]

Acute limb thrombosis. Early graft limb thrombosis may occur in 2% of patients because of 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.[359,360,370,394-396]

Postimplantation 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.[397-401]

Ischemic colitis. Colon ischemia due to occlusion of the IMA or HA or embolization is rare after EVAR (<1%).[334,364,396,402,403] Circumflex femoral and circumflex iliac arteries should be preserved should HA occlusion be planned. Suspected colonic ischemia should be assessed by endoscopy, and if it is confirmed, antibiotics should be 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 with observation and found no benefit to EVAR for patients who were considered unfit for open repair because of a history of MI or cardiac revascularization, stable angina, valvular heart disease, significant arrhythmia, uncontrolled congestive heart failure, forced expiratory volume in 1 second <1 L, or serum creatinine concentration >2.3 mg/dL.[309] However, of those randomized to EVAR, only 179 of 197 (91%) underwent surgery; 14 deaths and 9 ruptures occurred before surgery, which was performed 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 that there is a subgroup of patients not fit for open repair who 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 nonoperative management. There may also be patients who although they are at high risk for open surgery are at reasonable risk for EVAR. In addition, as noted in one-third of patients randomized to observation in EVAR 2,

fitness may be improved by optimization of comorbid disease to the extent that EVAR may then be considered.

We suggest informing high-risk patients of their VQI perioperative mortality risk score for them to make an informed decision to proceed with aneurysm repair.	
Level of recommendation	2 (Weak)
Quality of evidence	C (Low)

OSR

Indications. OSR of an AAA continues to be used for patients who do not meet the anatomic requirements for endovascular repair, including short or angulated landing zones, excessive thrombus, multiple large accessory renal arteries, and small and tortuous access vessels with concomitant occlusive disease. However, fenestrated, branched, and chimney or snorkel grafts have expanded the range of complex aortic anatomy potentially treatable by EVAR. OSR 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 transperitoneal or left flank retroperitoneal approach (Table VII). Indications for each type of approach are largely based on the patient's anatomy and comorbidities and the surgeon's preference. The transperitoneal approach is typically performed using a generous midline incision from the xiphoid process to the symphysis pubis. Extension of the incision alongside the xiphoid process releases rectus aponeurosis and facilitates exposure in the obese patient or in those with more proximal aortic disease. A minilaparotomy (15 cm) has been used in select patients. A transperitoneal approach can be performed rapidly and is versatile, allowing assessment of intra-abdominal disease 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.[404,405] The measurable benefits attributed to retroperitoneal exposure were primarily a shorter duration of ileus and earlier resumption of oral intake. Sieunarine and colleagues 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.[406]

The retroperitoneal approach may be preferred for repair of a suprarenal aneurysm because exposure can be facilitated by division of the left diaphragmatic crus. However, in a majority of patients, repair of juxtarenal and pararenal aneurysms can be performed using a transperitoneal approach with excellent outcomes.[407,408] 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.[407,408] An important indication for the retroperitoneal approach is the presence of a hostile abdomen because of prior intra-abdominal operations, irradiation, incisional hernia, stoma, or enterocutaneous fistula. In addition, a retroperitoneal approach can facilitate repair of an inflammatory aneurysm or aneurysm associated with a horseshoe kidney.[409,410]

We recommend a retroperitoneal approach for patients requiring OSR of an inflammatory aneurysm, a horseshoe kidney, or an aortic aneurysm in the presence of a hostile abdomen.	
Level of recommendation	1 (Strong)
Quality of evidence	C (Low)
We suggest a retroperitoneal exposure or a transperitoneal approach with a transverse abdominal incision for patients with significant pulmonary disease requiring OSR.	
Level of recommendation	2 (Weak)
Quality of evidence	C (Low)

Aortic clamping. Selection of the ideal clamp site and extent of reconstruction is based on analysis of cross-sectional aortic imaging. These features include 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 retroaortic 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; 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 xiphoid to the symphysis pubis or, in select cases, a minilaparotomy 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 retroperitoneum and aortic aneurysm. The retroperitoneum is

Table VII. Surgical approaches for open aneurysm repair

	Transperitoneal	Retroperitoneal
Advantages	• Most rapid, greatest versatility • Provides widest access • Enables evaluation and treatment of concomitant intra-abdominal disease	• Avoids hostile abdomen • Facilitates suprarenal exposure and control • Potential reduction of postoperative ileus • Obesity • Inflammatory AAA • Horseshoe kidney
Disadvantages	• Longer postoperative ileus • Potential for greater fluid losses • Difficulty with exposure and control for suprarenal aneurysms • Higher incidence of incisional hernia	• Poor access to right renal and iliac arteries • Cannot evaluate intra-abdominal disease • Flank bulge

AAA, Abdominal aortic aneurysm.
Adapted from Chaikof EL, Brewster DC, Dalman RL, Makaroun MS, Illig KA, Sicard GA, et al. The care of patients with an abdominal aortic aneurysm: the Society for Vascular Surgery practice guidelines. J Vasc Surg 2009;50(Suppl):S2-49.

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 is required, division of the gonadal, adrenal, and lumborenal 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.[411,412] Patients in whom an aortic aneurysm required a suprarenal clamp have an increased risk of renal dysfunction and morbidity but similar 30-day mortality compared with those in whom an infrarenal clamp site was sufficient for repair of an AAA.[407,408,413] Visceral vessel control is not necessary as backbleeding is minimal after supraceliac aortic cross-clamp 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 because 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 units/kg of intravenous heparin is recommended for elective aneurysm repair, irrespective of the location of the aortic clamp. Heparin may be omitted or administered in lower doses in special circumstances of a ruptured aneurysm or other unusual situations. In these cases, the graft is vigorously flushed before restoration of blood flow, or limited amounts of heparinized saline may be instilled directly into the distal vessels after placement of 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	1 (Strong)
Quality of evidence	B (Moderate)

Graft type and configuration. There is no significant difference in patency, durability, resistance to infection, or risk of degeneration or dilation of currently used prosthetic materials. Differences in methods of fabrication of polyester grafts include knitted or woven, external or double velour, and high or low porosity. Polyester grafts can be rendered impermeable by various biologic coatings, including collagen, gelatin, and albumin. Graft impregnation with silver or rifampin has been used to enhance resistance to infection. Routine use of rifampin-impregnated polyester grafts, in which gelatin-coated polyester grafts are soaked in rifampin solution (1 mg/mL) for 15 to 30 minutes, or of silver-impregnated grafts to limit the risk of device-associated infection has not proved to be beneficial in prospective or multicenter studies.[414-417]

Graft configuration can be a straight tube or bifurcated. The location of the distal anastomosis is at the aortic

bifurcation, iliac artery, or femoral artery. A tube graft is preferable when it is feasible because of shortened operative time and reduced blood loss and need for dissection, minimizing risk of inadvertent injury to the ureter, iliac veins, and autonomic nerves. In the era before widespread adoption of EVAR, approximately 40% to 50% of patients could be treated with a tube graft.[418] In the Canadian aneurysm trial, graft configuration was straight in 39%, aortobi-iliac in 31%, aortobifemoral in 24%, and aortoiliac and femoral in 7%.[419] Bifurcated grafts are indicated when the distal aorta and common iliac arteries are aneurysmal, which occurs in one-third of patients.[420] 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.[419] Assuming that normal iliac arteries have been selected for the distal anastomosis, the risk of progressive distal aneurysmal or occlusive iliac disease is relatively low.[418,421]

We recommend straight tube grafts for OSR of AAA in the absence of significant disease of the iliac arteries.	
Level of recommendation	1 (Strong)
Quality of evidence	A (High)
We recommend performing the proximal aortic anastomosis as close to the renal arteries as possible.	
Level of recommendation	1 (Strong)
Quality of evidence	A (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	1 (Strong)
Quality of evidence	A (High)

Maintenance of pelvic circulation. Perfusion of the colon, rectum, and pelvis is provided by a complex collateral network from the SMA and IMA through the marginal artery of Drummond, the internal iliac arteries, and additional collaterals from the circumflex iliac and 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 eightfold (0.3% to 2.6%) when both internal iliac arteries were occluded compared with when at least one of the internal iliac arteries was preserved.[419,420] Thus, all efforts should be made to preserve perfusion to at least one internal iliac artery.

Colonic ischemia after aortic repair is multifactorial in origin, but ligation of a patent IMA during reconstruction remains a risk factor.[422] 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 reimplantation of a patent IMA has not been established, but selective reimplantation may be of value in the presence of compromised pelvic perfusion, particularly when the marginal artery is interrupted because of prior colectomy.[403,423] A prospective randomized study suggested that IMA reimplantation is beneficial in patients of advanced age and when intraoperative blood loss has been substantial.[424] Likewise, reimplantation 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 before opening the aneurysm by 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 reimplantation was used in 5% of the patients but was associated with increased risk of postoperative bleeding.[419] Preservation of antegrade flow into at least one of the internal iliac arteries is recommended whenever possible. For patients treated by OSR, this can usually be achieved by a distal anastomosis to the iliac bifurcation, an 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.

We recommend reimplantation of a patent IMA under circumstances that suggest an increased risk of colonic ischemia.	
Level of recommendation	1 (Strong)
Quality of evidence	A (High)
We recommend preserving blood flow to at least one HA in the course of OSR.	
Level of recommendation	1 (Strong)
Quality of evidence	A (High)

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

Pearce and colleagues reported a 30-day mortality of 3% among 678 patients treated for AAAs with concomitant renal artery reconstruction.[425] However, mesenteric artery reconstruction combined with aortic reconstruction carries a higher mortality rate and should be avoided unless it is clinically indicated.[426] 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 OSR in symptomatic patients who are not candidates for catheter-based intervention.	
Level of recommendation	2 (Weak)
Quality of evidence	B (Moderate)

Management of associated intra-abdominal nonvascular disease. In the occurrence of an AAA and associated intra-abdominal disease, 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 disease, with a prevalence of 5% to 20%. Asymptomatic cholelithiasis should be left untreated because the risk of acute cholecystitis after elective AAA repair is <1%.[427] 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 4 to 6 weeks after AAA repair.[428] 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 coexistent cholecystitis or an intra-abdominal tumor in patients who are not candidates for EVAR or staged intervention.	
Level of recommendation	2 (Weak)
Quality of evidence	C (Low)

Perioperative outcomes of open AAA repair

Factors affecting mortality of OSR include the surgeon and hospital volume, urgency of the procedure, age of the patient, presence and severity of comorbidities, and proximal aneurysm extension. Symptomatic coronary artery disease, congestive heart failure, severe chronic pulmonary disease, and advanced chronic kidney disease remain the most important predictors of mortality.[364] There has been considerable variation in 30-day mortality rates in the literature, depending on the type of study reported and its design.[429] Elective 30-day mortality for infrarenal AAA OSR in most contemporary large single-center institutional reports has ranged from 1% to 4%.[109,430,431] 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.[34,109,145,419,432-437] Recent multicenter, prospective, randomized trials have demonstrated 30-day mortality of 3% to 4.7%.[368,438,439] Analyses of outcomes in Medicare beneficiaries indicate that OSR mortality, although improved in the last decade, remains higher than that associated with EVAR for every age category.[364] Similarly, the morbidity of OSR is significantly higher compared with EVAR, particularly cardiac, pulmonary, renal, gastrointestinal, and wound-related complications (Table VIII). Finally, recent studies have shown that >20% of the patients treated by OSR require reoperations for laparotomy-related complications within 8 years.[364]

Table VIII. Estimated perioperative complications after elective open surgery for abdominal aortic aneurysm (AAA)

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

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

The impact of individual surgeon and hospital volume on outcomes of AAA OSR has been documented in several studies.[145,434,440,441] A review of national Medicare claims by Birkmeyer and coworkers indicated that 30-day mortality was 8% for low-volume hospitals (<17/y) compared with 4.4% in high-volume hospitals (>79/y).[440] Surgeon volume and prior dedicated vascular training also affect mortality of OSR. Dimick and associates reported that elective AAA mortality was lowest when operations were performed by vascular surgeons (2.2%) compared with cardiac surgeons (4%) and general surgeons (5.5%; $P < .001$).[442] 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 by high-volume surgeons was 30% and 40%, respectively. AAA repair performed by a general surgeon increased risk of death by 76% compared with repair performed by a vascular surgeon.[442] A recent risk-adjusted analysis of 122,495 Medicare patients undergoing elective AAA repair between 2001 and 2008 noted that the mortality for OSR is directly related to medical center volume.[367]

The OR for elective perioperative mortality was lowest for centers that perform at least 18 open repairs and was <5%.[367]

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 EVAR. 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	2 (Weak)
Quality of evidence	C (Low)

The patient with a ruptured aneurysm

Preoperative management and considerations for patient transfer. A ruptured AAA 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 before hospitalization or without treatment.

Establishing a protocol or algorithm for urgent or emergent management of a patient with a ruptured AAA is essential for optimizing outcomes.[443] In the presence of a protocol, 30-day mortality was 18%, whereas in the absence of a protocol, 30-day mortality was 32%.[444-446] Based on review of the literature, including existing guidelines endorsed in the United Kingdom[447] and by the Western Vascular Society, an algorithm for the initial evaluation, diagnosis, immediate management, and triage of patients with a suspected ruptured AAA is presented (Fig 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 AAA. Diagnosis in the emergency department is usually ascertained on the basis of history and physical examination. Radiologic confirmation, either by 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 patient's clinical condition in the preoperative 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 and 90 mm Hg, should be implemented to limit excessive hemorrhage.[448-450] Laboratory or imaging studies should be obtained only to confirm the diagnosis of ruptured AAA. Other actions that may help improve outcomes are the immediate availability of blood and blood products, warming, and avoidance of elective intubation.[451]

With the increasing use of endovascular methods to treat patients presenting with a ruptured AAA, vital resources, including advanced imaging, trained staff, and robust endovascular inventory, must be available. In cases in which transfer is not necessary, the vascular team should be notified as soon as a ruptured AAA is suspected. It may be prudent, however, to transfer a patient to a higher level facility when such resources are unavailable.[452,453] Patients with good functional status and without severe comorbidity should be transferred without delay. Furthermore, patients who previously declined elective surgery should be considered for transfer and treatment. Some patients experiencing a ruptured AAA may not be medically fit to undergo open repair and 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.[454] Preoperative predictors of death after open repair include age >76 years, serum creatinine concentration of >2.0 g/dL, pH <7.2, and blood pressure <70 mm Hg at any time. Whereas these risk factors require more robust validation, when all four are present, open repair is uniformly fatal.[455] 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 to 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 be transmitted, preferably by an electronic method.[456] Few data exist to guide best management during this critical transfer time. Patients should receive intravenous nitroglycerin, esmolol, sodium nitroprusside, and pain medication, as needed, to avoid hypertension and to 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.[457]

Systems of care and time goals for intervention. Timeliness of intervention for the patient with a ruptured AAA affects outcomes.[458-460] A goal of *door-to-intervention time of <90 minutes* is recommended, with time zero defined as the time of first medical contact and

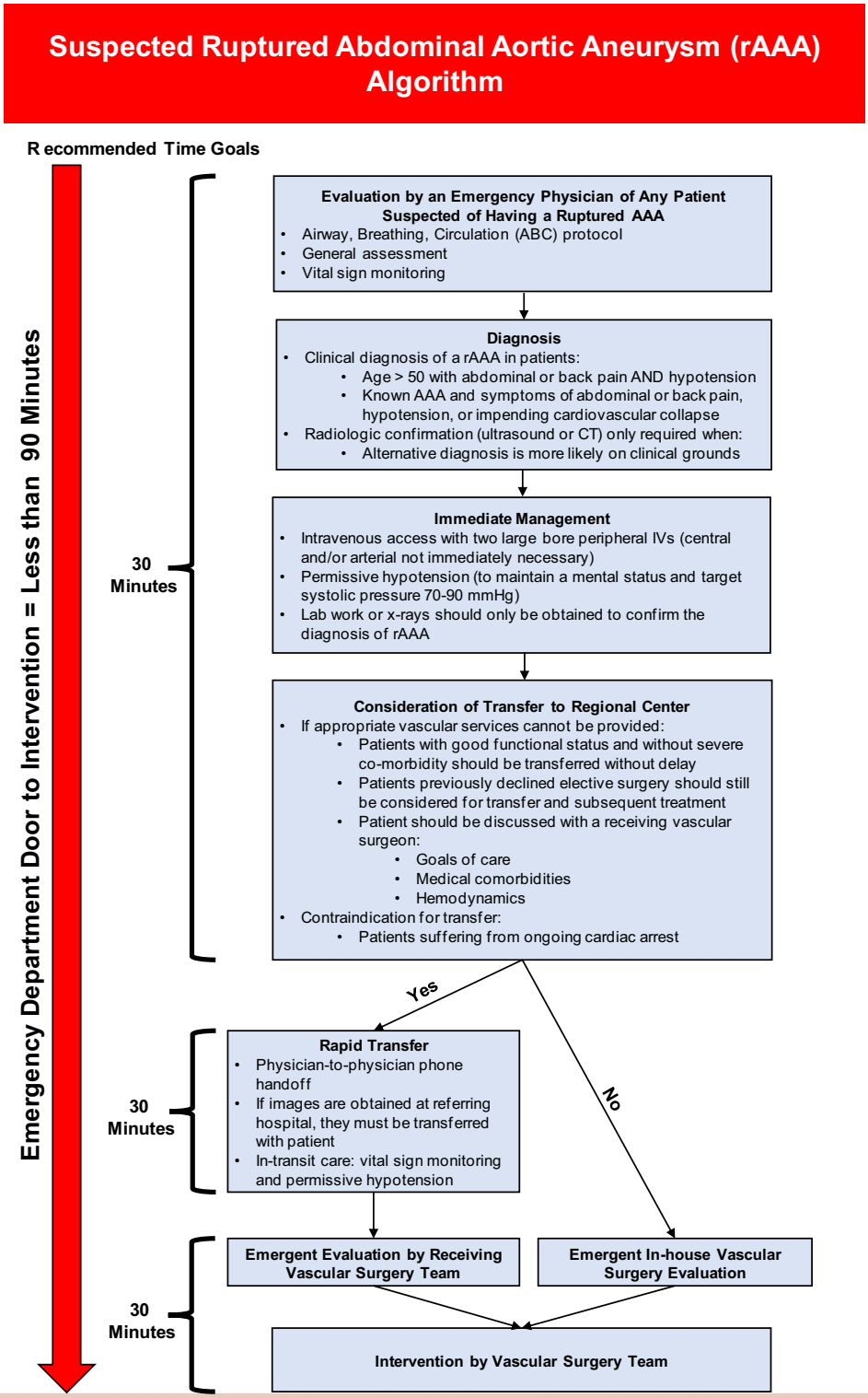

Fig 5. Algorithm for management of the patient with a suspected or confirmed ruptured abdominal aortic aneurysm (*AAA*). *CT,* Computed tomography; *IVs,* intravenous lines.

intervention defined as initial arterial access and placement of an aortic occlusion balloon (Fig 5). Given limited studies benchmarking time to intervention for a ruptured AAA,[458,460] this goal has been proposed on the basis of the 2004 American College of Cardiology (ACC) Foundation/American Heart Association (AHA) guidelines established for the management of ST-segment elevation MI (STEMI).[461] For the patient needing transfer

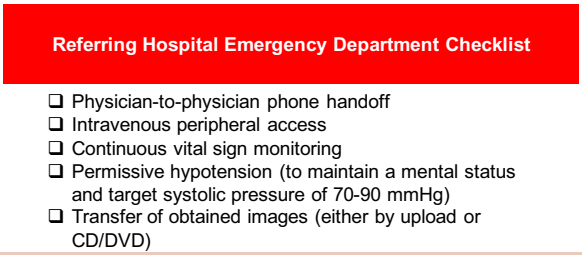

Fig 6. Referring hospital checklist for the patient with a suspected or confirmed ruptured aneurysm.

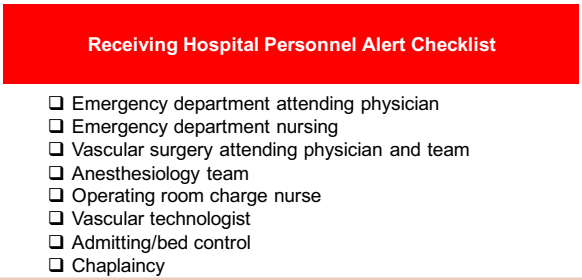

Fig 7. Receiving hospital personnel alert checklist for management of the patient with a suspected or confirmed ruptured aneurysm.

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 emergent in-house vascular surgery evaluation is initiated. 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 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 (Figs 6 and 7). More important, and similar to STEMI management,[462,463] the establishment of *systems of care* will be necessary. With an organized regional transfer system, operative repair can be performed in >95% of patients with a ruptured AAA, with 67% survival.[458] 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 time 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.

The benchmark of <90-minute door-to-balloon time for the management of the patient with a STEMI was initially extremely challenging to meet.[461] In 2004, the National Cardiovascular Data Registry reported that for patients requiring interhospital transfer, only 8.6% had total door-to-balloon times of <90 minutes, with a median time of 152 minutes.[464] Nonetheless, the establishment of an ambitious time goal promoted the development of STEMI systems of care to decrease time to intervention and to improve overall patient survival.[463] The challenge faced by rural centers was recognized when the 2004 ACC/AHA 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.[465]

We suggest a door-to-intervention time of <90 minutes, based on a framework of 30-30-30 minutes, 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	1 (Strong)
Quality of evidence	B (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

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, and anatomic limitations that prevent expeditious repair.[451,466] A femoral artery approach with use of a long sheath is preferred to a brachial approach. The sheath may be advanced into the supraceliac aorta to support the balloon and permit its removal after endograft placement.[466]

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 intravascular ultrasound. Both bifurcated and aortouni-iliac endografts have been used for emergent EVAR.[451,467-469] Although it is used less commonly, an aortouni-iliac device may be helpful in the treatment of an anatomically challenging AAA. The Nellix Endovascular Aneurysm Sealing System (Endologix, Irvine, Calif) has also been proposed for treatment of ruptured AAA.[470,471]

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%).[472] 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.[469] The Immediate Management of Patients with Rupture: Open Versus Endovascular Repair (IMPROVE) trial was a multicenter randomized trial of EVAR and open repair for patients presenting with a ruptured AAA.[473] Patients were randomized before CT imaging was performed; 316 patients were randomized to EVAR and 297 patients to open repair. The 30-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 1-year outcomes demonstrated that EVAR was most cost-effective compared with open repair, but no survival benefit was observed.

An analysis of national trends in the United States confirms that EVAR is being used with increasing frequency for the treatment of ruptured AAA, with a decrease in associated mortality.[474,475] Outcomes are superior when EVAR for a ruptured aneurysm is performed in teaching hospitals and high-volume centers.[474]

If it is anatomically feasible, we recommend EVAR over open repair for treatment of a ruptured AAA.	
Level of recommendation	1 (Strong)
Quality of evidence	C (Low)

Management of postoperative complications.

Abdominal compartment syndrome. Abdominal compartment syndrome is a well-recognized complication after both OSR and EVAR for ruptured aneurysm and may occur in approximately 7% of patients.[476] Use of an aortic occlusion balloon, coagulopathy, massive transfusion, and conversion to an aortouni-iliac device are all predictors of abdominal compartment syndrome.[477] 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.[478]

Ischemic colitis. Colonic ischemia after repair of a ruptured AAA may occur in as many as one in five to one in three patients.[479] Ischemic colitis after vascular surgery has been associated with mortality rates of 45% to 67%, with recent reports demonstrating only modest improvement in outcomes.[480] Delayed diagnosis, with advanced ischemic colitis leading to perforation, is associated with a mortality rate in excess of 90%,[481] and a retrospective review of 222 patients revealed that ischemic colitis is the most common cause of death after open repair of a ruptured aneurysm.[482] Colonic ischemia is much less frequent after EVAR than after OSR for ruptured aneurysm (23% vs 42%), but the risk remains.[403,480,483] Prompt endoscopy is recommended when ischemic colitis is suspected to confirm the diagnosis and to help guide management.

Multisystem organ failure. Given the associated hemodynamic instability and ischemia-reperfusion injury among patients presenting with a ruptured aneurysm, multisystem organ failure may occur in 1% to 3% of patients after EVAR or open repair.[484,485] Once multisystem organ failure develops, organ dysfunction leads to a prolonged ICU stay, high resource consumption, and a 50% to 70% mortality rate.[486] Dedicated ICU teams and regionalized care at high-volume centers have led to reduced mortality and decreased length of stay.[487,488]

Special considerations

Inflammatory aneurysm. An inflammatory aortic aneurysm occurs in between 5% and 10% of patients.[489] An inflammatory aortic aneurysm may not be readily apparent on CT imaging but may be associated with retroperitoneal fibrosis[490,491] and displays a similar natural history to the more common degenerative aortic aneurysm.[492,493] An inflammatory aortic aneurysm is typically adherent to the duodenum and, less commonly, the ureters, renal vein, and inferior vena cava.[494] 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 is associated with decreased mortality compared with open repair.[495-500]

Horseshoe kidney. A horseshoe kidney occurs in 0.25% of the general population and in 0.12% of patients presenting with an aortic aneurysm.[501] 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.[502-504] If the main renal arteries are located proximal to the aneurysm, EVAR can be safely performed.[505,506] Small accessory renal arteries may be covered by EVAR.[507,508] In cases with anomalous blood supply, OSR (preferably through a retroperitoneal approach), hybrid repair, or fenestrated or branched EVAR may be considered.[509-511] Multiple renal arteries can be surgically reimplanted using a Carrel patch or through an inclusion technique.[512] Should open repair be required and CT imaging reveal that the horseshoe kidney is associated with a thin fibrous isthmus, a transperitoneal approach may be considered with division of the "isthmus."[513-515] 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.[516,517] Likewise, repair has also been described using a fenestrated endograft and snorkel grafts.[518,519]

Aortocaval fistula. A ruptured aneurysm associated with an aortocaval fistula has been reported in 0.22% to 6% of patients.[520] The triad of abdominal pain, pulsatile mass, and abdominal "machinery" bruit is present in up to 80% of cases.[521-524] 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.[525-529] Duplex ultrasound imaging will reveal an arterial flow pattern in the inferior vena cava, and CT imaging will demonstrate contrast material in the inferior vena cava during the arterial phase.[530-533] EVAR is preferred,[534-540] with expected resolution of preoperative heart failure and other physiologic disturbances.[541-544] 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.[545,546]

Since 2013, an additional 53 patients presenting with an aortocaval fistula have been added to the previously reported 250 cases.[547] The majority have been successfully treated with EVAR alone, with occasional use of an Amplatzer plug[548] or additional placement of a covered stent in the inferior vena cava.[549-552]

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.[553] Insertion of monitoring lines before 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.[554] In addition, it has been postulated but not proven that there is decreased hemodynamic lability and cardiac ischemia.[555,556] 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 COPD.[557-559]

EVAR can be safely performed under general, epidural, or local anesthesia. Whereas 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 4000 patients in the EUROSTAR registry, local anesthesia was associated with shorter operative times, reduced ICU admission, shorter hospital stay, and fewer systemic complications.[560] There was a modest advantage of epidural anesthesia compared with general anesthesia. A recent review of the American College of Surgeons NSQIP database demonstrated that general anesthesia was associated with longer hospital stay and increased pulmonary morbidity compared with local or regional anesthesia.[561] 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.[562] 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.

We recommend general endotracheal anesthesia for patients undergoing open aneurysm repair.	
Level of recommendation	1 (Strong)
Quality of evidence	A (High)

Anesthetic considerations in the patient with a ruptured aneurysm. Regardless of the choice of EVAR or OSR, there is evidence to support the implementation of a standardized protocol for the efficient evaluation and treatment of ruptured aneurysm, including anesthetic management.[451,469] Notably, the surgical field should be initially draped and a transfemoral aortic balloon placed, especially if general anesthesia is required, because of the likelihood of vasodilation, hypotension, and cardiovascular collapse. Permissive hypotension to maintain a systolic blood pressure of 80 mm Hg limits volume overload and appears sufficient to maintain critical end-organ perfusion.[563] 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 confirmed that prophylactic antibiotics, administered before incision, reduce the risk of wound infection and early graft infection in arterial reconstructive surgery.[564] However, continuing antibiotics for >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 of an aortic prosthesis.

We recommend intravenous administration of a first-generation cephalosporin or, in the event of penicillin allergy, vancomycin within 30 minutes before OSR or EVAR. Prophylactic antibiotics should be continued for no more than 24 hours.	
Level of recommendation	1 (Strong)
Quality of evidence	A (High)
We recommend that any potential sources of dental sepsis be eliminated at least 2 weeks before implantation of an aortic prosthesis.	
Level of recommendation	Good Practice Statement
Quality of evidence	Ungraded

Intraoperative fluid resuscitation and blood conservation. Allogeneic blood transfusion remains associated with immunologic 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 nonused blood.[565] 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.[566-568] 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.[569] Various methods of cell salvage, including use of a cell saver or hemofiltration, have not been associated with meaningful differences in clinical outcomes.[570] A Cochrane review of cell salvage used in a variety of operations demonstrated an overall reduction of <1 unit per patient with decreased likelihood of requiring an allogeneic blood transfusion but no alteration in clinical outcome.[571] A meta-analysis of cell salvage in open aneurysm repair confirmed a reduced requirement for blood transfusion.[572] In addition, one retrospective study has demonstrated that cell salvage is associated with improved survival among patients undergoing open repair of a ruptured aneurysm.[573] Cell salvage is contraindicated in the presence of infection or malignant disease.

The benefit of maintaining a predefined hematocrit level during OSR of aortic aneurysm is unknown, but pre-emptive transfusion in the setting of rapid ongoing blood loss is well supported.[574] 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.[457,575,576] 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 ruptured aneurysm.[573,577] Furthermore, 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.[578] 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.[579]

A Cochrane review of perioperative administration of crystalloid and colloid fluids for open abdominal aortic surgery did not identify a superior regimen.[580] However, a recent prospective randomized study of elective open repair of AAA concluded that a more restrictive perioperative fluid regimen reduces complications and length of hospital stay.[581]

We recommend using cell salvage or an ultrafiltration device if large blood loss is anticipated.	
Level of recommendation	1 (Strong)
Quality of evidence	B (Moderate)
If the intraoperative hemoglobin level is <10 g/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	1 (Strong)
Quality of evidence	B (Moderate)

Cardiovascular monitoring. Central venous pressure and arterial line monitoring are suggested for all

patients undergoing OSR of an aortic aneurysm.[582] However, multiple randomized trials have shown no measurable benefit to the routine use of pulmonary artery catheters in nonselected patients.[583-586] Whereas transesophageal echocardiography 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.[587,588]

Perioperative MI is associated with adverse short- and long-term outcomes and can be prevented by early recognition of myocardial ischemia.[589-592] Electrocardiographic monitoring, using 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.[593] However, myocardial ischemia detection by transesophageal echocardiography in the form of wall motion abnormalities precedes ST-segment changes and is a more sensitive monitor for ischemia. Whereas troponin measurement after vascular surgery has been advocated,[594] routine measurement has not been associated with improved clinical outcomes.[586]

We suggest using pulmonary artery catheters only if the likelihood of a major hemodynamic disturbance is high.	
Level of recommendation	1 (Strong)
Quality of evidence	B (Moderate)
We recommend central venous access and arterial line monitoring in all patients undergoing open aneurysm repair.	
Level of recommendation	1 (Strong)
Quality of evidence	B (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	1 (Strong)
Quality of evidence	B (Moderate)
We recommend postoperative troponin measurement for all patients with electrocardiographic changes or chest pain after aneurysm repair.	
Levels of recommendation	1 (Strong)
Quality of evidence	A (High)

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.[586,595] Prospective randomized data support the use of forced air warming blankets rather than circulating water mattresses.[595] In addition, prospective studies support the use of low fresh flow rate anesthetic gases[596] and the use of intravenous fluid and blood warmers to maintain normothermia.[597]

We recommend maintaining core body temperature at or above 36°C during aneurysm repair.	
Levels of recommendation	1 (Strong)
Quality of evidence	A (High)

Role of the ICU. Increasingly, the care of the postoperative patient is occurring in a step-down unit or other monitored settings to best focus use of the ICU on those patients in greatest need.[598] Selective use of the ICU after aneurysm surgery is most effective if preoperative criteria, such as pre-existing 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.[599] 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.[600]

Goal-directed therapy using noninvasive monitoring of cardiac output by esophageal Doppler or lithium indicator dilution and pulse power analysis has been shown to improve short-term outcomes.[601,602] Monitoring cardiac output with a defined treatment protocol has also been shown to be cost-effective in the setting of major abdominal surgery.[603] Whereas studies focused on the care of patients with an aortic aneurysm have not been conducted, randomized trials have included patients with vascular disease.[604]

Fast-track surgical pathways or "enhanced recovery" pathways are being used increasingly to decrease length of stay and to 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.[605] The pathway included a limited retroperitoneal incision and specialized intraoperative retractors.[605] A recent trial confirmed benefit in 101 patients randomized to a fast-track surgery care pathway, which included no bowel preparation, reduced fasting, and 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; $P < .001$) was reduced along with the incidence of postoperative complication (16% vs 36%; $P = .039$).

We recommend postoperative management in an ICU 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	1 (Strong)
Quality of evidence	A (High)

Nasogastric decompression and perioperative nutrition. Routine nasogastric decompression is not recommended after aortic surgery. A Cochrane review examined 37 studies involving 5711 patients randomized to routine or selective nasogastric decompression after emergency or elective abdominal surgery.[606] Selective decompression was associated with a decreased risk of pulmonary complications without untoward adverse effects.[606] Although postoperative malnutrition is uncommon after EVAR, given the anticipated short length of hospital stay,[607] a risk for malnutrition exists for patients who undergo open aneurysm repair, particularly those with pre-existing renal insufficiency.[608] Early feeding reduces the likelihood of malnutrition, as demonstrated in a randomized trial of 128 patients undergoing colorectal and abdominal vascular surgery.[609]

We recommend optimization of preoperative nutritional status before elective open aneurysm repair if repair will not be unduly delayed.	
Level of recommendation	1 (Strong)
Quality of evidence	A (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	1 (Strong)
Quality of evidence	A (High)
We recommend parenteral nutrition if a patient is unable to tolerate enteral support 7 days after aneurysm repair.	
Level of recommendation	1 (Strong)
Quality of evidence	A (High)

Prophylaxis for deep venous thrombosis. Early mobilization and shorter length of stay have 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 was <2% and 1%, respectively.[610,611] The risk of postoperative deep venous thrombosis after open aneurysm repair was first highlighted by Olin and associates, who performed postoperative venography in 50 consecutive patients.[612] Although most patients were asymptomatic, 21% had evidence of an acute deep venous thrombosis, predominantly within the calf veins.

Whereas 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 of 3 or 4) or high (Caprini risk score >5) risk.[613] 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 >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 OSR, or effectiveness.[614-621]

We recommend thromboprophylaxis that includes intermittent pneumatic compression and early ambulation for all patients undergoing OSR or EVAR.	
Level of recommendation	1 (Strong)
Quality of evidence	A (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	2 (Weak)
Quality of evidence	C (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.[110,152] 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, MI, and pneumonia.[622] A hemoglobin concentration of <7 g/dL has been recommended as a transfusion threshold for a number of high-risk conditions in both critical and ambulatory care.[623] Given the prevalence of coronary artery disease among patients undergoing vascular surgery, blood transfusion for a hemoglobin concentration of <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.[624] In this regard, motivated by a desire to reduce the established risks of blood transfusion, to decrease blood use, and to lower costs, a recent Cochrane analysis supported more restrictive guidelines for all patients, including those with cardiovascular disease.[625] 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 g/dL or below.	
Level of recommendation	2 (Weak)
Quality of evidence	C (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.[626] The geriatric population warrants special consideration, and incorporation of acetaminophen is recommended in the postoperative pain plan.[626]

A Cochrane analysis reviewed 1498 patients enrolled in 15 trials who were treated with either epidural or systemic opioid analgesia, most often after open aortic surgery.[627] The method of pain control had no impact on 30-day mortality, but initial pain scores, duration of ventilation, postoperative respiratory failure, gastrointestinal bleeding, ICU length of stay, and incidence of MI were all reduced among patients treated with epidural analgesia.[627,628] Epidural anesthesia may also be beneficial for patients with COPD.[629] Complications after the placement of an epidural catheter are uncommon but include epidural abscess and hematoma.[630]

There is limited evidence that a preincisional transversus abdominis plane block decreases the use of pain medication after major abdominal surgery.[631,632] A Cochrane review of eight studies with 358 participants found that a transversus abdominis plane block reduced opioid consumption in a subset of studies and had no impact on nausea, vomiting, or sedation scores.[633]

We recommend multimodality treatment, including epidural analgesia, for postoperative pain control after OSR of an AAA.	
Level of recommendation	1 (Strong)
Quality of evidence	A (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%.[634-636] Although an endoleak may often resolve without intervention, some require immediate or delayed treatment to prevent aneurysm rupture. In addition, 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.[637,638] There are four main types of endoleak.[371,639] 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 (IA). A type IA endoleak most often occurs in the presence of a short or severely angulated neck or 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.[640-642]

Whereas a small endoleak may seal and can be observed, it is preferable that when a type I endoleak is identified at the time of repair, every attempt should be made to treat it 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.[643] Endostaples may reduce the risk of endograft migration and a type IA endoleak, but long-term data are limited.[643,644]

Other options for type IA endoleak treatment include embolization with coils or glue,[645,646] proximal extension with a chimney approach,[376] and conversion to a fenestrated endograft.[375] A type IB endoleak is treated with distal extension of the iliac limb if repeated angioplasty fails to eliminate the endoleak. It may be necessary to extend the endograft to the external iliac artery with coil occlusion of the HA. Conversion to open repair should be considered in the presence of a persistent type IA endoleak.[647]

Type II endoleak. Persistent filling of the aneurysm sac from patent lumbar arteries or the IMA constitutes a type II endoleak.[634-636] Type II endoleaks are the most common endoleak, present at the time of repair in up to one-fourth of patients. When a type II endoleak is identified at the time of the procedure, treatment is not indicated as at least 50% will spontaneously resolve.[640,645,646,648] The incidence of type II endoleak at 6 months is 10% to 15%.[649-651] Factors that increase the risk of a persistent type II endoleak include a patent IMA, number and diameter of patent lumbar arteries (especially L3 and L4 lumbar arteries), and ongoing anticoagulation.[652-655]

The fate of persistent type II endoleaks is variable. Aneurysm sac size may decrease in up to 25%,[649,656] remain stable in 50% to 70%,[657] 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[658]; however, expansion in sac diameter >10 mm is uncommon.[659]

Treatment of a type II endoleak includes embolization of the IMA or lumbar arteries with coils or glue,[646] direct translumbar injection of the aneurysm sac,[639] transcaval embolization,[660,661] and laparoscopic ligation of the IMA and lumbar arteries,[662] all with variable success rates. Up to 60% of treated aneurysms continue to expand, requiring multiple procedures and in some cases explantation with conversion to open repair.[663,664] Stent graft preservation with oversewing of the IMA and lumbar arteries from within the sac has been reported.[665-667] Type II endoleak remains a challenge to treat effectively.[668]

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, the type and size of patent inflow and outflow vessels, and the presence of symptoms.[669,670] Selective intervention appears both safe and cost-effective.[659,671]

Type III endoleak. A type III endoleak occurs when there is incomplete seal between components or component separation and less frequently is due to fabric erosion. The aneurysm sac becomes repressurized 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.[640,641]

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 discernible endoleak. It may be caused by blood flow that is undetectable at the limits of the imaging modality, pressure transmission through fabric,[672,673] or accumulation of a serous ultrafiltrate across a microporous fabric.[635] Endotension is less common with the newer generation grafts. Management should be individualized and may entail observation, relining of low-porosity endografts, or rarely explantation and conversion to open repair.

We recommend treatment of type I endoleaks.	
Level of recommendation	1 (Strong)
Quality of evidence	B (Moderate)
We suggest treatment of type II endoleaks associated with aneurysm expansion.	
Level of recommendation	2 (Weak)
Quality of evidence	C (Low)
We recommend surveillance of type II endoleaks not associated with aneurysm expansion.	
Level of recommendation	1 (Strong)
Quality of evidence	B (Moderate)
We recommend treatment of type III endoleaks.	
Level of recommendation	1 (Strong)
Quality of evidence	B (Moderate)
We suggest no treatment of type IV endoleaks.	
Level of recommendation	2 (Weak)
Quality of evidence	C (Low)
We recommend open repair if endovascular intervention fails to treat a type I or type III endoleak with ongoing aneurysm enlargement.	
Level of recommendation	1 (Strong)
Quality of evidence	B (Moderate)
We suggest open repair if endovascular intervention fails to treat a type II endoleak with ongoing aneurysm enlargement.	
Level of recommendation	2 (Weak)
Quality of evidence	C (Low)
We suggest treatment for ongoing aneurysm expansion, even in the absence of a visible endoleak.	
Level of recommendation	2 (Weak)
Quality of evidence	C (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 2 years or more after implantation.[674-676] Factors that predispose to migration include hostile neck anatomy, inadequate device fixation, and progressive aortic dilation and elongation.[674,677-679]

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 because treatment with an aortic cuff is often associated with continued risk of device migration.[680] Alternatives include proximal extension with a branched or fenestrated endograft.[375] Fenestrated EVAR for EVAR failure is technically complex because the existing endograft may interfere with rotational torque and visualization of the radiopaque markers.

Limb occlusion. Nearly 25% of all arterial reinterventions after open repair are due to limb occlusion, and they are most common in patients with associated occlusive disease.[681] Limb occlusion appears to be greater in women and in grafts extending to the femoral artery. Isolated limb occlusion usually is manifested with claudication, but occlusion of the entire graft may be manifested 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.[682] 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 2 months and nearly all within the first year after EVAR.[394,683,684] Nonsupported limbs are at especially high risk of limb occlusion.[685] However, stented limbs may also occlude by fabric infolding and kinking between stents.[683,686] Whereas 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.[687,688] Acute endograft limb occlusion often is manifested with worsening claudication rather than with 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 ultrasound or by a reduction in ABI, can be treated by stent placement. An occluded limb after EVAR or open repair can be treated by thrombectomy, pharmacolytic therapy with secondary endovascular or local surgical intervention, or femoral-femoral or axillofemoral bypass. Mechanical balloon thrombectomy is less likely to be successful for treatment of an endograft limb thrombosis because of difficulty in 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 it is placed in the treatment of aneurysmal disease, exceeds 80%.[689,690]

We recommend that follow-up of patients after aneurysm repair include a thorough lower extremity pulse examination or ABI.	
Level of recommendation	1 (Strong)
Quality of evidence	B (Moderate)
We recommend a prompt evaluation for possible graft limb occlusion if patients develop new-onset lower extremity claudication, ischemia, or reduction in ABI after aneurysm repair.	
Level of recommendation	1 (Strong)
Quality of evidence	A (High)

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%,[691] historically, it has been the indication for intervention in up to 25% of redo aortic surgery.[681] Although controversial, the risk of graft infection may be lower after EVAR than after open repair, perhaps because of delivery of the endoprosthesis through a completely enclosed system.[686,692] However, the EVAR 1 trial had a comparable incidence of device infection between EVAR and open repair during a 4-year follow-up period.[682] Similarly, in a recent analysis of >45,000 Medicare beneficiaries, graft infections or aortoenteric fistulas 4 years after EVAR or open repair were comparable for both groups (~0.3%).[86] Likewise, Vogel and coworkers reported a nearly identical 2-year incidence of graft infection (<0.2%) for OSR or EVAR in a review of 14,000 patients undergoing aneurysm repair.[693] Graft infection after EVAR or open repair may occur in isolation or with an aortoenteric fistula.[694,695]

Whereas aortic graft infection presents on average 3 years or later after open repair, endograft infection often is manifested earlier for reasons that remain unclear.[681,696,697] Femoral artery extension of a prosthesis increases the incidence of graft infection from 1% to 3%.[698] Other predisposing factors for graft infection include the need for surgical revision and emergent indication for initial surgery. Generalized sepsis, groin drainage, pseudoaneurysm formation, and ill-defined pain may be presenting symptoms, and staphylococcal organisms are the most frequent isolates.[699] 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.

The conventional treatment of graft infection is staged excision of all infected graft material with extra-anatomic reconstruction, particularly in the presence of extensive contamination and gross purulence.[681,700-704] In situ reconstruction using the femoral vein, a silver- or antibiotic-impregnated graft, or a cryopreserved allograft represents an additional surgical option, 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.[700,705-709]

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. Furthermore, hooks and the suprarenal segment may be embedded into the aortic wall or covered with a pseudointima, requiring careful removal to avoid injury to the operator and aortic wall as well as to preserve the aortic neck for closure or in situ reconstruction. Despite the introduction of endograft resheathing techniques, attendant renal ischemia is an established risk. Percutaneous drainage and antibiotic therapy have been suggested for patients unfit to undergo open repair.[696,710-712]

An aortoenteric fistula can complicate a graft infection in 1% to 2% of patients.[694,699] Although the duodenum is most frequently affected, all viscera, including small and large bowel, have been implicated.[695,699] A common presentation is upper gastrointestinal bleeding, as a herald bleed, which may progress to exsanguinating hemorrhage.[713] 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.[714-716] Bleeding is more common when the anastomosis erodes into the gastrointestinal tract, whereas sepsis and abscess formation are more common with paraprosthetic fistula involving the body of the graft. Treatment strategies are similar to those for primary graft infections but must include closure of the visceral defect.[717] Whereas 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.[718,719]

Prevention of an aortic graft infection. Recommendations for antibiotic prophylaxis after placement of an aortic prosthesis following OSR or EVAR have historically followed guidelines for the prevention of infective endocarditis with a prosthetic heart valve. Recent guidelines have sought to reduce the indications for antibiotic prophylaxis, particularly with the publication of the National Institute for Health and Clinical Excellence 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.[720] A recent report has observed a small but statistically significant increase in infective endocarditis cases in the United Kingdom since the implementation of the National Institute for Health and Clinical Excellence recommendations.[721] Although limitations exist in this study and causation has not been established, concerns have been raised.[722] Several investigations noted a relationship between dental procedures and infective endocarditis in high-risk patients.[447] Current European Society of Cardiology and ACC/AHA guidelines recommend ongoing use of antibiotic prophylaxis for patients with a prosthetic valve undergoing high-risk procedures.[723,724] High-risk procedures, as defined in both guidelines, include 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.[724] Both guidelines noted that there is no compelling evidence that bacteremia resulted from respiratory tract procedures, gastrointestinal or genitourinary procedures, and dermatologic or musculoskeletal procedures, and prophylaxis was not recommended for patients undergoing these procedures unless the procedures were performed in the presence of an infection.[724] 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 urologic procedures, especially the elderly and those with cancer or who are immunocompromised, require antibiotic prophylaxis.[725,726] Both the European and ACC/AHA 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 OSR or EVAR, we suggest antibiotic prophylaxis to prevent graft infection before 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 before respiratory tract procedures, gastrointestinal or genitourinary procedures, and dermatologic or musculoskeletal procedures if the potential for infection exists or the patient is immunocompromised.

We recommend antibiotic prophylaxis to prevent graft infection before 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 OSR or EVAR.	
Level of recommendation	1 (Strong)
Quality of evidence	B (Moderate)
We suggest antibiotic prophylaxis before respiratory tract procedures, gastrointestinal or genitourinary procedures, and dermatologic or musculoskeletal procedures for any patient with an aortic prosthesis if the potential for infection exists or the patient is immunocompromised.	
Level of recommendation	2 (Weak)
Quality of evidence	C (Low)
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	1 (Strong)
Quality of evidence	A (High)
We recommend prompt evaluation for possible aortoenteric fistula in a patient presenting with gastrointestinal bleeding after aneurysm repair.	
Level of recommendation	1 (Strong)
Quality of evidence	A (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	1 (Strong)
Quality of evidence	B (Moderate)
In patients presenting with an infected graft with minimal contamination, we suggest in situ reconstruction with cryopreserved allograft.	
Level of recommendation	2 (Weak)
Quality of evidence	B (Moderate)
In a stable patient presenting with an infected graft, we suggest in situ reconstruction with femoral vein after graft excision and débridement.	
Level of recommendation	2 (Weak)
Quality of evidence	B (Moderate)
In unstable patients with infected graft, we recommend in situ reconstruction with a silver- or antibiotic-impregnated graft, cryopreserved allograft, or PTFE graft.	
Level of recommendation	1 (Strong)
Quality of evidence	B (Moderate)

Incisional hernia. Retroperitoneal incisions for aortic aneurysm repair may lead to denervation of the eleventh 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 muscle atrophy in 7% to 15%.[405,727] 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.[728,729] Should a midline incision be used, continuous suturing technique and avoidance of rapidly absorbable sutures are recommended.[730] Wound infections of the abdominal incisions are rare (0.4%).[729]

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.[731-735] In the era before CT imaging, Szilagyi and colleagues 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%).[734] Subsequent studies have reported an incidence after open repair of between 4% and 10% at 10-year follow-up.[732] In one study of 511 patients, Kaplan-Meier analysis revealed a probability of para-anastomotic aneurysm of 0.8% at 5 years, 6.2% at 10 years, and 35.8% at 15 years.[733] This observation has been confirmed by others, particularly the risk of femoral pseudoaneurysm formation among patients treated with an aortobifemoral graft.[732,735] Indolent graft infection should be suspected in all pseudoaneurysms.

Given the inability to precisely differentiate anastomotic disruption from degenerative aneurysmal dilation, indications for repair of 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.[736,737] Infrarenal and fenestrated endografts have been used with chimney as well as snorkeling techniques.[738-742]

Recommendation for postoperative surveillance

Systematic reviews by the Society for Vascular Surgery 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. Magnetic resonance imaging was more sensitive than CT angiography and contrast-enhanced 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 OSR, an anastomotic aneurysm or aneurysmal dilation in the adjacent visceral aorta or iliac arteries may occur in 1%, 5%, and 20% of patients at 5, 10, and 15 years.[732,735] Thus, abdominal and pelvic CT imaging is recommend every 5 years after OSR.

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 >5%.[364] Unfavorable anatomy for endovascular repair predisposed to most ruptures, which developed from type I or type III endoleaks with sac enlargement.[743-745]

Surveillance imaging modality. Initially recommended surveillance protocols were consistent with those used by FDA-sponsored pivotal trials, with CT imaging at 1 month, 6 months, and 12 months and yearly thereafter. The 6-month CT scan can be eliminated from routine surveillance if the 1-month scan shows no concerning endoleak or sac enlargement.[746,747]

Color duplex ultrasound, contrast-enhanced color duplex ultrasound, and three-dimensional contrast-enhanced ultrasound have all been shown to be accurate in detecting type I and type III endoleaks as well as sac enlargement.[748-752] Ultrasound eliminates radiation exposure, reduces cost, and avoids use of a nephrotoxic contrast agent. Further surveillance with ultrasound is safe if CT imaging 1 year after EVAR demonstrates no endoleak and stable sac size[747,752] or for those patients with a type II endoleak and a stable aneurysm size.[751] A new endoleak, graft migration, or aneurysm sac growth >5 to 10 mm should prompt further evaluation with a CT scan.

Surveillance outcomes. Surveillance noncompliance rates approach 60%,[753,754] with gaps observed 3 to 4 years after EVAR, particularly among patients of advanced age, with Medicaid eligibility, or after treatment at a low-volume center.[753,755] Although the risks 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.[754,756] Not all late ruptures are preceded by endoleak or sac enlargement, which suggests that not all late ruptures can be prevented by vigilant surveillance.[757,758]

Summary. Current recommendations for surveillance after EVAR include a CT scan at 1 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 ultrasound 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 type I or

type III endoleaks. Abdominal and pelvic CT imaging should be performed every 5 years after OSR or EVAR.

We recommend baseline imaging in the first month after EVAR with contrast-enhanced CT and color duplex ultrasound imaging. In the absence of an endoleak or sac enlargement, imaging should be repeated in 12 months using contrast-enhanced CT or color duplex ultrasound imaging.	
Level of recommendation	1 (Strong)
Quality of evidence	B (Moderate)
If a type II endoleak is observed 1 month after EVAR, we suggest postoperative surveillance with contrast-enhanced CT and color duplex ultrasound imaging at 6 months.	
Level of recommendation	2 (Weak)
Quality of evidence	B (Moderate)
If neither endoleak nor AAA enlargement is observed 1 year after EVAR, we suggest color duplex ultrasound when feasible, or CT imaging if ultrasound is not possible, for annual surveillance.	
Level of recommendation	2 (Weak)
Quality of evidence	C (Low)
If a type II endoleak is associated with an aneurysm sac that is shrinking or stable in size, we suggest color duplex ultrasound for continued surveillance at 6-month intervals for 24 months and then annually thereafter.	
Level of recommendation	2 (Weak)
Quality of evidence	C (Low)
If a new endoleak is detected, we suggest evaluation for a type I or type III endoleak.	
Level of recommendation	2 (Weak)
Quality of evidence	C (Low)
We suggest noncontrast-enhanced CT imaging of the entire aorta at 5-year intervals after open repair or EVAR.	
Level of recommendation	2 (Weak)
Quality of evidence	C (Low)

COST AND ECONOMIC CONSIDERATIONS IN ANEURYSM REPAIR

The complexity and intensity of aortic aneurysm treatment result in significant costs and resource utilization. These initial costs include imaging, preoperative risk management, operating room, personnel, implants, and recovery. Long-term follow-up and imaging have costs, as does 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 United States, with lower cost estimates in Canada (U.S. $16,000) and other countries.[759,760] 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.[761,762] Whereas open repair is slightly more expensive during the initial hospitalization, no significant differences are seen in long-term follow-up because of the need for surveillance imaging and reinterventions 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 with open repair, patients undergoing EVAR generally have better health-related quality of life within the first 12 months, although there is no significant difference beyond the first year.[763]

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 is cost-effective compared with open repair, with an incremental cost-effectiveness ratio of $22,826.[764] However, contemporary Markov models using data from the DREAM,[765] EVAR 1,[682] OVER,[766] and ACE[362] randomized trials showed EVAR to be cost-effective on the basis of 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.[767] 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.[768]

Most of the data for cost-effectiveness pertain to elective cases, for which expected morbidity and mortality can be managed through selection of patients 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.[769]

A single screening ultrasound for AAA in asymptomatic men older than 65 years has been shown to be cost-effective in the United Kingdom[261] and through Markov modeling.[770] In the United Kingdom, the cost per life-year saved with screening was $1173, which is less costly than screening programs for breast, cervical, and colorectal cancer.[771] The cost-effectiveness of screening for younger cohorts, women, and reimaging intervals for small aneurysms remains uncertain. Early treatment of a small aneurysm, <5 cm in diameter, is not cost-effective in comparison to serial imaging.[772]

Because of the lower perioperative 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 beneficiaries.[773,774] Whereas cost-effectiveness results can vary among different populations of patients and health care 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 health care policy that sustains health care coverage for all.

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

Advances in biotechnology, drug discovery, and the engineering sciences hold significant promise for the development of new diagnostic tests, bioactive compounds, and intraoperative tools and devices that will enhance the care of the patient with an aortic aneurysm. Research is needed (1) to ascertain genetic or other biologic factors that accurately measure the lifelong risk for development of an aortic aneurysm; (2) to discover pharmacologic agents to limit aneurysm enlargement; (3) to characterize biomarkers or imaging-derived determinants of rupture risk; (4) to design prostheses that resist infection and thrombosis; (5) to develop tools, intraoperative imaging or robotic systems, and improved endovascular grafts that facilitate repair in the presence of challenging anatomy and improve the safety and accuracy of device deployment; and (6) to identify approaches that reliably treat type I and type 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 AAA in the application of existing technology that would benefit from further investigation. Furthermore, given the role of sex differences in the pathophysiologic process 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. Whereas the following list is not meant to be comprehensive, future research efforts should consider addressing these 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 OSR 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 that incorporates risk of repair, risk of aneurysm rupture, and anticipated life expectancy?
- Would a risk-benefit scoring scheme that incorporates risk of repair, risk of aneurysm rupture, and anticipated life expectancy assist in mutual decision-making between the surgeon, the patient, and the patient's 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 venous thrombosis needed for the patient undergoing EVAR?
- Does the patient undergoing OSR and at low or moderate risk for deep venous 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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APPENDIX (online only).

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:

#	Searches	Results
1	exp Aortic Aneurysm, Abdominal/di, dh, dt, ri, rt, su, th, us [Diagnosis, Diet Therapy, Drug Therapy, Radionuclide Imaging, Radiotherapy, Surgery, Therapy, Ultrasonography]	12817
2	exp abdominal aorta aneurysm/di, dm, dt, rt, su, th [Diagnosis, Disease Management, Drug Therapy, Radiotherapy, Surgery, Therapy]	14097
3	("abdominal aorta aneurysm*" or "abdominal aortic aneurysm*" or "aortic abdominal aneurysm*").mp.	39942
4	(((volume* or PET or spiral or helical or 3d or beam or 4d or "whole body") adj (ct or cat or cts or scan*)) or agent* or "cat scan*" or chemotherap* or "ct scan*" or diagnos* or drug* or image or images or imaging or intervention* or manag* or medication* or microtomograph* or "micro-tomograph*" or operat* or pharmacotherap* or radiotherap* or reconstruction* or repair* or resect* or SPECT or surg* or therap* or tomograph* or treat* or ultrasonograph* or ultrasound* or xray* or "x-ray*").mp.	28727775
5	3 and 4	36488
6	1 or 2 or 5	41292
7	exp meta analysis/	220499
8	exp Meta-Analysis as Topic/	49995
9	exp Randomized Controlled Trial/	863768
10	exp triple blind procedure/	186
11	exp Double-Blind Method/	381145
12	exp Single-Blind Method/	63322
13	exp latin square design/	560
14	exp Placebos/	333090
15	exp Placebo Effect/	9804
16	((meta adj analys*) or (randomized adj3 study) or (randomized adj3 trial) or (randomised adj3 study) or (randomised adj3 trial) or "pragmatic clinical trial" or (doubl* adj blind*) or (doubl* adj mask*) or (singl* adj blind*) or (singl* adj mask*) or (tripl* adj blind*) or (tripl* adj mask*) or (trebl* adj blind*) or (trebl* adj mask*) or "latin square" or placebo* or nocebo*).mp,pt.	2185771
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Scopus.

1 TITLE-ABS-KEY("abdominal aorta aneurysm*" OR "abdominal aortic aneurysm*" OR "aortic abdominal aneurysm*")
2 TITLE-ABS-KEY(((volume* or PET or spiral or helical or 3d or beam or 4d or "whole body") W/1 (ct or cat or cts or scan*)) OR agent* OR "cat scan*" OR chemotherap* OR "ct scan*" OR diagnos* OR drug* OR image OR images OR imaging OR intervention* OR manag* OR medication* OR microtomograph* OR "micro-tomograph*" OR operat* OR pharmacotherap* OR radiotherap* OR reconstruction* OR repair* OR resect* OR SPECT OR surg* OR therap* OR tomograph* OR treat* OR ultrasonograph* OR ultrasound* OR xray* OR "x-ray*")
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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