

Clinical Practice Guidelines for Endovascular Abdominal Aortic Aneurysm Repair: Written by the Standards of Practice Committee for the Society of Interventional Radiology and Endorsed by the Cardiovascular and Interventional Radiological Society of Europe and the Canadian Interventional Radiology Association



T. Gregory Walker, MD, Sanjeeva P. Kalva, MD, Kalpana Yeddula, MBBS, Stephan Wicky, MD, Sanjoy Kundu, MD, Peter Drescher, MD, B. Janne d'Othee, MD, MPH, Steven C. Rose, MD, and John F. Cardella, MD

J Vasc Interv Radiol 2010; 21:1632–1655

Abbreviations: AAA = abdominal aortic aneurysm, CIN = contrast medium–induced nephropathy, EVAR = endovascular aneurysm repair, FDA = Food and Drug Administration, IIA = internal iliac artery, IMA = inferior mesenteric artery, SIR = Society of Interventional Radiology

PREAMBLE

THE membership of the Society of Interventional Radiology (SIR) Standards of Practice Committee represents experts in a broad spectrum of interventional procedures from both the private and academic sectors of medicine. Generally, Standards of Practice Committee members dedicate the vast majority of their professional

time to performing interventional procedures; as such they represent a valid broad expert constituency of the subject matter under consideration for standards production.

Technical documents specifying the exact consensus and literature review methodologies as well as the institutional affiliations and professional credentials of the authors of this docu-

ment are available upon request from SIR, 3975 Fair Ridge Dr., Suite 400 N., Fairfax, VA 22033.

METHODOLOGY

SIR produces its Standards of Practice documents using the following process. Standards documents of relevance and timeliness are conceptualized by the Standards of Practice Committee members. A recognized expert is identified to serve as the principal author for the standard. Additional authors may be assigned dependent upon the magnitude of the project.

An in-depth literature search is performed using electronic medical literature databases. Then, a critical review of peer-reviewed articles is performed with regard to the study methodology, results, and conclusions. The qualitative weight of these articles is assembled into an evidence table, which is used to write the document such that it contains evidence-based data with

From the Department of Radiology, Division of Vascular Imaging and Intervention (T.G.W., S.P.K., K.Y., S.W.), Massachusetts General Hospital, Boston, Massachusetts; Department of Medical Imaging (S.K.), Scarborough General Hospital, Toronto, Ontario, Canada; Department of Interventional Radiology (P.D.), West Allis Memorial Hospital, Milwaukee, Wisconsin; Department of Radiology, Division of Interventional Radiology (B.J.D.), University of Maryland Medical Center, Baltimore, Maryland; Department of Radiology (S.C.R.), University of California San Diego Medical Center, San Diego, California; and Department of Radiology (J.F.C.), Geisinger Health System, Danville, Pennsylvania. Received May 4, 2010; final revision received May 24, 2010; accepted July 11, 2010. Address corre-

spondence to T.G.W., c/o Debbie Katsarelis, 3975 Fair Ridge Dr., Suite 400 N., Fairfax, VA 22033; E-mail: tgwalker@partners.org

T.G.W. is a paid consultant for Medtronic (Minneapolis, Minnesota). S.P.K. has research funded by Angiodynamics (Queensbury, New York). S.C.R. is a paid consultant for Sirtex (Lane Cove, Australia) and Terumo (Somerset, New Jersey) and is an owner of or shareholder in Boston Scientific (Natick, Massachusetts) and Sirtex. None of the other authors have identified a conflict of interest.

© SIR, 2010

DOI: 10.1016/j.jvir.2010.07.008

respect to content, rates, and thresholds.

When the evidence of literature is weak, conflicting, or contradictory, consensus for the parameter is reached by a minimum of 12 Standards of Practice Committee members using a Modified Delphi Consensus Method (Appendix A) (1). For purposes of these documents, consensus is defined as 80% Delphi participant agreement on a value or parameter.

The draft document is critically reviewed by the Standards of Practice Committee members, either by telephone conference calling or face-to-face meeting. The finalized draft from the Committee is sent to the SIR membership for further input/criticism during a 30-day comment period. These comments are discussed by the Standards of Practice Committee, and appropriate revisions made to create the finished standards document. Prior to its publication the document is endorsed by the SIR Executive Council.

The current guidelines are written to help determine appropriate preprocedural, intraoperative, and postoperative management of patients with abdominal aortic aneurysms (AAAs) who are treated with endovascular aneurysm repair (EVAR). Although practicing physicians should strive to achieve perfect outcomes, in practice all physicians will fall short of ideal outcomes to a variable extent. Therefore, in addition to quality improvement case reviews conducted after individual complications, outcome measure thresholds should be used to assess treatment safety and efficacy in ongoing quality improvement programs. For the purpose of these guidelines, a threshold is a specific level of an indicator that, when reached or crossed, should prompt a review of departmental policies and procedures to determine causes and to implement changes if necessary. Thresholds may vary from those listed here; for example, patient referral patterns and selection factors may dictate a different threshold value for a particular indicator at a particular institution. Therefore, setting universal thresholds is difficult and each department is urged to adjust its thresholds as needed to meet its specific quality improvement program situation.

SIR is committed to the basic prin-

ciples of outcomes-focused, evidence-based medicine. Ideally, every Standards of Practice Committee recommendation would be based on evidence derived from multiple prospective randomized trials of adequate statistical power. SIR recognizes the potential pitfalls of developing evidence-based standards for EVAR and of making recommendations regarding EVAR based solely on literature studies. However, these difficulties are far outweighed by the potential improvements in safety and treatment efficacy that may be gained by implementing the key lessons learned from the existing peer-reviewed scientific literature that has evaluated outcomes of EVAR procedures. The recommendations presented in this document are intended to guide clinical practice rather than to mandate the use of specific algorithms. The authors fully anticipate that these guidelines will be appropriately revised when future studies are available.

INTRODUCTION

AAAs are a leading cause of death worldwide, with increasing incidence and prevalence. In the United States, AAAs occur in an estimated 5%–7% of the population older than 60 years of age, often as an unrecognized disease (2). With a high propensity for rupture, AAAs are the 15th leading cause of death overall in the United States (3) and the 10th leading cause of death in men older than age 55, with approximately 9,000 AAA-related deaths occurring annually (2,3). EVAR for AAA represents an advance in patient care, serving as an effective alternative to traditional open surgical AAA repair, and is now the most common treatment method for AAA repair in the United States. Continued technologic refinements have occurred since the first documented EVAR in 1991, with multiple United States Food and Drug Administration (FDA)-approved devices now available. The reported technical and clinical outcomes of EVAR now parallel or exceed the same outcome parameters for open surgical repair (4). The procedure has resulted in reduced operative times, decreased intraoperative blood loss and transfusion requirements, decreased perioperative morbidity and mortality, and

reduced intensive care unit and hospital lengths of stay. Although these reductions, together with the improved patient recovery time, may decrease the immediate costs of AAA repair, this initial financial advantage may be offset by the costly lifelong follow-up imaging that is recommended after EVAR (5). The Dutch Randomized Endovascular Aneurysm Management trial (6–8) concluded that EVAR provides only a marginal overall survival benefit, and is associated with a substantial, if not prohibitive, cost increase. Additionally, although EVAR has been shown to reduce death and complication rates in the first month after the procedure compared with open repair (7,9), subsequent longer-term analysis of these randomized trials showed a sustained benefit in terms of aneurysm-related mortality up to 4 years, but the overall survival difference did not persist beyond the first two postoperative years (8,10,11).

EVAR outcomes are strongly dependent on appropriate patient and device selection; physician factors such as training, experience, and procedure volume; and various institutional factors (12). EVAR procedures can be extremely challenging and thus require operators who have substantial endovascular experience and refined technical skills. Successful outcomes further depend on meticulous assessment of the pertinent vascular anatomy and proper preprocedural planning. These guidelines are intended for use in assessing the standard of care expected from all physicians who perform EVAR procedures. The most important processes of care are: (i) preprocedural imaging and planning, (ii) appropriate graft and patient selection, (iii) performance of the procedure, (iv) postprocedural surveillance, and (v) management of EVAR-related complications. The outcome measures or indicators for these processes are indications, success rates, and complication rates, and are assigned threshold levels.

DEFINITIONS

Abdominal Aortic Aneurysm

Abdominal Aortic Aneurysm is a permanent pathologic dilation of the abdominal aorta. Currently, intervention is indicated when (i) the di-

iameter reaches 5.5 cm or is 2.5 times the normal aortic diameter or (ii) there is aneurysm growth at a rate exceeding 1 cm per year (13–17). Repair is always indicated for AAA rupture or when there is a symptomatic aneurysm (eg, back pain), which may be a sign of impending rupture (18). AAAs are classified according to their anatomic relationship to the renal arteries, as this significantly affects treatment. An infrarenal AAA has an intervening normal aortic segment (proximal neck) of at least 10 mm between the renal arteries and the most cephalad extent of the aneurysm, whereas a juxtarenal aneurysm extends to the renal artery level, with a normal-caliber aorta above. A suprarenal AAA not only involves the renal arteries, but extends cephalad so that the superior mesenteric artery and celiac artery arise from the aneurysmal aorta. AAAs may also extend distally beyond the aortic bifurcation to involve the common iliac arteries and occasionally the internal and/or external iliac arteries. A common iliac artery aneurysm has a diameter that reaches or exceeds 2 cm. Iliac artery involvement may significantly affect EVAR, as ancillary procedures may be necessary to ensure a successful outcome. AAAs may also be categorized by configuration: a fusiform AAA typically involves the entire circumference of an extended section of the aorta, and is a “true” aneurysm involving all three layers of the aortic wall. Saccular AAAs are more focal and localized and may be asymmetric or may occur as pseudoaneurysms that do not involve all mural layers.

Endovascular Aneurysm Repair

Endovascular abdominal aortic aneurysm repair is defined as treatment of an AAA through image-guided placement of a stent-graft device (endoprosthesis) within the native abdominal aorta, securing device fixation to the vascular wall proximal and distal to the diseased aneurysmal segment(s), thus eliminating AAA sac pressurization.

EVAR stent-graft devices or endoprostheses have three components, consisting of (i) a delivery system for graft introduction and deployment; (ii) a high radial force, self-expanding

metallic stent framework that supports the endoprosthesis and allows for vascular attachment; and (iii) graft fabric that excludes the aneurysm and serves as a new conduit for blood flow. Successful EVAR requires suitable proximal and distal landing zones for stable fixation and complete sealing of the endoprosthesis to the vascular wall. Various devices have different methods of fixation to the vascular wall. The methodology for graft attachment above the AAA (ie, proximal fixation) may be divided into suprarenal and infrarenal fixation. Suprarenal fixation devices employ a bare metallic stent component that extends proximally above the fabric-covered stent-graft, with the latter positioned immediately below the most inferiorly located renal artery, so that apposition is to the vascular tissue in the neck located between the renal arteries and the AAA. The bare metallic stent of such a device extends cephalad, anchoring the graft to vascular tissue adjacent to and above the renal arteries. This has been proposed as a more effective means of ensuring proximal fixation in the presence of unfavorable anatomy (eg, short infrarenal neck length, circumferential thrombus or calcification, severe angulation, conical neck configuration) (19). Because there is no fabric attached to the stent, the mesenteric and renal arteries remain perfused, although concerns have been raised regarding the potential risks of embolization to or occlusion of the renal and/or mesenteric arteries by the suprarenal components (20). Several studies have reported the safety and efficacy of suprarenal fixation (21–25), with similar rates of renal dysfunction in EVAR performed with suprarenal and infrarenal fixation (24). However, there are reports of renal infarction and renal artery occlusions in patients with preexisting renal arterial disease (26,27), as well as of visceral compromise and arterial occlusion (28,29) with the use of suprarenal fixation.

Infrarenal fixation devices do not have any components that extend above the renal arteries; the proximal margin of the endoprosthesis is positioned immediately below the most inferiorly located renal artery. Fixation is achieved through the radial force of the metallic stent framework of the device, where it is in apposition to the

vascular tissue located between the renal arteries and the AAA. Some infrarenal fixation devices also have small “barbs” or retention hooks to further ensure fixation.

Stent-graft design allows for customization to an individual patient’s vascular anatomy. All current FDA-approved devices have a modular design that permits the combination of various graft components into a composite conduit that is patient-specific for the particular AAA morphology that is to be treated. There are three basic graft configurations: (i) a completely modular stent-graft design that is composed of a graft body and unilateral limb extension that is paired with a contralateral docking limb to achieve a bifurcated graft that simulates the native aortic anatomy; (ii) a one-piece, fully supported, unibody self-expanding endoprosthesis that is positioned on the aortic bifurcation and to which another modular component is added and is extended cephalad to attach immediately distal to the lowest renal artery (this latter component is available with suprarenal or infrarenal fixation); and (iii) an aortouniiliac graft that requires the addition of a surgically created femoral artery-to-femoral artery crossover bypass conduit to provide perfusion of the contralateral lower extremity. This graft configuration is a modification of a bifurcated endoprosthesis and is used when iliac stenotic or occlusive disease does not permit bilateral introduction of graft components. It is also frequently used when EVAR is performed for AAA rupture, as the potentially time consuming placement of a contralateral docking limb is avoided.

Endoleaks are defined as continued perfusion of the residual aneurysm sac after EVAR (30). Endoleaks, along with graft migration, represent the most common threat to EVAR durability as an alternative therapy to open surgical aneurysm repair and are the most commonly occurring complication following EVAR (31). Endoleaks are classified according to the source of continued sac perfusion (30,31). Type I endoleak occurs when there is continued sac perfusion as a result of inadequate fixation at the proximal (type IA endoleak) or distal (type IB endoleak) attachment sites of the endoprosthesis. The lack of graft fixation allows arterial blood to enter the an-

eurysm sac through the space between the stent-graft and the vascular endothelium. These endoleaks invariably require further intervention, as they rarely resolve spontaneously; when identified during or after EVAR, every effort should be made to correct this endoleak, as the continued arterial perfusion places the patient at risk for aneurysm rupture. If this endoleak cannot be corrected, open surgical conversion may be necessary. Type II endoleak, the most common type, allows perfusion of the residual AAA sac through patent branch vessels that normally arise from the abdominal aorta (eg, lumbar artery and inferior mesenteric artery [IMA]). Reversal of arterial flow through a branch vessel arising from the aneurysm provides inflow to the residual AAA sac while another patent arterial branch serves as the outflow vessel. Although the majority of these resolve spontaneously, persistent type II endoleaks that result in continued expansion of the residual AAA sac require intervention. Type III endoleaks occur when there is a functional problem with the device such as a defect in the graft fabric or separation (ie, disarticulation) of one or more modular components of the stent-graft, allowing residual AAA sac perfusion. This type does not spontaneously resolve and requires additional intervention, as this places the patient at significant risk for aneurysm rupture. Type IV endoleaks result from excessive graft porosity that allows continued residual sac perfusion; noncellular elements (eg, serum) may predominate as a transudate and may cause continued sac expansion. This endoleak type is significantly less common than it has been in the past as a result of manufacturer improvements in fabric composition. A type V endoleak (also termed endotension) is one in which there is continued post-EVAR expansion of the residual AAA sac without a demonstrable source of sac perfusion by imaging. Some investigators believe this actually represents one of the previous four types and that there has merely been a failure in demonstrating the endoleak type and source (31). Thus, one cause for continued pressurization of the aneurysm may be blood flow that is beyond the detection capabilities of currently available imaging technology (32,33). Other explana-

tions include transgraft passage of serous fluid ultrafiltrate into the aneurysm sac through microporous fabric (16,17). The treatment of endotension must be individualized based on the suspected cause of continued sac expansion.

INDICATIONS AND CONTRAINDICATIONS FOR EVAR

Despite advances in elective open surgical AAA repair, there is still considerable associated morbidity and mortality. Excellent outcomes are generally achieved in high-volume referral centers but there are still reported mortality rates as high as 7% and morbidity rates that approach 50% in some centers (34). Although open surgical repair has been the standard of care for the treatment of AAA, there are associated well recognized disadvantages, including lengthy operative, anesthesia, and recovery times, as well as a potentially prolonged hospitalization that involves use of the critical care unit. Additionally many patients are poorly suited for open repair because of coexisting medical conditions such as cardiac, pulmonary, or renal dysfunction, which present a high operative and/or anesthesia risk, and thus unacceptably high potential complication rates (35,36). Endovascular abdominal aortic aneurysm repair has been used as an alternative therapy for patients who are unsuitable for open repair as well as for those who would be well suited for traditional open surgical AAA repair. Various clinical trials have questioned the benefit of EVAR in patients with poor surgical risk and have also failed to definitely establish the sustained benefit and durability of EVAR (6,8,10,37). Indications for EVAR are currently the same as for open repair, although presently there are ongoing randomized prospective clinical trials that seek to determine whether intervention with EVAR for smaller-diameter AAAs may be of benefit (38–40). The recently published results of the Positive Impact of Endovascular Options for Treating Aneurysms Early trial (41) showed that early treatment with EVAR and image-based surveillance, with aneurysm treatment as clinically indicated, appear to be equally safe alternatives for patients with small an-

eurysms of 4.0–5.0 cm in diameter. The study noted, however, that the results are based on early data that might change as longer-term data accrue.

Patient suitability for EVAR is determined by various clinical and anatomic factors. Anatomic factors may directly affect the technical outcome and long-term durability of EVAR, whereas clinical factors, such as medical comorbidities, affect morbidity and mortality (both early and late).

Anatomic factors that influence suitability for EVAR include adequacy of vascular access for device introduction; aneurysm morphology; neck length and morphology; and iliac artery involvement (42–45). Various series have suggested that there is considerable variability as to what percentage of patients will qualify for EVAR, depending on the criteria that are used to determine suitability (45,46).

Clinical factors affecting patient suitability for EVAR may be scored based on medical comorbidities. There have been various studies documenting a decreased incidence of periprocedural complications with EVAR compared with open surgical AAA repair (47,48). However, this has not been confirmed in all studies, as the Dutch Randomized Endovascular Aneurysm Management trial demonstrated similar incidences of cardiac complications for EVAR and open surgical AAA repair of 5.3% and 5.7%, respectively (6). As cardiac deaths represent the primary cause of periprocedural and delayed mortality for EVAR, an individual's cardiac risk should be stratified before treatment. Limiting preprocedural patient assessment solely to cardiac risk fails to stratify for other comorbid conditions that may also profoundly impact outcomes. Thus, classification systems have been developed that attempt to predict morbidity and mortality, including the Acute Physiology and Chronic Health Enquiry score, the Physiological and Operative Severity Score for the enumeration of Mortality and morbidity, the American Society of Anesthesiology classification system, and the Society for Vascular Surgery/American Association for Vascular Surgery classification system. These scoring systems include the age and cardiac, pulmonary, and renal status of the patient and are intended to

represent factors that are likely to affect major morbidity and mortality associated with EVAR or open AAA repair. Preprocedurally, the clinical suitability of a patient for EVAR should be quantified as much as possible, based on any medical comorbidities. Endovascular abdominal aortic aneurysm repair should be considered as having an intermediate to high cardiac risk that ranges from 3% to 7% (49). If preexisting cardiac disease is identified, appropriate intervention should be considered before elective EVAR. Preexisting renal insufficiency has a known association with poor EVAR outcomes (50,51). Renal status should be assessed before EVAR and renal protection strategies should be used to address any renal dysfunction.

Diabetes mellitus has been shown in the European Collaborators on Stent Graft Techniques for AAA Repair registry to be associated with a significantly higher risk of device-related complications and a higher early mortality rate (41). However, patients with insulin-controlled type 2 diabetes had lower rates of endoleaks and fewer secondary interventions than diet-controlled type 2 diabetic patients and nondiabetic patients (52).

Regarding patient age and AAA repair, one study (53) has suggested that the mortality rate after open or endovascular AAA repair in carefully selected octogenarians seems acceptable but is higher than the mortality rate in younger patients, whereas another concluded (54) that EVAR suitability is not influenced by age, aneurysm size, or patient fitness. Additionally, one single-center study (55) cited significantly higher endoleak, open conversion, and renal infarction rates ($P < .05$) among a subgroup of patients older than 75 years, and also showed significantly elevated ($P = .0011$) aneurysm-related morbidity and mortality rates.

Patient preference for EVAR versus open repair should be considered when appropriate; when informing patients about the choice, one can present figures from major trials. The midterm (4-year follow-up) results of the EVAR-1 trial (9) showed a significantly lower aneurysm-related mortality rate for patients in the EVAR group: 4% compared with 7% for open repair. The lower postoper-

ative morbidity and mortality rates, the shorter hospital stay, and the potential for use of local or regional anesthesia may favor EVAR, but these need to be balanced against higher late complication and repeat intervention rates and the need for long-term surveillance (8,10). Until recently, there were no objective studies substantiating whether there was a patient preference for EVAR. However, a recent study examining patient preferences for elective future AAA repair (56) showed 84% preferred EVAR, 13% preferred open repair, and 3% could not express a preference. The results of this study support the general trend toward offering EVAR to patients in whom it is technically feasible.

Contraindications for EVAR generally are related to anatomic or clinical factors that render a patient unsuitable for this therapy. Poor anatomic preprocedural patient selection is generally associated with a higher risk for complications and compromised long-term outcomes (57,58). Similarly, patients at high risk for significant perioperative or postoperative morbidity and mortality because of severe medical comorbidities may be poor candidates for any form of AAA repair. Decisions regarding EVAR versus open surgical AAA repair should be made in concert with other physicians involved in the patient's care.

EVAR in Patients Requiring Urgent or Emergent Repair

Recent studies of national trends in the United States have observed increasing use of EVAR in the emergency management of ruptured AAA, with decreasing mortality rates (59,60). Results in teaching and high-volume hospitals have been significantly superior to those in nonteaching and low-volume institutions (60). Achieving optimal EVAR results for ruptured AAA requires establishment of a treatment protocol involving the emergency department, the endovascular team, anesthesiology, and the operating room personnel (61). Hemodynamically stable patients in whom ruptured AAA is suspected should undergo emergency computed tomographic (CT) angiography, whereas those in

unstable condition should be taken directly to the operating room, limiting fluid resuscitation to maintenance of patient consciousness and a systolic blood pressure of 70 mm Hg (ie, "hypotensive hemostasis") to limit ongoing hemorrhage (62,63). Inflation of a compliant aortic occlusion balloon in a supraceliac or para-renal location may be useful in hemodynamically unstable patients or in anatomic situations that prevent expeditious EVAR (22). If a preprocedural CT angiography examination could not be obtained, determination of the aneurysm morphology, landing zones, and appropriate device must be based on intraoperative angiography. Endoprostheses used in emergency EVAR for ruptured AAA have included bifurcated and aortouniiliac devices (61,64–66).

One must be vigilant for development of abdominal compartment syndrome in these patients. This well recognized complication after EVAR for ruptured AAAs occurs in hemodynamically unstable patients in whom a large retroperitoneal hematoma and diffuse visceral edema cause elevated intraabdominal pressure and multiorgan dysfunction (61,66,67). It is seen most frequently in patients requiring an aortic occlusion balloon, massive transfusion, or conversion to an aortouniiliac device, and in those with coagulopathy (67).

PREPROCEDURAL ASSESSMENT BEFORE EVAR

Preoperative Imaging and Planning

Preprocedural imaging is essential for evaluating the suitability of an AAA for EVAR, as there are multiple anatomic factors that may preclude this as a treatment option. Imaging helps to gauge the degree of technical difficulty, allows for selection of the most appropriate endograft, predicts whether any ancillary procedures may be necessary before or during EVAR, and may also help predict immediate and long-term outcomes. The imaging modalities employed consist of multidetector CT angiography, magnetic resonance (MR) imaging/MR angiography, duplex ultrasonography (US), and catheter angiography. Usually the preoperative imaging is initially done with contrast-enhanced CT, supple-

Table 1
Aortic Angle and Tortuosity Scoring

Grade	Tortuosity Index	Aortic Angle
0	≤ 1.05	160°–180°
1	> 1.05 but ≤ 1.15	140°–159°
2	> 1.15 but ≤ 1.20	120°–139°
3	> 1.2	< 120°

mented with three-dimensional volumetric reformation images. A preoperative AAA scoring system has been proposed and is derived from multiple morphologic characteristics (68). The scoring system ranges from the most optimal morphologic features (grade 0) to the most severe (grade 3). A summation of the various scored components may be used to preoperatively assess suitability for EVAR and predict outcomes. Higher scores are associated with poorer outcomes.

Aneurysm Morphology Scoring

Aneurysm morphology may impact endograft delivery and deployment, may present a risk for distal embolization if the AAA is filled with laminar thrombus, and may also influence the long-term performance of the device and the ability to achieve complete aneurysm exclusion (68). In addition to the thrombus burden, other important determinants include aneurysm tortuosity and angulation. Aortic tortuosity may be quantified by an aortic tortuosity index and an aortic angle. The aortic tortuosity index is defined by dividing L1 by L2, where L1 is the distance along the central lumen line between the lowest renal artery and the aortic bifurcation (without deviation into saccular areas) and L2 is the straight-line distance from the lowest renal artery to the aortic bifurcation. The aortic angle is the most acute angle in the centerline between the lowest renal artery and the aortic bifurcation. Ideally, both the aortic angle and tortuosity index are measured from spatially correct three-dimensional data. They are scored as in **Table 1**, whereas thrombus within the AAA (or iliac arteries) may be scored as in **Table 2**.

The aneurysm neck morphology is a critical determinant of suitability for EVAR, as this is the site where

Table 2
Thrombus Scoring

Grade	Amount of Thrombus
0	No visible thrombus
1	< 25% of the cross-sectional area
2	25%–50% of cross-sectional area
3	> 50% of cross-sectional area

the endograft attaches proximally and where secure fixation must be achieved. The proximal AAA neck is defined as the length of normal caliber aorta between the inferior-most renal artery and the beginning of the aneurysm, whereas the distal neck is defined as the length of normal caliber aorta between the caudal aspect of the aneurysm and the aortic bifurcation (68). Neck characteristics such as diameter, amount of calcification, presence or absence of thrombus, and angulation are important parameters that affect endograft fixation and should therefore be evaluated when considering a patient for EVAR (69). A proposed scoring system for the aortic neck (68) includes the following parameters: neck length and diameter, the angle between the flow axis of the suprarenal aorta and the infrarenal neck, the angle between the flow axis of the infrarenal neck and the body of the aneurysm, and the amount of thrombus and calcification that are present in the neck. The flow axis of the aneurysm is defined as the line running from the proximal neck of the aneurysm to the aortic bifurcation. At the proximal neck, morphologic features should be evaluated, as they are critical for securing adequate device fixation. For the distal aortic neck, the length is usually not an important factor unless a tube endograft is used; in fusiform aneurysms, there may not be a distal aortic neck. The diameter, angulation, and degree of calcium and thrombus, however, are important for all features of the abdominal aortic distal neck if one is present, as they impact endograft delivery and deployment. Additionally, if the diameter of the distal neck is excessively small, this might potentially impact endograft delivery and deployment, constrain the self-expanding endoprosthesis components, and potentially lead to limb thrombosis.

The shape or configuration of the aneurysm neck is also a consideration when considering morphology. Necks have been classified as flared, parallel, irregular, conical, barrel, or hourglass in configuration (70). The parallel configuration is the most favorable for satisfactory EVAR outcomes, whereas other configurations may be less favorable for achieving fixation at the proximal attachment site (70). The flared and conical neck configurations may make it difficult to determine the actual point of transition from neck to aneurysm (70). Similarly, if there is a distal neck to the AAA, the actual transition point may be difficult to determine, as there is often a gradual distal tapering of the AAA.

Aortic neck scoring.—The proximal aortic neck length is scored as in **Table 3**, the proximal neck angle as in **Table 4**, the proximal neck diameter as in **Table 5**, and the proximal neck calcification or thrombus as in **Table 6**. A similar grading scheme can be used to score the distal aortic neck and is defined as the angle between the flow axis of the distal infrarenal neck and the aortic bifurcation (68).

Quality of vascular access for device introduction.—The morphology of the common femoral and iliac arteries is critical in allowing vascular access for device introduction. Additionally, iliac artery morphology is critical to obtaining adequate fixation at the distal attachment site or sealing zone of the endoprosthesis limbs and maintaining limb patency. Thus, the pelvic arteries may be graded based on diameter, calcification, tortuosity, and length (68). The diameter of the iliac arteries may limit device delivery and will affect the adequacy of the distal attachment sites of the endoprosthesis. Stenotic or occlusive disease impacts device delivery and outflow patency, whereas aneurysmal disease affects the distal seal zone. Typically a minimum diameter of 7 mm is required for main body device delivery; the presence of any focal or diffuse stenoses as well as the overall diameter of a vascular segment length should be considered. Arterial calcification may also impact device delivery; calcification is typically quantified as none, 25% or less of the vascular segment length, 25%–50% of the vascular segment length, or more than 50% of the segment length or circumference. Vessel tortuosity must

Table 3
Aortic Neck Length Scoring

Grade	Aortic Neck Length (mm)
0	≥ 25
1	≥ 15 but < 25
2	≥ 10 but < 15
3	≤ 10

Table 4
Proximal Aortic Neck Angle Scoring

Grade	Aortic Neck Angle
0	≥ 150°
1	≥ 150° but < 135°
2	≥ 135° but < 120°
3	≤ 120°

Table 5
Proximal Aortic Neck Diameter Scoring

Grade	Aortic Neck Diameter (mm)
0	< 24
1	≥ 24 but < 26
2	≥ 26 but < 28
3	> 28

also be considered when assessing the quality of access. An iliac tortuosity index may be determined in the same manner as was described for aortic tortuosity, with L1 the distance along the central lumen line between the common femoral artery and the aortic bifurcation and L2 the straight-line distance from the common femoral artery and the aortic bifurcation. Likewise, an iliac angle may be determined, which is the most acute angle in the pathway between the common femoral artery and the aortic bifurcation. Ideally, both the iliac angle and iliac tortuosity index are measured from spatially correct three-dimensional data and are scored as in **Table 7**.

Branch vessels.—Although recognition of the number and caliber of patent aneurysm branch vessels is important, the true preprocedural risk for a type II endoleak remains unknown. However, it is known that patent branch vessels predispose to type II endoleak. Aortic branch vessels are scored as follows: grade 0, no lumbar arteries, IMA, or other branches visibly patent; grade 1, one patent

lumbar artery or patent IMA; grade 2, at least two patent branch vessels (lumbar arteries or IMA with none more than 4 mm in diameter; and grade 3, any one of the following with at least two patent branch vessels: paired lumbar arteries, low-resistance outflow vessel such as a noncovered accessory renal artery, or IMA more than 4 mm in diameter.

Optimal Endograft Type for Patient Anatomy

Selection of an appropriate stent-graft for an individual patient is performed before the procedure after the accurate analysis of the diagnostic images. There are currently five FDA-approved devices available for use in the United States:

The Zenith Flex AAA endovascular graft (Cook, Bloomington, Indiana) is a three-piece modular bifurcated graft made of woven polyester fabric with a self-expanding exoskeleton of modified Gianturco stainless-steel Z stents to provide support. The proximal end of the graft body has a series of 12 bare metal stents with caudally angled protruding barbs for suprarenal fixation to the aortic wall. The stainless-steel composition of the stents render this device incompatible with MR for post-procedural surveillance. Zenith endograft diameters range between 22 mm and 36 mm for the proximal component and from 8 mm to 24 mm for the iliac limbs. There are also proximal aortic body extensions, as well as proximal aortic converter cuffs that are designed for conversion of the bifurcated graft design to an aortouni-iliac configuration. Conversion to this latter configuration also requires the use of an available iliac plug. The Flexor delivery system uses a flexible, kink-resistant sheath that ranges from 18 F to 22 F in inner diameter and has a hydrophilic coating to facilitate device introduction. Because of the available large aortic component diameters, this graft is indicated for implantation in AAAs with neck diameters as large as 32 mm and lengths that are greater than 10 mm; the neck angulation should be less than 60°.

The Powerlink AAA endovascular graft (Endologix, Irvine, California) is a unibody bifurcated design graft that is made of expanded polytetrafluoroethylene that is attached to a self-ex-

panding cobalt/chromium endoskeleton. The tubular component of the unibody graft is available in 25- and 28-mm diameters; the iliac limb components have a distal diameter of 16 mm. There are tubular and flared iliac limb extensions available. Proximal tubular aortic extensions are available without suprarenal fixation or with a 20-mm-long bare metal suprarenal fixation design. This unibody design device is positioned so the graft bifurcation is placed at the aortic bifurcation to prevent any caudal migration. The aortic extension is then introduced and positioned with the proximal fabric-covered end immediately below the most inferiorly located renal artery origin. Because of the unibody graft design, cannulation and contralateral placement is unnecessary. The IntuiTrak introducer system employs a 19-F inner body hydrophilic delivery sheath. Placement of a contralateral 9-F sheath is required to allow for positioning of the contralateral iliac component. This smaller diameter requirement for the contralateral limb allows for percutaneous access and may be advantageous if there is a unilaterally small caliber or diseased access vessel.

The Excluder AAA endoprosthesis (W.L. Gore and Associates, Flagstaff, Arizona) is a two-piece modular device consisting of a main-body ipsilateral limb and a contralateral limb, both constructed with a self-expanding nitinol stent skeleton lined by expanded polytetrafluoroethylene. The original fabric of this graft had porosity issues that resulted in high rates of post-EVAR residual AAA sac expansion, but this problem has resolved since replacement of the original fabric. The graft main body is designed for infrarenal fixation, with a series of small proximal fixation anchors or “whiskers” designed to imbed within the arterial wall at the proximal attachment site and impede caudal graft migration. The Excluder AAA endoprosthesis body ipsilateral limb component is available in diameters of 23 mm, 26 mm, 28.5 mm, and 31 mm, with aortic extender cuffs available. The contralateral iliac docking limb component has available tapered, straight, and flared configurations. The device is delivered through 20–24-F sheaths and is deployed with the sheathless Sim-pull system.

The AneuRx endoprosthesis (Med-

Table 6
Proximal Aortic Neck Calcification or Thrombus Scoring

Grade	Aortic Neck Diameter
0	Calcification < 25% of circumference; atheroma or thrombus (> 2 mm thick) < 25% of circumference
1	Calcification 25%–50% of circumference; atheroma or thrombus (> 2 mm thick) 25%–50% of circumference
2	Calcification > 50% of circumference; atheroma or thrombus (> 2 mm thick) > 50% of circumference

Table 7
Iliac Angle and Tortuosity Scoring

Grade	Tortuosity Index	Iliac Angle
0	≤ 1.25	160°–180°
1	> 1.25 but ≤ 1.5	121°–159°
2	> 1.5 but ≤ 1.6	90°–120°
3	> 1.6	< 90°

tronic, Minneapolis, Minnesota) is a two-piece modular device that has a main-body ipsilateral limb and a contralateral docking limb, both of which are constructed of low-porosity woven polyester fabric, with a self-expanding nitinol exoskeleton composed of multiple diamond shaped stents. The AneuRx bifurcated body ipsilateral limb component comes in diameters from 20 mm to 28 mm with and flared contralateral iliac docking limbs as well as straight and flared iliac extender cuffs. Aortic extension cuffs (4-cm length) are available in the same diameters as the proximal main body. The graft is introduced with the kink-resistant hydrophilic-coated Xcelerant delivery system. This device is indicated for use in AAAs with a maximum aneurysm neck diameter of 26 mm.

The Talent endoprosthesis (Medtronic) is a modular bifurcated device that employs suprarenal struts for proximal fixation. It is constructed of flexible low-porosity woven polyester that is reinforced with a self-expanding nitinol exoskeleton composed of a series of Z stents. Columnar strength is provided by a metallic bar that courses longitudinally along the medial portion of the main-body ipsilateral iliac limb component and is also incorporated into the limb components. The Talent main-body ipsilateral iliac limb component of this device ranges in diameter from 22 mm to 36 mm and is approved for implantation in AAAs with neck diameters as large as 32 mm and neck lengths of 10 mm or greater.

Iliac docking limbs are available in tapered, straight, and flared configurations. Aortic extension cuffs with bare metal suprarenal fixation stents are available in the same diameters as the main body component. The device is introduced with the hydrophilic-coated Xcelerant delivery system.

Informed Consent

Previously, patients with AAA faced the decision of whether to undergo surgery for a condition that is usually asymptomatic, but they now must decide between open repair or EVAR, and thus must fully understand the distinct risks and benefits of each alternative as presented during informed consent. When patients are fully engaged in the informed consent process, they are likely to be more satisfied with their decision and may experience better outcomes (71).

Patient Preparation

Because iodinated contrast medium is used during EVAR, if there is baseline renal dysfunction, preoperative renal protection strategies should be considered. As hydration remains the primary intervention for preventing contrast medium-induced nephropathy (CIN) (72), admission for vigorous overnight preoperative hydration may be useful. Intravenous sodium bicarbonate has been used for the prevention of CIN, despite conflicting study results on its use; its efficacy in preventing CIN should be clearer following the completion of prospective randomized trials currently under way (73). The efficacy of N-acetylcysteine in CIN prevention remains unclear because of the presence of positive and negative published clinical studies; it is hard to discourage its use given its lack of toxicity and inexpensive nature. Nephrotoxic drugs such as non-steroidal antiinflammatory drugs should

be avoided before contrast agent exposure.

PROCEDURE: TECHNICAL ASPECTS

Anesthesia

Traditionally, EVAR is performed under regional or general anesthesia. As mortality, postoperative complications, and length of stay are the consequences of surgical and anesthetic techniques, a change in the latter may contribute to reduce morbidity and costs after EVAR. Feasibility and small cohort studies (74–76) have reported encouraging results with the use of locoregional anesthesia for EVAR. Other authors, however, found no difference in cardiac mortality and morbidity in a retrospective cohort of patients receiving locoregional anesthesia or general anesthesia (76). There is no level I evidence for or against locoregional anesthesia in EVAR, as there are no randomized controlled trials or large prospective studies documenting its role in EVAR. However, the European Collaborators on Stent Graft Techniques for AAA Repair (77) data indicate that patients appeared to benefit when locoregional anesthesia was used for EVAR and suggest that these techniques should be used more often to enhance the perioperative advantage of EVAR. Additionally, a recent review of selected studies showed that, although patients undergoing locoregional anesthesia were less medically fit compared with those in the general anesthesia group, there were reductions in the cardiovascular support required during and after surgery, postoperative hospital stay, intensive care unit stay, and postoperative mortality and morbidity (78). Choice of anesthesia must be tailored to the individual patient, and will typically vary by operator and by institution.

Access for Device Introduction

Traditionally, access for EVAR has required arterial exposure via “cut-down” skin incisions with creation of an open arteriotomy for device introduction. In general, this process is safe, but requires practitioners experienced in open surgical technique. Newer endograft technology, smaller access sheath sizes, and the development of suture-mediated arterial closure devices has made complete percutaneous access for EVAR feasible in most patients. Since the technique of percutaneous closure of femoral arteries after EVAR was initially described by Haas et al in 1999 (79), there have been several published series reporting technical success rates ranging from 62% to 100% (80–82). There are currently no large multicenter randomized controlled trials comparing open with percutaneous access for EVAR. Several investigators have identified risk factors associated with potentially poor outcomes involving percutaneous access techniques for EVAR, including morbid obesity and heavy arterial wall calcification (83). There are well known complications that can occur following open access, including wound complications after hospital discharge (eg, infection, hematoma, or femoral neuropathy) and those arising from lymphatic disruption. In several authors’ experience (80,81,84), such late complications were nonexistent after percutaneous treatment. Totally percutaneous aneurysm repair requires good patient selection, technical expertise, and a thorough understanding of the device (84) and should be performed in a sterile environment or where open arterial access can be obtained rapidly if required.

Open surgical conduits fashioned to the common iliac artery or the aorta have been used to deal with unfavorable iliac anatomy during EVAR. A recently published 10-year experience regarding iliac conduits (85) details the various approaches to these conduits. In most cases, a retroperitoneal approach through a lower-quadrant oblique incision provides access to the common iliac artery or distal aorta. Preprocedural determination of optimal conduit placement may be made from preoperative imaging studies.

A more recent technique for deal-

ing with unfavorable arterial access anatomy uses an “internal endoconduit” (86,87), in which an iliac stent-graft is placed across the diffusely diseased or stenotic area. When adequate proximal and distal seal has been established with the endoconduit, aggressive angioplasty with “controlled rupture” of the iliac artery may be performed, with theoretically less concern for hemorrhage from rupture of a non-protected vessel. This may then permit safe passage of the delivery sheath or device.

Intraprocedural Imaging

Endovascular abdominal aortic aneurysm repair should be performed in an operating room or an angiography suite with a sterile configuration, a radiolucent operating table, and all necessary equipment should surgical conversion be required. State-of-the-art imaging equipment with a stationary fluoroscopy machine or dedicated new-generation C-arm should be used. As procedure times and potential radiation doses to the operators and the patient may be substantial, imaging equipment should be equipped with dose reduction technology such as variable-rate pulsed fluoroscopy. The operator should be familiar with dose reduction strategies such as collimation, appropriate x-ray tube-to-patient distance, and avoidance of redundant views.

If a C-arm is used intraprocedurally, one must be aware of the potential for delays that may occur during prolonged equipment use, as a result of the accumulation of heat energy that may limit x-ray production if insufficient cooling is allowed. There is also more rapid heat energy accumulation in patients with large body habitus and with use of magnification fluoroscopy. Appropriate use of pulsed fluoroscopy, collimation, and limitation of magnification and steep angulation may prevent delays from excessive x-ray tube heat loading.

Thorough knowledge of appropriate geometric adjustments of the imaging equipment relative to the vascular anatomy is essential for optimal outcomes. Parallax correction, as well as the appropriate use of oblique projections to optimally display the origins of vessels such the renal or internal iliac arteries, for example, is

required to insure appropriate placement of the graft components at the landing zones.

Some investigators have advocated routinely use of intravascular US during EVAR (88). Intravascular US use during EVAR requires complete familiarity with this technology in target site and branch vessel identification, landing zone measurement, neck analysis, and intraprocedural troubleshooting. Intravascular US may be beneficial in patients with excessive abdominal girth in whom intraoperative fluoroscopy and angiographic imaging do not provide adequate imaging for procedural precision during EVAR. Although the routine use of intravascular US might minimize renal contrast medium exposure, as well as reduce radiation exposure, in most centers intravascular US has an ancillary role (89), when and if it is used, and is not currently considered the standard of care.

There has been debate as to whether certain iodinated contrast agents may pose less risk of CIN than others. A recent metaanalysis of randomized controlled trials (90) comparing the nephrotoxicity of isoosmolar iodixanol with nonionic low-osmolar contrast media concluded that there is no significant reduction in the relative risk of CIN with the use of iodixanol compared with nonionic low-osmolar contrast media pooled together. In patients with renal insufficiency who require intraarterial administration of contrast medium, iohexol is associated with a greater risk of CIN than is iodixanol, whereas there is no significant difference in the relative risk of CIN between iodixanol and the other nonionic low-osmolar contrast media in patients at high risk. Thus, iodixanol or a nonionic low-osmolar contrast agent other than iohexol may be used in patients at risk for CIN (90).

Graft Body Deployment

By convention, the side of arterial access for introduction of the bifurcated main body of an endograft is termed ipsilateral, and the side from which the opposite iliac limb is introduced is referred to as contralateral. As the main body component requires a larger-caliber access vessel, the choice of the ipsilateral access is influenced by vessel diameter, tortuosity, degree of calcification, and/or throm-

bus, and the presence or absence of any stenotic or occlusive lesions. Other factors that may influence the choice of the ipsilateral access include the length and caliber of the distal landing zone, the anatomic level of the internal iliac artery (IIA), the orientation of the aortic bifurcation, and any AAA tortuosity or unusual anatomy that might complicate the process of cannulation, introduction, and deployment of the contralateral iliac limb.

An angiographic flush catheter, such as a calibrated pigtail catheter, is initially introduced via the contralateral access side to be used to obtain digital subtraction angiographic images that document the positions of the renal arteries. The ipsilateral access side is used for introduction of the graft body component through a vascular access sheath or in "bareback" fashion, using only the delivery system that contains the endograft. The device is advanced over a previously introduced super-stiff guide wire through the pelvic vessels, aortic bifurcation, and aneurysm, under fluoroscopic control, until the fabric-covered superior end of the endoprosthesis is positioned immediately below the renal arteries. Parallax-corrected digital subtraction angiographic images are obtained at renal artery level before and during deployment of the proximal end of the endoprosthesis. After deployment of the proximal portion of the bifurcated main body component, the remainder of the main body is deployed under fluoroscopic control. For modular-design devices, the ipsilateral limb component of the bifurcated main body endoprosthesis may also be deployed at this time, but if there is concern regarding stability of the endograft attachment at the neck, the operator may choose to place the contralateral limb before completing deployment of the ipsilateral limb. Placement of the contralateral limb may provide columnar strength to the endoprosthesis and thereby stabilize the proximal attachment site of the main body of the graft and prevent distal migration of the endograft below the renal arteries. This may be of particular importance in a "hostile" neck that is extremely short, angulated, or thrombus-laden.

Deployment of a single-piece unibody bifurcated graft design requires use of catheter and guide wire systems

that are directed across the aortic bifurcation from the contralateral side, with the unibody graft introduced directly into the aorta from the ipsilateral side. The contralateral limb component of the graft is then manipulated into position by retracting the wire introduced from the contralateral side, so that the graft is "seated" on the aortic bifurcation. The main body component is then extended cephalad, by means of an aortic extension cuff, to terminate just below the renal arteries. An aortic extension cuff, with or without suprarenal fixation, may also be used with a modular bifurcated graft if the main body component has been positioned at a level that is suboptimally low relative to the renal artery origins. The delivery system for an aortic extension cuff requires the same diameter access vessel as does a main-body endograft component.

Contralateral Limb Deployment

For modular devices, the contralateral limb must be "docked" with the main body component to complete a bifurcated configuration. This requires the selective retrograde catheterization (ie, cannulation) of an opening or "gate" that is incorporated into the main body component and that is indicated by radiopaque markers. Cannulation involves the use of a catheter to direct a guide wire through the gate and into the body of the bifurcated endoprosthesis. After confirming successful intraluminal passage of the guide wire into the endoprosthesis body and determining the correct contralateral limb length, the latter is delivered under fluoroscopic control over a super-stiff guide wire and is deployed with the appropriate overlap within the contralateral gate stub. The distal end of this limb should optimally terminate within 1 cm of the IIA origin to provide maximal columnar strength and prevent cephalad migration of the distal attachments if there are extreme AAA morphology changes following EVAR. If necessary, an iliac extension limb may be used to provide sufficient additional length to the ipsilateral or contralateral graft limb to reach the iliac bifurcation.

If gate cannulation is problematic and difficult to successfully achieve in the usual retrograde fashion, an alternate antegrade or crossover cannula-

tion technique may be used. A catheter is introduced from the ipsilateral limb and is used to direct a guide wire over the graft bifurcation or "flow divider" into the contralateral gate stub. The guide wire is subsequently passed into either the lower segment of the aneurysm or the common iliac artery, where it may be retrieved with a snare catheter introduced from the contralateral femoral site, thus providing guide wire access for passage of the contralateral iliac docking limb.

Intraoperative Ancillary Procedures

Various ancillary procedures may be required to ensure successful outcomes with EVAR. Some of these may be performed before EVAR, whereas others may be done intraoperatively. Careful analysis of the preprocedural images allows for planning of any ancillary procedures and determining appropriate timing. Some ancillary procedures, because of their complexity, may be best performed as a staged procedure in advance of EVAR, thus potentially avoiding an unacceptably lengthy EVAR procedure, whereas relatively simple supplemental procedures such as angioplasty may be performed intraoperatively. Certain procedures, such as creation of a surgical conduit for device introduction or creation of a cross-femoral bypass conduit, must be performed at the time of EVAR.

One of the most common ancillary procedures required during EVAR is angioplasty of stenotic iliac access vessels. If intravascular stents are required in access vessels, these should be placed after completion of EVAR rather than before, as stents may be dislodged during device introduction.

If a suitable distal landing zone in the common iliac artery is not present, the iliac limb may need to be extended into the external iliac artery. To prevent a type II endoleak from the IIA, embolization of one or both internal iliac arteries may be required. IIA embolization is performed preoperatively by some operators and intraoperatively by others. Some authors (91,92) state that the risk of bilateral IIA embolization is greater than in a unilateral procedure and includes potential complications such as erectile dysfunction, buttock claudication, and spinal cord, bladder, and colonic ische-

mia. Others believe that ischemic complications can be minimized by staged procedures before EVAR to allow time for collateral vessels to develop (93). Metaanalysis of the literature regarding IIA embolization (92) yields overall incidences of buttock claudication and erectile dysfunction of 28% and 17%, respectively. IIA revascularization by surgical bypass (94) or endovascular branched grafts are possible solutions to the potential problems associated with embolization, but add procedural complexity and possible additional complications. Additionally, a significant association between the position of embolization coils in the IIA and the development of ischemic symptoms has been suggested, prompting many investigators (95,96) to perform very proximal embolization and thus preserve collateral flow from the contralateral IIA and ipsilateral external iliac artery. Materials used for embolization include coils and nitinol plugs (eg, Amplatzer vascular plug; AGA Medical, Golden Valley, Minnesota).

Preoperative or intraoperative embolization of various aortic side branches, including patent lumbar artery and IMA, has been previously investigated as a method to decrease the incidence of type II endoleak. These studies have demonstrated feasibility but no clear benefit from the procedure (97,98). IMA patency has been described as a risk factor for type II endoleak formation after EVAR (98–100). A lower percentage of lumbar branches remain open after EVAR compared with IMAs (99), suggesting that lumbar leaks may be more likely to thrombose spontaneously. A previous report has shown technical success rates for preoperative embolization to be considerably higher for the IMA than for lumbar branches, at 100% versus 65%, respectively (97). In some centers, a patent IMA is embolized preoperatively or intraoperatively in all patients (101), whereas other centers have a more selective approach to IMA embolization.

The presence of concurrent vascular disease is often demonstrated angiographically during EVAR. Studies of incidental findings at diagnostic aortography (102,103) have documented stenoses of more than 50% in 20–40% of renal arteries, 10%–15% of visceral arteries, and 20%–30% of iliac

arteries, whereas other investigators (104) have documented angiographic progression of preexisting renal arterial disease. Although these findings raise the issue of prophylactic treatment of renal or mesenteric arterial disease during EVAR, there are no definitive data regarding such treatment of incidentally noted coexistent renal or mesenteric arterial disease during EVAR. Any interventions must be individualized, based on a consideration of the lesion severity and the presumed potential for disease progression versus the risk/benefit ratio of such an intervention.

Certain ancillary open surgical procedures may be performed during EVAR, in addition to creation of a vascular access conduit. Patients in whom an aortouniliac endograft configuration is used require a surgically created cross-femoral bypass conduit to provide perfusion of the contralateral lower extremity. Other ancillary surgical procedures include IIA bypass,endarterectomy, and patch angioplasty of the arterial access site.

Implantable telemetric pressure sensors have been developed for monitoring the pressure within the AAA sac following EVAR, as a noninvasive means of postoperative surveillance (105). The EndoSure wireless AAA pressure sensor (CardioMems, Atlanta, Georgia) is the only pressure sensor with FDA approval for acute implantation and initial confirmation of AAA exclusion. The EndoSure sensor is deployed through its own delivery catheter during the EVAR procedure and is positioned inside the aneurysm sac. The sensor is kept in place by means of a surrounding wire basket, which does not have any electrical function (106).

Intraoperative Assessment for Endoleak

After placement of all device components, a large-volume compliant balloon is introduced and is used to distend the proximal and distal graft attachments as well as the junctions of all overlapping modular components. This purpose of this maneuver is to eliminate any folds or “pleats” in the graft fabric that may be present as a result of incomplete expansion of the endograft. When present, these folds may serve as direct channels inter-

posed between the endograft and the arterial wall and may thus result in continued perfusion of the AAA sac, creating a type I endoleak at the landing zones or a type III endoleak at component junction sites. After use of the compliant balloon, a completion arteriogram is obtained to confirm patency of all graft components, to exclude endoleak, and to confirm patency of the renal arteries, IIA, and external iliac arteries. The imaging sequence should be prolonged, so that delayed AAA sac perfusion may be excluded. One must carefully evaluate the images for incomplete sealing at the proximal and distal endograft landing zones (type I endoleak), evidence of sac perfusion at a modular junction (type III endoleak), or evidence of retrograde perfusion of the AAA sac through patent aortic branch vessels (type II endoleak). Type I and type III endoleaks should be treated intraoperatively, making every attempt to eliminate such endoleaks before terminating the procedure, as the AAA remains at risk of rupture until these are corrected (31,107). Type II endoleaks are typically followed and do not require intraprocedural intervention, as the majority of these spontaneously resolve (108).

If the completion arteriogram demonstrates narrowing or kinking of the graft limbs, this should be corrected because these abnormalities will predispose to eventual thrombosis of the compromised component. Adjunctive angioplasty with or without stent placement may eliminate the abnormality. If completion images demonstrate any compromise of essential arteries (eg, renal artery, IIA) by a malpositioned graft, corrective measures such as intravascular stent placement should be undertaken to avoid complications.

Intraoperative Anticoagulation and Other Pharmacology

Intravenous heparin is routinely administered during vascular surgical procedures to prevent thrombosis resulting from stasis distal to hemostasis-controlling clamps or ligatures. The safety of intraoperative heparin administration during vascular surgical procedures has been demonstrated in large studies (109). Heparin is usually given intravenously and during

open AAA repair is typically administered 2–3 minutes before cross-clamping the aorta. The goals are to achieve adequate anticoagulation quickly and to maintain a steady anticoagulation concentration until cross-clamp removal. Although the risk of thrombotic events is not well established in this context, there is consensus on the necessity of anticoagulation during vascular surgery (110). Approximately half the physician respondents in one survey (111) regarding their use of heparin in elective AAA surgery reported using the same dose in all patients, whereas the remainder varied the dose, usually on the basis of weight or patient “size.” Most surgeons use 5,000 U but dosages vary from 400 to 20,000 U of heparin. The Seventh American College of Chest Physicians Conference on Antithrombotic and Thrombolytic Therapy (112) recently recommended a fairly high concentration of anticoagulation during surgery, with an initial dose of 100–150 U/kg of unfractionated heparin before cross-clamping. It was also recommended to supplement this dose every 50 min until circulation is reestablished. However, these recommendations apply only to situations in which there is no monitoring of heparin anticoagulation such as activated clotting time, and no anticoagulation monitoring device was specified. With a high variability of heparin responsiveness and plasma elimination half-life (113–115), anticoagulation monitoring may improve anticoagulation during vascular surgery. Although there are no trial data regarding routine use of intraoperative heparin during EVAR, the open surgical experience with heparinization has been widely applied to endograft procedures.

POSTOPERATIVE MANAGEMENT

Following an uncomplicated EVAR procedure, the patient is admitted for overnight observation, with appropriate analgesia for pain management, continuation of intravenous fluids as necessary, evaluation of appropriate dietary intake, and monitoring of the access sites, cardiopulmonary status, ambulation ability, and overall general postoperative state. Complicated procedures may require the administra-

tion of blood products, a potentially lengthier hospital stay that may include intensive care, and early postoperative evaluation with CT.

Unfortunately, there is a lack of prospective randomized trials concerning the medical management of patients who have undergone EVAR. Treatment is in line with the medical management of coronary artery disease including antiplatelet therapy and statins (116,117). Antiplatelet therapy may prevent complications such as graft-limb thrombosis and peripheral arterial disease (116,118). Especially in patients with PAD, aspirin, clopidogrel, and statins remain mainstays of medical management.

Postoperative Surveillance

Lifelong imaging surveillance of patients after EVAR is critical for (i) the detection and, if possible, the characterization of endoleaks; (ii) evidence of expansion or shrinkage of the residual AAA sac through measurement of aneurysm size, volume calculation, and identification of substantial changes in aneurysm dimensions; (iii) detection of mechanical changes in the stent-graft, such as migration, kinking, or fracture; and (iv) evaluation of the long-term performance of the endoprosthesis. Although CT angiography is the most commonly used examination for imaging surveillance, MR angiography, US, and digital subtraction angiography all have a role in endoleak detection and management (119).

Imaging modalities for surveillance.—Radiography continues to be used in basic post-EVAR surveillance despite the availability of advanced imaging modalities, and is still considered by some to be superior to CT for demonstrating the conformation of thoracic stent-grafts (120) and for detecting kinks in abdominal stent-grafts (121). Anteroposterior and lateral radiographs can depict stent-graft migration and component separation and oblique radiographs may improve detection of wire fractures, but radiographs have little if any role in surveillance for sac enlargement and endoleak detection. Improvements in multidetector CT and advances in image visualization tools may require a reevaluation of the role of radiographs (119).

Contrast-enhanced CT angiography remains the most widely used post-EVAR surveillance tool. The importance of CT angiography has increased with the advent of multidetector scanners and the routine availability of volumetric data sets. The combination of speed, reproducibility, and spatial and contrast resolution have made this the preferred method of imaging follow-up, despite the associated radiation dose and the potential for nephrotoxicity (122). High-resolution CT datasets allow reconstruction of thin transverse sections, multiplanar reformatted images, and highly accurate three-dimensional volumes. Additionally, CT angiography is able to detect endoleaks with a higher sensitivity than conventional angiography (123,124). Because endoleaks have variable flow rates and are thus detected at variable times after injection of contrast material, multiphasic CT angiography is essential; a typical protocol includes imaging before and after the administration of contrast agent in arterial and delayed phases. Precontrast images are used to differentiate calcification in the aneurysm sac from an endoleak. Delayed phase imaging is critical for demonstrating endoleaks that are not visualized during the arterial phase (125,126). Radiation dosage concerns have led some investigators to consider eliminating portions of the multiphasic surveillance CT angiography examination. Some believe precontrast scanning may be necessary in only the first post-EVAR examination (usually 30–90 days after the procedure), with all subsequent examinations acquiring only postcontrast images with the initial precontrast scan for comparison (127). As delayed postcontrast imaging depicts endoleaks with higher sensitivity than in the arterial phase, some advocate eliminating the arterial-phase acquisition altogether (128). However, arterial-phase images are important in planning exactly where to access the endoleak during trans-lumbar embolization and there are occasional cases in which an endoleak is seen only on arterial-phase images and not on delayed images (119).

Because impaired renal function occurs commonly in patients undergoing EVAR, repeated routine administration of iodinated contrast medium for post-EVAR surveillance may be problematic in a substantial number of

patients. Recently, investigators have proposed the use of nonenhanced CT volumetric analysis of the residual AAA sac for routine surveillance following the initial 3-month post-EVAR contrast-enhanced CT scan (129). If the interval volumetric change is 2% or less, this strategy of surveillance with nonenhanced CT volumetric analysis is continued. However, if there is a volumetric increase that exceeds 2%, contrast-enhanced CT angiography is immediately performed to evaluate for suspected endoleak (129).

MR angiography with gadolinium enhancement is capable of depicting endoleaks, but its performance depends on the endograft composition. Nitinol-based stents are generally suitable for MR imaging, whereas Elgiloy stents can obscure the lumen and stainless-steel stents cause artifact that renders the study nondiagnostic (130). In several studies involving small numbers of patients with predominantly nitinol stents, MR angiography was at least as sensitive as CT angiography (131–135), and in some cases demonstrated endoleaks that were not detected on CT angiography. In one case report (136), endotension was suspected in two patients with enlarging aneurysm sacs and no endoleak on CT angiography, but a subsequent MR angiogram demonstrated type II endoleaks. In a study of thoracic aortic endografts (137), MR angiography and CT angiography were found to be equally reliable for aneurysm size measurement and stent-graft position, but endoleak detection was not as accurate with MR angiography. Whereas most MR angiography studies rely on dynamic gadolinium-enhanced three-dimensional gradient-echo and delayed two-dimensional gradient-echo sequences, new techniques such as time-resolved MR angiography may allow better characterization of the endoleak type by demonstrating the temporal evolution of contrast material in the aneurysm sac, analogous to a conventional angiogram (135,138), in which dynamic information is displayed, as opposed to static CT angiography images (139). New blood-pool MR contrast agents may improve the detection of endoleaks with slow flow rates that are occult on CT angiography (140), although the clinical importance of these endoleaks is unknown.

US has been found by some investigators to be useful in post-EVAR surveillance, particularly as it is convenient, portable, safe, and inexpensive. It is also operator-dependent, with scan quality and protocols varying significantly from one institution to another (141). There have been technologic advances, such as the use of contrast agents and tissue harmonic imaging, that may improve diagnostics, whereas standardization of scanning techniques may reduce some of the quality variability. US may be of poor quality in obese patients or in the presence of considerable bowel gas. AAA size measurements obtained with US generally correlate well with those obtained with CT (142–145). However, when there are disagreements in the size measurements, US aneurysm measurements are typically smaller than those taken with CT (143,144). Some investigators have found that changes in AAA measurement may be misrepresented on US (143). The demand for precise, reproducible AAA size measurements has led to the persistence of CT angiography in the follow-up imaging regimen (119). Doppler US is capable of depicting endoleaks, but with extremely varied reported sensitivities compared with CT, ranging from 25% to 100% (141–144,146–148). This large range can partially be attributed to the differences in technique, technologist experience, and diagnostic criteria. A recent large study (149) involving 367 paired CT and US examinations after EVAR reported the sensitivity of US to be 68%, whereas a recent metaanalysis (150) studying US detection of endoleaks reported a similar sensitivity of 69%. The use of microbubble contrast agents improves the capability of US to detect endoleaks, as shown in several series (151–154) and in a recent metaanalysis (155). Contrast-enhanced US occasionally depicts endoleaks that could not be seen with CT angiography or conventional Doppler US (154). This suggests that contrast-enhanced US could have a role as a problem-solving tool in situations of suspected endotension (156,157). There are proponents of the use of US as the sole long-term post-EVAR surveillance method after stability of the aneurysm has been established by other imaging modalities (158). In a metaanalysis of 10 published studies comparing color

duplex sonography with contrast-enhanced CT (149,150,159), US had a sensitivity of 69% and a specificity of 91% in endoleak detection, with a higher sensitivity in detection of types I and III versus type II endoleaks. However, there have been recent studies suggesting that there is a high degree of correlation between CT imaging and color duplex Doppler US in the detection of clinically significant endoleaks (159–162). As a result, some investigators recommend annual post-EVAR surveillance with color duplex Doppler US alone if the first annual contrast-enhanced CT fails to demonstrate an endoleak or enlargement of the residual AAA sac (158,163). If US detects an endoleak or an increase in the residual AAA sac size at any time during surveillance, this would require additional imaging with contrast-enhanced CT for clarification (164). Although the risk for endoleak development diminishes over time in the presence of unremarkable surveillance studies, significantly delayed onset of new endoleaks is known to occur, and thus lifetime post-EVAR surveillance in some form is currently recommended (165).

Implantable telemetric pressure sensors have been developed and used as a means of enabling surveillance based on pressure monitoring within the aneurysm sac following EVAR. The previously described FDA-approved EndoSure wireless AAA pressure sensor (CardioMems) that may be used to monitor the pressure within the aneurysm sac is considered by some investigators to be an easy and convenient method for the surveillance of EVAR (105). The pressure sensor, the placement of which is integrated into the EVAR procedure, is designed to help to detect endoleaks with the aim of obviating further surveillance investigations, thereby reducing the need for costly and time-consuming imaging procedures as well as reducing radiation exposure.

Frequency of surveillance.—The current surveillance regimens for EVAR were derived empirically from early multicenter trials and codified in the instructions for use for the devices. Long-term data were not available at that time and thus these surveillance regimens were not data-driven. Protocols that were established for EVAR surveillance include serial three-phase

noncontrast and contrast-enhanced CT scans with CT angiographic reformatted images at 1, 6, and 12 months following the EVAR procedure and yearly thereafter (166). The cumulative contrast load from these CT scans is worrisome for its deleterious effect on renal function (167,168). The potential carcinogenic effect of the cumulative radiation dose for patients is more difficult to quantify, but still troubling (169,170). Finally, the cost associated with current EVAR surveillance regimens is significant, comprising 30%–35% of the total costs of EVAR follow-up during a 5-year period (171). Thus, if neither an endoleak nor residual AAA sac enlargement is detected on the initial annual post-EVAR contrast-enhanced CT scan, color duplex Doppler US may be considered as an alternative means of annual surveillance.

OUTCOMES

There is no longer debate about the early benefits of EVAR, including shorter hospital stays, less blood loss, shorter operating times, and lower early morbidity and mortality rates (4,9,10,172,173). Recent studies have focused on late outcomes, including the need for repeat interventions (172, 174–176). Thus, any assessment of EVAR outcomes should include the initial technical success, the long-term or delayed success, the rate of secondary intervention, and the complication rate.

Success Rates

A systematic review of the evidence of the safety and efficacy of elective EVAR in the management of asymptomatic infrarenal AAA (177) identified 606 reports, of which 61 met the inclusion criteria (three randomized and 15 nonrandomized controlled trials and 43 uncontrolled studies). There were 29,059 participants in total; 19,804 underwent EVAR. Deployment was successful in 97.6% of cases. Technical success (ie, complete aneurysm exclusion) was achieved in 81.9% at discharge and 88.8% at 30 days. Secondary intervention to treat endoleak or maintain graft patency was required in 16.2% of patients. Mean stay in the intensive care unit and mean hospi-

tal stay were significantly shorter following EVAR than open surgical repair. The 30-day mortality rates for EVAR were 1.6% (randomized controlled trials) and 2.0% in nonrandomized trials and case series, respectively. Technical complications comprised stent migration (4.0%), graft limb thrombosis (3.9%), endoleak (type I, 6.8%; type II, 10.3%; type III, 4.2%) and access artery injury (4.8%).

COMPLICATIONS

Endoleaks are the most common complication that occurs in association with EVAR (126,178–179) and are fully discussed in the Secondary Interventions section of this document. CIN is another complication that is associated with EVAR and is discussed in the section on intraprocedural imaging. Additional complications can occur during and after EVAR. The most commonly recognized are as follows:

Injuries Related to Vascular Access

Local access site injuries include hematoma, infection, and lymphocele (incidence, 1%–10%). Access artery injuries include thrombosis, dissection, pseudoaneurysm or arteriovenous fistula formation, vessel perforation or transection, and vascular avulsion. These have been reported in as many as 3% of EVAR cases. The incidence of these complications may potentially be decreased by careful preoperative planning, with meticulous attention to the quality of the access vessels (eg, diameter, tortuosity, and calcification). Judicious use of a surgical conduit may also decrease these complications. For postoperative pseudoaneurysms, surgical repair may be necessary, as opposed to US compression or thrombin injection, as the arterial defect may be excessively large. Similarly complications such as arteriovenous fistulas and large hematomas may require surgical intervention.

Infection

The incidence of endograft infection following EVAR is 0.2%–1% (180–182). Failure to recognize and treat this complication can result in sepsis and eventually death. Infection may result

from intraoperative contamination of the endograft, as a secondary infection from a remote source (183), or as a result of an aortoenteric fistula. The latter may be caused by stent-graft migration; vascular and/or bowel erosions from metallic components of the endoprosthesis, coils, or other devices; graft fabric failure; or aortitis or other vascular inflammatory processes. (116, 184–186). The procedural environment is another potential risk factor for stent-graft infection, with one study (187) showing that 62.5% of stent-graft infections occurred with procedures carried out in an interventional radiology suite, in contrast to 37.5% after procedures performed in a conventional operating room. Perioperative intravenous antibiotics (usually a cephalosporin) are administered to reduce the risk of graft infection. Inadequate antibiotic prophylaxis could increase the risk of postoperative infection (188). Reports have shown that patients with infected stent-grafts who undergo conservative management with antimicrobial therapy and percutaneous drainage can still survive, although most require removal of the infected prosthesis and either extraanatomic bypass or in situ graft replacement (187).

Ischemic Complications

Ischemic complications can occur as a result of thrombosis, embolization, or malpositioning of endograft components that cover branch vessels. These ischemic complications may be further subcategorized as follows:

Colonic ischemia.—There is the same 1%–3% incidence of bowel ischemia following open surgical AAA repair and EVAR (189,190). Postoperative colonic ischemia following AAA repair is a serious complication with a mortality rate of 50% within 1 month (189). Bowel ischemia following EVAR is usually characterized by a multifocal and patchy distribution, unlike that following open repair. The mechanism for EVAR-induced colonic ischemia may be one in which there is dislodgment of thrombotic and atheromatous deposits, with microembolization into the superior mesenteric artery or IMA as well as into the renal artery, IIA, and lower extremity arteries. This would account for the segmental and multifocal nature of the ischemia

(191). The presence of extensive circumferential thrombus or atheroma in the aneurysm neck has a well known potential for causing EVAR-related complications such as embolization and poor fixation or seal at the proximal endograft attachment site.

Spinal cord ischemia.—The European Collaborators on Stent Graft Techniques for AAA Repair data showed an incidence of spinal cord ischemia of 0.21% (192), indicating that this is a rare occurrence. Although the ischemic mechanism is unproven, it is likely related to embolization combined with interruption of collateral vessels from the lumbar and iliac circulation. The treatment is the same as for open repair and consists of drainage of cerebrospinal fluid and, in some cases, reestablishment of collateral flow (193–197).

Renal ischemia/infarction.—Thrombotic or embolic occlusion of one or more renal arteries can occur during EVAR, particularly when there is extensive thrombus or atheroma in the aneurysm neck. Partial or complete coverage of one or more renal arteries through malpositioning of the endograft occurs in fewer than 5% of cases (198,199). If endograft malpositioning is the cause of renal artery compromise, there are maneuvers for graft repositioning that may be attempted (200). If there is only partial renal artery coverage by the device, renal artery stent placement may be effective for improving renal perfusion (199).

Limb Occlusion

Limb thrombosis frequently occurred in unsupported stent-grafts but is much more infrequent with newer devices, with an incidence of 0%–5% (201). Most limb thromboses occur relatively soon after EVAR and are a result of kinking of components or poor outflow (202,203). Delayed limb occlusion may result from endograft migration or from development/progression of atherosclerotic occlusive disease in the outflow arteries (201). To evaluate for potential graft limb compromise, follow-up should include a thorough lower-extremity pulse examination and/or determination of ankle-brachial index. Similarly development of claudication, lower-extremity ischemia, or a decreased ankle-brachial index follow-

ing EVAR should be further assessed with imaging. The treatment options for an occluded limb following EVAR include thrombectomy or thrombolysis followed by secondary endovascular or open limited surgical intervention, or extraanatomic bypass surgery (eg, femoral–femoral or axillofemoral bypass graft).

Success/complications Thresholds

Although practicing physicians should strive to achieve perfect outcomes (eg, 100% success, 0% complications), in practice all physicians will fall short of this ideal to a variable extent. Thus, indicator thresholds may be used to assess the efficacy of ongoing quality improvement programs. For the purposes of these guidelines, a threshold is a specific level of an indicator which should prompt a review. “Procedure thresholds” or “overall thresholds” reference a group of indicators for a procedure, eg, major complications. Individual complications may also be associated with complication-specific thresholds. When measures such as indications or success rates fall below a (minimum) threshold, or when complication rates exceed a (maximum) threshold, a review should be performed to determine causes and to implement changes if necessary. For example, if the incidence of open surgical repair is one measure of the quality of EVAR, values in excess of the defined threshold should trigger a review of policies and procedures within the department to determine the causes and to implement changes to lower the incidence of the complication. Thresholds may vary from those listed here; for example, patient referral patterns and selection factors may dictate a different threshold value for a particular indicator at a particular institution. Thus, setting universal thresholds is very difficult and each department is urged to alter the thresholds to higher or lower values as needed to meet its own quality improvement program needs.

Complications can be stratified on the basis of outcome. Major complications result in admission to a hospital for therapy (for outpatient procedures), an unplanned increase in the level of care, prolonged hospitalization, permanent adverse sequelae, or death. Minor complications result in

no sequelae; they may require nominal therapy or a short hospital stay for observation (generally overnight; see Appendix B). The complication rates and suggested thresholds in **Table 8** refer to major complications.

Published rates for individual types of complications are highly dependent on patient selection and are based on series comprising several hundred patients, which is a volume larger than most individual practitioners are likely to treat. Generally, the complication-specific thresholds should be set higher than the complication-specific reported rates listed here. It is also recognized that a single complication can cause a rate to cross above a complication-specific threshold when the complication occurs within a small patient volume, (eg, early in a quality improvement program). In this situation, the overall procedure threshold is more appropriate for use in a quality improvement program. The overall procedure threshold refers to all major complications resulting from EVAR. In **Table 8**, all values were supported by the weight of literature evidence and panel consensus.

Secondary Interventions

The need for secondary procedures reached 33% within 3 years of endovascular aneurysm repair in patients entered into the European Collaborators on Stent Graft Techniques for AAA Repair registry (174,204). Persistent endoleak, device migration, and limb thrombosis were common causes for these procedures, approximately 25% of which were open surgical and 75% were endovascular procedures (205). The most common indication for secondary intervention is type II endoleak. Although the incidence of type II endoleaks varies greatly in the literature and has been reported to be as high as 30%, the clinical significance of these endoleaks is unclear. More than 50% of initially identified type II endoleaks seal spontaneously, and the subsequent clinical course of patients with sealed endoleaks does not differ from that of patients who never had one documented (206,207). Endoleak onset is highly variable and unpredictable. Conventional wisdom currently dictates that type II endoleaks, in particular, do not justify intervention unless there is also evidence of continu-

Table 8
Published Specific Major Complications for EVAR

Complication	Reported Rate (%)	Suggested Threshold (%)
Perioperative mortality	0–6	< 4
30-D mortality	0–3.1	< 2
1-Y mortality	3.4–8	< 8
Chronic kidney disease stage change	18–29	< 20
Local injury at access site	1–10	< 5
Access artery injury	0–3	< 3
Colonic ischemia	1–3	< 2
Spinal cord ischemia	0.21	< 0.21
Renal ischemia/infarction	0–5	< 2
Infection	0.5–1	< 1
Limb occlusion	0–5	< 5
Endoleak (persistent)	10–15	< 15

ing expansion of the AAA sac. The most common cause of secondary intervention was endoleak in the new generation of devices, and migration was the prime indication in half of all secondary interventions with older-generation devices (208). Type I or attachment site endoleaks at the upper end may be a result of endograft undersizing or inadequate fixation (ie, early type I), and alternatively, late dilation of the infrarenal neck may occur with similar effects (ie, late type I) (209–211). Since the inception of EVAR, there has been controversy about the management of a patent lumbar artery and IMA arising from the sac. Oversewing of these vessels is an integral part of conventional open surgery for this condition. Although type II endoleaks caused by perfusion of the sac from these vessels are seen in 20%–30% of patients, it is thought that half of early leaks seal spontaneously within several months of follow-up. However, endoleaks may persist in 10%–15% of patients, and late endoleaks may develop in another 5%–10% of patients (31).

When there is documented expansion of the residual AAA sac in the presence of a type II endoleak, intervention is mandated, as there is continued sac pressurization. Various approaches to the management of type II endoleaks have been described, including the following:

Selective transarterial catheterization and embolization of patent inflow and outflow branches.—The transarterial approach for persistent type II endoleaks involves selective embolization of patent branches that arise from, and

continue to perfuse and pressurize, the residual AAA sac. For example, an endoleak arising from a patent IMA may be approached via selective superior mesenteric artery catheterization, with subsequent negotiation of a microcatheter into the IMA through the marginal artery or the arc of Riolo. The catheter may then be advanced into the endoleak nidus within the residual sac and embolization performed, or the IMA may be occluded at the origin from the AAA sac. In addition, it has also been shown that type II endoleaks are dynamic and resemble an arteriovenous malformation with multiple feeder and draining vessels (212,213). Thus, embolization of a single feeding vessel does not provide a durable repair. A type II endoleak must be managed similar to the treatment of an arteriovenous malformation: either the nidus or all contributing branches must be occluded, as vessel recruitment is otherwise likely to occur, with resultant continued risk of aneurysm enlargement and possible rupture (214). Because of anatomic constraints, selective transarterial embolization cannot be used to occlude patent arterial branches in some cases and, in others, multiple patent branches can make this approach cumbersome and often ineffective in providing a durable repair (212,213).

Direct sac puncture and embolization via translumbar approach.—The translumbar approach is typically performed with the patient in a prone or oblique position to avoid traversing cavities or organs. In certain instances, the inferior vena cava can lie in the desired needle trajectory, but intro-

ducing the needle into the AAA sac through the inferior vena cava has been shown to be safe (215). The standard translumbar approach is typically performed under fluoroscopic guidance using bony landmarks referenced from earlier CT angiography (213). Alternately, translumbar needle placement can be initially performed with CT guidance, with the patient then transferred to the angiography suite for fluoroscopic guidance after the needle has been successfully placed in the sac. After the translumbar approach is made, the sheath needle is advanced under fluoroscopic guidance to pass just anterior to the vertebral body. The endoleak nidus may be in close proximity to the endograft, so caution must be exercised to avoid accidental puncture of the prosthesis, as this could potentially create a type III endoleak. The proper positioning of the catheter within the endoleak is signaled by free and pulsatile return of blood. When a sheath is in place, a catheter may be introduced into the sac so that it can be manipulated within the nidus as necessary for depositing the embolic agent(s). Upon injection of contrast material, the structure of the endoleak can be visualized, including the feeding vessels such as lumbar arteries or the IMA. Leaks are occluded with embolization of the endoleak sac using coils, thrombin, a mixture of coils and glue, or more recently the liquid embolic agent Onyx (ev3, Irvine, California).

Direct sac puncture and embolization via transabdominal approach.—A transabdominal approach with US guidance has been described (216,217). US guidance is used to monitor needle placement on a real-time basis. The disadvantage of a transabdominal approach is that the needle must traverse the abdominal cavity, including the bowel. This approach is additionally complicated by respiratory movements and overlying bowel gas that may obscure visualization of the AAA sac. Thrombotic occlusion of the endograft and peripheral embolism can be regarded as potential risks of this treatment.

Type I endoleaks that are recognized during the EVAR procedure typically should be corrected at that time, as the continued arterial perfusion and sac pressurization places the

patient at risk for aneurysm rupture. As previously noted, these may be at either the proximal (type IA) or the distal (type IB) attachment sites. Delayed type IA endoleaks may occur as a result of continued neck dilation at the proximal attachment site or graft migration. Endovascular techniques used to treat type IA endoleaks include placement of an aortic extension cuff or a high radial force noncovered stent. Delayed type IB endoleaks may also result from continued native arterial dilation at the distal attachment site and from cephalad graft migration that may occur if there was inadequate graft limb length or there has been excessive aneurysm remodeling following EVAR. Treatment options for type IB endoleaks depend on the etiology and location of the leak. An iliac limb extension may be appropriate if there has been cephalad graft migration, whereas a flared limb or distal cuff may be appropriate if there has been progressive dilation at the attachment site and there is a sufficient landing zone proximal to the iliac bifurcation. Limb extension into the external iliac artery may be necessary in some cases, and if so, this may require IIA embolization to prevent retrograde perfusion and persistent endoleak. If a type I endoleak cannot be successfully treated by endovascular means, open surgical conversion may be necessary.

Type III endoleaks require placement of additional graft components at the site of a modular dehiscence or a graft defect to restore the integrity of the endograft.

Treatment must be individualized for type V endoleaks, based on the suspected cause of continued sac expansion. In cases of sac seromas resulting from an ultrafiltrate, investigators have described relining of the original endograft with a new low-porosity endoprosthesis (218,219). If a source of perfusion cannot be demonstrated and corrected, this may require open surgical conversion if sac expansion continues.

Endovascular conversion is a potential secondary intervention that involves placement of a new aortouniiliac graft within the previous graft. If the original graft was bifurcated, the contralateral limb is blocked with an occluder and flow to that lower extremity is restored with a femorofemo-

ral bypass or an iliofemoral bypass graft. Endovascular conversion may be useful in cases of failed attempts to treat entities such as severe limb kinking, modular disconnection with type III endoleak, type I proximal endoleak, and device migration.

Graft limb thrombosis may occur as a result of severe limb kinking or constriction of the limb at some point along its course (such as the aortic bifurcation), or may result from stenotic or occlusive disease involving the native pelvic arteries. Symptomatic stenotic or occlusive lesions that develop within the graft or the iliofemoral arteries distal to the graft generally should be treated aggressively; hemodynamically significant stenoses or severe kinking (eg, 90°) of graft limbs detected on routine duplex or CT angiographic surveillance studies may be similarly treated to prevent potential limb thrombosis. Treatment strategies for stenoses or limb kinking include angioplasty and/or placement of noncovered or covered stents (201). Treatment of thromboses may involve embolectomy, thrombolysis, or cross-femoral bypass surgery (201,220). Conversely, asymptomatic stenoses or occlusions may be conservatively managed.

Open surgical conversion following EVAR refers to the late removal or open surgical modification of an aortic endograft. This is technically challenging, especially when performed in the acute setting. The overall delayed conversion rate for patients undergoing EVAR has been estimated to be between 0.6% and 4.5% (173,221–224). The exact approach of the open surgical conversion depends on several factors, including the type and condition of the original endograft as well as the presence of suprarenal stents and/or hooks or barbs; the presence of any additional grafts, cuffs, or coils placed as secondary interventions; the condition of proximal and distal fixation points and how intact they are; the current aneurysm morphology; the presence of periaortic scarring or inflammation; and the urgency of the repair. Although most endograft failures can be treated by endovascular means, in some cases, the development of endoleak with aneurysm enlargement, aneurysm enlargement without demonstrable

endoleak, aortoenteric fistula, graft migration, or rupture may necessitate open surgical conversion on an elective or sometimes emergent basis.

Acknowledgments: T. Gregory Walker, MD, and Sanjeeva P. Kalva, MD, authored the first draft of this document and served as topic leaders during the subsequent revisions of the draft. Sanjoy Kundu, MD, FRCPC, is chair of the SIR Standards of Practice Committee. John F. Cardella, MD, is Councilor of the SIR Standards Division. Other members of the Standards of Practice Committee and SIR who participated in the development of this clinical practice guideline are as follows, listed alphabetically:

John "Fritz" Angle, MD, Ganesh Annamalai, MD, Sriharsha Athreya, MS, Stephen Balter, PhD, Daniel B. Brown, MD, Danny Chan, MD, Christine P. Chao, MD, Timothy W.I. Clark, MD, MSc, Bairbre L. Connolly, MD, Horacio D'Agostino, MD, Sean R. Dariushnia, MD, Jon C. Davidson, MD, Brian D. Davison, MD, B. Janne d'Othee, MD, MPH, Eduardo P. Eyheremendy, MD, Suvranu Ganguli, MD, Debra Ann Gervais, MD, S. Nahum Goldberg, MD, Alan T. Hirsch, MD, Eric J. Hohenwarter, MD, Maxim Itkin, MD, Arshad Ahmed Khan, MD, Hyun S. "Kevin" Kim, MD, Venkataramu Krishnamurthy, MD, Ashish Mahajan, MD, Patrick C. Malloy, MD, Gloria M. Martinez-Salazar, MD, J. Kevin McGraw, MD, Donald L. Miller, MD, Philip M. Meyers, MD, Steven F. Millward, MD, FRCPC, Boris Nikolic, MD, MBA, Charles A. Owens, MD, Darren Postoak, MD, Dheeraj K. Rajan, MD, FRCPC, Anne C. Roberts, MD, Wael E.A. Saad, MD, Tarun Sabharwal, MD, Marc R. Sapoval, MD, PhD, Cindy Kaiser Saiter, NP, Marc S. Schwartzberg, MD, Samir S. Shah, MD, Nasir H. Siddiqi, MD, Constantinos T. Sofocleous, MD, PhD, LeAnn Stokes, MD, Rajeev Suri, MD, Timothy L. Swan, MD, Raymond H. Thornton, MD, Patricia E. Thorpe, MD, Richard Towbin, MD, Aradhana Venkatesan, MD, Michael J. Wallace, MD, Joan Wojak, MD, Ronald R. Wardrop, RN, BSN, and Darryl A. Zuckerman, MD.

Ex Officio members are as follows: Curtis W. Bakal, MD, Curtis A. Lewis, MD, MBA, JD, Kenneth S. Rholl, MD, David Sacks, MD, Dimitrios Tsetis, MD, Dimitris Karnabatidis, MD, Thomas Rand, MD, Salvatore Masala, MD, and Vlastimil Valek, MD.

APPENDIX A: CONSENSUS METHODOLOGY

Reported complication-specific rates in some cases reflect the aggregate of

major and minor complications. Thresholds are derived from critical evaluation of the literature, evaluation of empirical data from Standards of Practice Committee members' practices, and, when available, the SIR HI-IQ System national database.

APPENDIX B: SOCIETY OF INTERVENTIONAL RADIOLOGY STANDARDS OF PRACTICE COMMITTEE CLASSIFICATION OF COMPLICATIONS BY OUTCOME

Minor Complications

- A. No therapy, no consequence.
- B. Nominal therapy, no consequence; includes overnight admission (up to 23 hours) for observation only.

Major Complications

- C. Require therapy, minor hospitalization (≥ 24 h but < 48 h).
- D. Require major therapy, unplanned increase in level of care, prolonged hospitalization (> 48 hours).
- E. Permanent adverse sequelae.
- F. Death.

References

1. Fink A, Kosecoff J, Chassin M, Brook RH. Consensus methods: characteristics and guidelines for use. *Am J Public Health* 1984; 74:979–983.
2. Gillum RF. Epidemiology of aortic aneurysm in the united states. *J Clin Epidemiol* 1995; 48:1289–1298.
3. Silverberg E, Boring CC, Squires TS. Cancer statistics, 1990. *CA Cancer J Clin*. 1990; 4:9–26.
4. Matsumura JS, Brewster DC, Makaroun MS, Naftel DC. A multicenter controlled clinical trial of open versus endovascular treatment of abdominal aortic aneurysm. *J Vasc Surg* 2003; 37:262–271.
5. Geller SC; Society of Interventional Radiology Device Forum. Imaging guidelines for abdominal aortic aneurysm repair with endovascular stent grafts. *J Vasc Interv Radiol* 2003; 14 (suppl):S263–S264.
6. Prinssen M, Verhoeven EL, Buth J, et al. A randomized trial comparing conventional and endovascular repair of abdominal aortic aneurysms. *N Engl J Med* 2004; 351:1607–1618.
7. Prinssen M, Buskens E, de Jong SE, et al. Cost-effectiveness of conventional and endovascular repair of abdominal aortic aneurysms: results of a randomized trial. *J Vasc Surg* 2007; 46:883–90.
8. Blankensteijn JD, de Jong SE, Prinssen M, et al. Two-year outcomes after conventional or endovascular repair of abdominal aortic aneurysms. *N Engl J Med* 2005; 352:2398–2405.
9. Greenhalgh RM, Brown LC, Kwong GP, Powell JT, Thompson SG; EVAR trial participants. Comparison of endovascular aneurysm repair with open repair in patients with abdominal aortic aneurysm (EVAR trial 1), 30-day operative mortality results: randomised controlled trial. *Lancet* 2004; 364:843–848.
10. EVAR Trial Participants. Endovascular aneurysm repair versus open repair in patients with abdominal aortic aneurysm (EVAR trial 1): randomised controlled trial. *Lancet* 2005; 365:2179–2186.
11. Prinssen M, Wixson CL, Buskens E, Blankensteijn JD. Surveillance after endovascular aneurysm repair: diagnostics, complications, and associated costs. *Ann Vasc Surg* 2004; 18:421–427.
12. De Centa I, Coggia M, Cochennec F, Alfonsi P, Javerliat I, Goeau-Brissonniere O. Laparoscopic abdominal aortic aneurysm repair in octogenarians. *J Vasc Surg* 2009; 49:1135–1139.
13. Lederle FA, Johnson GR, Wilson SE, et al. Rupture rate of large abdominal aortic aneurysms in patients refusing or unfit for elective repair. *JAMA* 2002; 287:2968–2972.
14. Brady AR, Thompson SG, Fowkes FG, Greenhalgh RM, Powell JT; UK Small Aneurysm Trial Participants. Abdominal aortic aneurysm expansion: risk factors and time intervals for surveillance. *Circulation* 2004; 110:16–21.
15. Hirsch AT, Haskal ZJ, Hertzner NR, et al. ACC/AHA 2005 Practice Guidelines for the Management of Patients with Peripheral Arterial Disease (Lower Extremity, Renal, Mesenteric, and Abdominal Aortic): a collaborative report from the American Association for Vascular Surgery/Society for Vascular Surgery, Society for Cardiovascular Angiography and Interventions, Society for Vascular Medicine and Biology, Society of Interventional Radiology, and the ACC/AHA Task Force on Practice Guidelines (writing committee to develop guidelines for the management of patients with peripheral arterial disease): Endorsed by the American Association of Cardiovascular and Pulmonary Rehabilitation; National Heart, Lung, and Blood institute; Society for Vascular Nursing; TransAtlantic Inter-society Consensus; and Vascular Disease Foundation. *Circulation* 2006; 113:e463–e654.
16. Peterson BG, Matsumura JS, Brewster DC, Makaroun MS; Excluder Bifurcated Endoprosthesis Investigators. Five-year report of a multicenter controlled clinical trial of open versus endovascular treatment of abdominal aortic aneurysms. *J Vasc Surg* 2007; 45:885–890.
17. Cho JS, Dillavou ED, Rhee RY, Makaroun MS. Late abdominal aortic aneurysm enlargement after endovascular repair with the excluder device. *J Vasc Surg* 2004; 39:1236–1241.
18. Assar AN, Zarins CK. Ruptured abdominal aortic aneurysm: a surgical emergency with many clinical presentations. *Postgrad Med J* 2009; 85:268–273.
19. Robbins M, Kritpracha B, Beebe HG, Criado FJ, Daoud Y, Comerota AJ. Suprarenal endograft fixation avoids adverse outcomes associated with aortic neck angulation. *Ann Vasc Surg* 2005; 19:172–177.
20. Alric P, Hinchliffe RJ, Picot MC, et al. Long-term renal function following endovascular aneurysm repair with infrarenal and suprarenal aortic stent-grafts. *J Endovasc Ther* 2003; 10:397–405.
21. Lobato AC, Quick RC, Vaughn PL, Rodriguez-Lopez J, Douglas M, Dietrich EB. Transrenal fixation of aortic endografts: intermediate follow-up of a single-center experience. *J Endovasc Ther* 2000; 7:273–278.
22. Malina M, Veith F, Ivancev K, Sonesson B. Balloon occlusion of the aorta during endovascular repair of ruptured abdominal aortic aneurysm. *J Endovasc Ther* 2005; 12:556–559.
23. Marin ML, Parsons RE, Hollier LH, et al. Impact of transrenal aortic endograft placement on endovascular graft repair of abdominal aortic aneurysms. *J Vasc Surg* 1998; 28:638–646.
24. Cayne NS, Rhee SJ, Veith FJ, et al. Does transrenal fixation of aortic endografts impair renal function? *J Vasc Surg* 2003; 38:639–644.
25. O'Donnell ME, Sun Z, Winder RJ, Ellis PK, Lau LL, Blair PH. Suprarenal fixation of endovascular aortic stent grafts: assessment of medium-term to long-term renal function by analysis of juxtarenal stent morphology. *J Vasc Surg* 2007; 45:694–700.
26. Kramer SC, Seifarth H, Pamler R, et al. Renal infarction following endovascular aortic aneurysm repair: incidence and clinical consequences. *J Endovasc Ther* 2002; 9:98–102.
27. Lau LL, Hakaim AG, Oldenburg WA, et al. Effect of suprarenal versus infrarenal aortic endograft fixation on renal function and renal artery patency: a comparative study with interme-

- diate follow-up. *J Vasc Surg* 2003; 37:1162–1168.
28. Burks JA Jr, Faries PL, Gravereaux EC, Hollier LH, Marin ML. Endovascular repair of abdominal aortic aneurysms: stent-graft fixation across the visceral arteries. *J Vasc Surg* 2002; 35:109–113.
 29. Choke E, Munneke G, Morgan R, et al. Visceral and renal artery complications of suprarenal fixation during endovascular aneurysm repair. *Cardiovasc Intervent Radiol* 2007; 30:619–627.
 30. Golzarian J, Valenti D. Endoleakage after endovascular treatment of abdominal aortic aneurysms: diagnosis, significance and treatment. *Eur Radiol* 2006; 16:2849–2857.
 31. Veith FJ, Baum RA, Ohki T, et al. Nature and significance of endoleaks and endotension: summary of opinions expressed at an international conference. *J Vasc Surg* 2002; 35:1029–1035.
 32. Gilling-Smith G, Brennan J, Harris P, Bakran A, Gould D, McWilliams R. Endotension after endovascular aneurysm repair: definition, classification, and strategies for surveillance and intervention. *J Endovasc Surg* 1999; 6:305–307.
 33. van Sambeek MR, Hendriks JM, Tseng L, van Dijk LC, van Urk H. Sac enlargement without endoleak: when and how to convert and technical considerations. *Semin Vasc Surg* 2004; 17:284–287.
 34. Hertzner NR. Current status of endovascular repair of infrarenal abdominal aortic aneurysms in the context of 50 years of conventional repair. *Ann N Y Acad Sci* 2006; 1085:175–186.
 35. Hertzner NR, Mascha EJ, Karafa MT, O'Hara PJ, Krajewski LP, Beven EG. Open infrarenal abdominal aortic aneurysm repair: The Cleveland Clinic experience from 1989 to 1998. *J Vasc Surg* 2002; 35:1145–1154.
 36. Tang T, Walsh SR, Fanshawe TR, et al. Estimation of physiologic ability and surgical stress (E-PASS) as a predictor of immediate outcome after elective abdominal aortic aneurysm surgery. *Am J Surg* 2007; 194:176–182.
 37. EVAR Trial Participants. Endovascular aneurysm repair and outcome in patients unfit for open repair of abdominal aortic aneurysm (EVAR trial 2): randomised controlled trial. *Lancet* 2005; 365:2187–2192.
 38. Lederle FA, Powell JT, Greenhalgh RM. Repair of small abdominal aortic aneurysms. *N Engl J Med* 2006; 354:1537–1538.
 39. Powell JT, Brown LC, Forbes JF, et al. Final 12-year follow-up of surgery versus surveillance in the UK Small Aneurysm Trial. *Br J Surg* 2007; 94:702–708.
 40. Powell JT, Greenhalgh RM. Clinical practice. small abdominal aortic aneurysms. *N Engl J Med* 2003; 348:1895–1901.
 41. Ouriel K, Clair DG, Kent KC, Zarins CK; Positive Impact of Endovascular Options for treating Aneurysms Early (PIVOTAL) Investigators. Endovascular repair compared with surveillance for patients with small abdominal aortic aneurysms. *J Vasc Surg* 2010; 51:1081–1087.
 42. Woodburn KR, Chant H, Davies JN, Blanshard KS, Travis SJ. Suitability for endovascular aneurysm repair in an unselected population. *Br J Surg* 2001; 88:77–81.
 43. Slater BJ, Harris EJ, Lee JT. Anatomic suitability of ruptured abdominal aortic aneurysms for endovascular repair. *Ann Vasc Surg* 2008; 22:716–722.
 44. Yau FS, Rosero EB, Clagett GP, et al. Surveillance of small aortic aneurysms does not alter anatomic suitability for endovascular repair. *J Vasc Surg* 2007; 45:96–100.
 45. Elkouri S, Martelli E, Glociczki P, et al. Most patients with abdominal aortic aneurysm are not suitable for endovascular repair using currently approved bifurcated stent-grafts. *Vasc Endovascular Surg* 2004; 38:401–412.
 46. Cotroneo AR, Iezzi R, Giancristofaro D, et al. Endovascular abdominal aortic aneurysm repair: how many patients are eligible for endovascular repair? *Radiol Med* 2006; 111:597–606.
 47. Anderson PL, Arons RR, Moskowitz AJ, et al. A statewide experience with endovascular abdominal aortic aneurysm repair: rapid diffusion with excellent early results. *J Vasc Surg* 2004; 39:10–19.
 48. Schermerhorn ML, O'Malley AJ, Jhaveri A, Cotterill P, Pomposelli F, Landon BE. Endovascular vs. open repair of abdominal aortic aneurysms in the medicare population. *N Engl J Med* 2008; 358:464–474.
 49. Chaikof EL, Brewster DC, Dalman RL, 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–S49.
 50. Park B, Mavanou A, Drezner AD, Gallagher J, Menzoian JO. Clinical impact of chronic renal insufficiency on endovascular aneurysm repair. *Vasc Endovasc Surg* 2007; 40:437–445.
 51. Bown MJ, Norwood MG, Sayers RD. The management of abdominal aortic aneurysms in patients with concurrent renal impairment. *Eur J Vasc Endovasc Surg* 2005; 30:1–11.
 52. Leurs LJ, Laheij RJ, Buth J; EUROSTAR Collaborators. Influence of diabetes mellitus on the endovascular treatment of abdominal aortic aneurysms. *J Endovasc Ther* 2005; 12:288–296.
 53. Henebiens M, Vahl A, Koelemay MJ. Elective surgery of abdominal aortic aneurysms in octogenarians: a systematic review. *J Vasc Surg* 2008; 47:676–681.
 54. Timaran CH, Rosero EB, Smith ST, Modrall JG, Valentine RJ, Clagett GP. Influence of age, aneurysm size, and patient fitness on suitability for endovascular aortic aneurysm repair. *Ann Vasc Surg* 2008; 22:730–735.
 55. Bockler D, Mansmann U, Krauss M, Schunn C, Tengg-Kobligh H, Raitchel D. Midterm results after endovascular aortic aneurysm repair in the elderly. *Zentralbl Chir* 2007; 132:198–204.
 56. Winterborn RJ, Amin I, Lyratzopoulos G, Walker N, Varty K, Campbell WB. Preferences for endovascular (EVAR) or open surgical repair among patients with abdominal aortic aneurysms under surveillance. *J Vasc Surg* 2009; 49:576–581.e3.
 57. Rockman C. Reducing complications by better case selection: anatomic considerations. *Semin Vasc Surg* 2004; 17:298–306.
 58. Tefera G, Carr SC, Turnipseed WD. Endovascular aortic repair or minimal incision aortic surgery: Which procedure to choose for treatment of high-risk aneurysms? *Surgery* 2004; 136:748–753.
 59. Mureebe L, Egorova N, Giacovelli JK, Gelijns A, Kent KC, McKinsey JF. National trends in the repair of ruptured abdominal aortic aneurysms. *J Vasc Surg* 2008; 48:1101–1107.
 60. Lesperance K, Andersen C, Singh N, Starnes B, Martin MJ. Expanding use of emergency endovascular repair for ruptured abdominal aortic aneurysms: disparities in outcomes from a nationwide perspective. *J Vasc Surg* 2008; 47:1165–1170.
 61. Mehta M, Taggart J, Darling RC III, et al. Establishing a protocol for endovascular treatment of ruptured abdominal aortic aneurysms: outcomes of a prospective analysis. *J Vasc Surg* 2006; 44:1–8.
 62. Veith FJ, Ohki T, Lipsitz EC, Suggs WD, Cynamon J. Endovascular grafts and other catheter-directed techniques in the management of ruptured abdominal aortic aneurysms. *Semin Vasc Surg* 2003; 16:326–331.
 63. Veith FJ, Ohki T, Lipsitz EC, Suggs WD, Cynamon J. Treatment of rup-

- ured abdominal aneurysms with stent grafts: a new gold standard? *Semin Vasc Surg* 2003; 16:171-175.
64. Ohki T, Veith FJ. Endovascular grafts and other image-guided catheter-based adjuncts to improve the treatment of ruptured aortoiliac aneurysms. *Ann Surg* 2000; 232:466-479.
 65. Azizzadeh A, Villa MA, Miller CC III, Estrera AL, Coogan SM, Safi HJ. Endovascular repair of ruptured abdominal aortic aneurysms: Systematic literature review. *Vascular* 2008; 16: 219-224.
 66. Rayt HS, Sutton AJ, London NJ, Sayers RD, Bown MJ. A systematic review and meta-analysis of endovascular repair (EVAR) for ruptured abdominal aortic aneurysm. *Eur J Vasc Endovasc Surg* 2008; 36:536-544.
 67. Mehta M, Darling RC III, Roddy SP, et al. Factors associated with abdominal compartment syndrome complicating endovascular repair of ruptured abdominal aortic aneurysms. *J Vasc Surg* 2005; 42:1047-1051.
 68. Chaikof EL, Fillinger MF, Matsumura JS, et al. Identifying and grading factors that modify the outcome of endovascular aortic aneurysm repair. *J Vasc Surg* 2002; 35:1061-1066.
 69. Tournoij E, Slisatkov W, Prokop M, Verhagen HJ, Moll FL. Thrombus and calcium in aortic aneurysm necks: validation of a scoring system in a dutch cohort study. *Vasc Endovasc Surg* 2007; 41:120-125.
 70. McDonnell CO, Halak M, Bartlett A, Baker SR. Abdominal aortic aneurysm neck morphology: proposed classification system. *Ir J Med Sci* 2006; 175:4-8.
 71. Berman L, Curry L, Gusberg R, Dardik A, Fraenkel L. Informed consent for abdominal aortic aneurysm repair: the patient's perspective. *J Vasc Surg* 2008; 48:296-302.
 72. Weisbord SD, Palevsky PM. Prevention of contrast-induced nephropathy with volume expansion. *Clin J Am Soc Nephrol* 2008; 3:273-280.
 73. Sterling KA, Tehrani T, Rudnick MR. Clinical significance and preventive strategies for contrast-induced nephropathy. *Curr Opin Nephrol Hypertens* 2008; 17:616-623.
 74. Henretta JP, Hodgson KJ, Mattos MA, et al. Feasibility of endovascular repair of abdominal aortic aneurysms with local anesthesia with intravenous sedation. *J Vasc Surg* 1999; 29: 793-798.
 75. Bettex DA, Lachat M, Pfammatter T, Schmidlin D, Turina MI, Schmid ER. To compare general, epidural and local anaesthesia for endovascular aneurysm repair (EVAR). *Eur J Vasc Endovasc Surg* 2001; 21:179-184.
 76. De Virgilio C, Romero L, Donayre C, et al. Endovascular abdominal aortic aneurysm repair with general versus local anesthesia: a comparison of cardiopulmonary morbidity and mortality rates. *J Vasc Surg* 2002; 36:988-991.
 77. Ruppert V, Leurs LJ, Steckmeier B, Buth J, Umscheid T. Influence of anesthesia type on outcome after endovascular aortic aneurysm repair: an analysis based on EUROSTAR data. *J Vasc Surg* 2006; 44:16-21.
 78. Sadat U, Cooper DG, Gillard JH, Walsh SR, Hayes PD. Impact of the type of anesthesia on outcome after elective endovascular aortic aneurysm repair: literature review. *Vascular* 2008; 16:340-345.
 79. Haas PC, Krajcic Z, Diethrich EB. Closure of large percutaneous access sites using the prostar XL percutaneous vascular surgery device. *J Endovasc Surg* 1999; 6:168-170.
 80. Morasch MD. Percutaneous thoracic and abdominal aortic aneurysm repair. *Ann Vasc Surg* 2005; 19:585-589.
 81. Morasch MD. Percutaneous techniques for aneurysm repair. *J Vasc Surg* 2006; 43:69A-72A.
 82. Lee WA, Brown MP, Nelson PR, Huber TS, Seeger JM. Midterm outcomes of femoral arteries after percutaneous endovascular aortic repair using the Preclose technique. *J Vasc Surg* 2008; 47:919-923.
 83. Teh LG, Sieunarine K, van Schie G, et al. Use of the percutaneous vascular surgery device for closure of femoral access sites during endovascular aneurysm repair: Lessons from our experience. *Eur J Vasc Endovasc Surg* 2001; 22:418-423.
 84. Starnes BW, Andersen CA, Ronsivalle JA, Stockmaster NR, Mullenix PS, Statler JD. Totally percutaneous aortic aneurysm repair: experience and prudence. *J Vasc Surg* 2006; 43:270-276.
 85. Criado FJ. Iliac arterial conduits for endovascular access: technical considerations. *J Endovasc Ther* 2007; 14: 347-351.
 86. Peterson BG, Matsumura JS. Creative options for large sheath access during aortic endografting. *J Vasc Interv Radiol* 2008; 19(suppl):S22-S26.
 87. Peterson BG, Matsumura JS. Internal endoconduit: an innovative technique to address unfavorable iliac artery anatomy encountered during thoracic endovascular aortic repair. *J Vasc Surg* 2008; 47:441-445.
 88. von Segesser LK, Marty B, Ruchat P, Bogen M, Gallino A. Routine use of intravascular ultrasound for endovascular aneurysm repair: angiography is not necessary. *Eur J Vasc Endovasc Surg* 2002; 23:537-542.
 89. Garret HE Jr, Abdullah AH, Hodgkiss TD, Bugar SR. Intravascular ultrasound aids in the performance of endovascular repair of abdominal aortic aneurysm. *J Vasc Surg* 2003; 37:615-618.
 90. Heinrich MC, Haberle L, Muller V, Bautz W, Uder M. Nephrotoxicity of iso-osmolar iodixanol compared with nonionic low-osmolar contrast media: meta-analysis of randomized controlled trials. *Radiology* 2009; 250: 68-86.
 91. Su WT, Stone DH, Lamparello PJ, Rockman CB. Gluteal compartment syndrome following elective unilateral internal iliac artery embolization before endovascular abdominal aortic aneurysm repair. *J Vasc Surg* 2004; 39:672-675.
 92. Rayt HS, Bown MJ, Lambert KV, et al. Buttock claudication and erectile dysfunction after internal iliac artery embolization in patients prior to endovascular aortic aneurysm repair. *Cardiovasc Intervent Radiol* 2008; 31: 728-734.
 93. Engelke C, Elford J, Morgan RA, Belli AM. Internal iliac artery embolization with bilateral occlusion before endovascular aortoiliac aneurysm repair-clinical outcome of simultaneous and sequential intervention. *J Vasc Interv Radiol* 2002; 13:667-676.
 94. Arko FR, Lee WA, Hill BB, Fogarty TJ, Zarins CK. Hypogastric artery bypass to preserve pelvic circulation: improved outcome after endovascular abdominal aortic aneurysm repair. *J Vasc Surg* 2004; 39:404-408.
 95. Kritpracha B, Pigott JP, Price CI, Russell TE, Corbey MJ, Beebe HG. Distal internal iliac artery embolization: a procedure to avoid. *J Vasc Surg* 2003; 37:943-948.
 96. Vandy F, Criado E, Upchurch GR Jr, Williams DM, Rectenwald J, Eliason J. Transluminal hypogastric artery occlusion with an Amplatzer vascular plug during endovascular aortic aneurysm repair. *J Vasc Surg* 2008; 48: 1121-1124.
 97. Bonvini R, Alerci M, Antonucci F, et al. Preoperative embolization of collateral side branches: A valid means to reduce type II endoleaks after endovascular AAA repair. *J Endovasc Ther* 2003; 10:227-232.
 98. Gould DA, McWilliams R, Edwards RD, et al. Aortic side branch embolization before endovascular aneurysm repair: Incidence of type II endoleak. *J Vasc Interv Radiol* 2001; 12: 337-341.
 99. Back MR, Bowser AN, Johnson BL, Schmach D, Zwiebel B, Bandyk DF.

- Patency of infrarenal aortic side branches determines early aneurysm sac behavior after endovascular repair. *Ann Vasc Surg* 2003; 17:27–34.
100. Velazquez OC, Baum RA, Carpenter JP, et al. Relationship between preoperative patency of the inferior mesenteric artery and subsequent occurrence of type II endoleak in patients undergoing endovascular repair of abdominal aortic aneurysms. *J Vasc Surg* 2000; 32:777–788.
 101. Axelrod DJ, Lookstein RA, Guller J, et al. Inferior mesenteric artery embolization before endovascular aneurysm repair: technique and initial results. *J Vasc Interv Radiol* 2004; 15:1263–1267.
 102. Brewster DC, Retana A, Waltman AC, Darling RC. Angiography in the management of aneurysms of the abdominal aorta. its value and safety. *N Engl J Med* 1975; 292:822–825.
 103. Olin JW, Melia M, Young JR, Graor RA, Risius B. Prevalence of atherosclerotic renal artery stenosis in patients with atherosclerosis elsewhere. *Am J Med* 1990; 88:46N–51N.
 104. Tollefson DF, Ernst CB. Natural history of atherosclerotic renal artery stenosis associated with aortic disease. *J Vasc Surg* 1991; 14:327–331.
 105. Springer F, Gunther RW, Schmitz-Rode T. Aneurysm sac pressure measurement with minimally invasive implantable pressure sensors: an alternative to current surveillance regimes after EVAR? *Cardiovasc Intervent Radiol* 2008; 31:460–467.
 106. Ohki T, Ouriel K, Silveira PG, et al. Initial results of wireless pressure sensing for endovascular aneurysm repair: the APEX trial—Acute Pressure measurement to confirm aneurysm sac EXclusion. *J Vasc Surg* 2007; 45:236–242.
 107. Zarins CK, White RA, Hodgson KJ, Schwarten D, Fogarty TJ. Endoleak as a predictor of outcome after endovascular aneurysm repair: AneuRx multicenter clinical trial. *J Vasc Surg* 2000; 32:90–107.
 108. Gelfand DV, White GH, Wilson SE. Clinical significance of type II endoleak after endovascular repair of abdominal aortic aneurysm. *Ann Vasc Surg* 2006; 20:69–74.
 109. Thompson JF, Mullee MA, Bell PR, et al. Intraoperative heparinisation, blood loss and myocardial infarction during aortic aneurysm surgery: a joint vascular research group study. *Eur J Vasc Endovasc Surg* 1996; 12:86–90.
 110. Tremey B, Szekely B, Schlumberger S, et al. Anticoagulation monitoring during vascular surgery: accuracy of the hemochron low range activated clotting time (ACT-LR). *Br J Anaesth* 2006; 97:453–459.
 111. Robinson MH, Studley JG, Powis SJ. Anticoagulation in abdominal aortic aneurysm surgery: the approach of vascular surgeons in Great Britain and Ireland. *Eur J Vasc Surg* 1989; 3:141–143.
 112. Clagett GP, Sobel M, Jackson MR, Lip GY, Tangelder M, Verhaeghe R. Antithrombotic therapy in peripheral arterial occlusive disease: the Seventh ACCP Conference on Antithrombotic and Thrombolytic Therapy. *Chest* 2004; 126(suppl):609S–265.
 113. Despotis GJ, Joist JH, Hogue CW Jr, et al. The impact of heparin concentration and activated clotting time monitoring on blood conservation. A prospective, randomized evaluation in patients undergoing cardiac operation. *J Thorac Cardiovasc Surg* 1995; 110:46–54.
 114. de Swart CA, Nijmeyer B, Roelofs JM, Sixma JJ. Kinetics of intravenously administered heparin in normal humans. *Blood* 1982; 60:1251–1258.
 115. McAvoy TJ. Pharmacokinetic modeling of heparin and its clinical implications. *J Pharmacokinet Biopharm* 1979; 7:331–354.
 116. Saratzis A, Saratzis N, Melas N, Kiskinis D. Pharmacotherapy before and after endovascular repair of abdominal aortic aneurysms. *Curr Vasc Pharmacol* 2008; 6:240–249.
 117. Leurs LJ, Visser P, Laheij RJ, Buth J, Harris PL, Blankensteijn JD. Statin use is associated with reduced all-cause mortality after endovascular abdominal aortic aneurysm repair. *Vascular* 2006; 14:1–8.
 118. Dagher NN, Modrall JG. Pharmacotherapy before and after revascularization: anticoagulation, antiplatelet agents, and statins. *Semin Vasc Surg* 2007; 20:10–14.
 119. Stavropoulos SW, Charagundla SR. Imaging techniques for detection and management of endoleaks after endovascular aortic aneurysm repair. *Radiology* 2007; 243:641–655.
 120. Chabbert V, Otal P, Bouchard L, et al. Midterm outcomes of thoracic aortic stent-grafts: complications and imaging techniques. *J Endovasc Ther* 2003; 10:494–504.
 121. Fearn S, Lawrence-Brown MM, Semmens JB, Hartley D. Follow-up after endovascular aortic aneurysm repair: the plain radiograph has an essential role in surveillance. *J Endovasc Ther* 2003; 10:894–901.
 122. Parmer SS, Carpenter JP, Stavropoulos SW, et al. Endoleaks after endovascular repair of thoracic aortic aneurysms. *J Vasc Surg* 2006; 44:447–452.
 123. Rozenblit A, Marin ML, Veith FJ, Cynamon J, Wahl SI, Bakal CW. Endovascular repair of abdominal aortic aneurysm: value of postoperative follow-up with helical CT. *AJR Am J Roentgenol* 1995; 165:1473–1479.
 124. Armerding MD, Rubin GD, Beaulieu CF, et al. Aortic aneurysmal disease: assessment of stent-graft treatment-CT versus conventional angiography. *Radiology*. 2000; 215:138–146.
 125. Rozenblit AM, Patlas M, Rosenbaum AT, et al. Detection of endoleaks after endovascular repair of abdominal aortic aneurysm: value of unenhanced and delayed helical CT acquisitions. *Radiology* 2003; 227:426–433.
 126. Golzarian J, Dussaussois L, Abada HT, et al. Helical CT of aorta after endoluminal stent-graft therapy: value of biphasic acquisition. *AJR Am J Roentgenol* 1998; 171:329–331.
 127. Jezzi R, Cotroneo AR, Filippone A, et al. Multidetector CT in abdominal aortic aneurysm treated with endovascular repair: are unenhanced and delayed phase enhanced images effective for endoleak detection? *Radiology* 2006; 241:915–921.
 128. Macari M, Chandarana H, Schmidt B, Lee J, Lamparello P, Babb J. Abdominal aortic aneurysm: can the arterial phase at CT evaluation after endovascular repair be eliminated to reduce radiation dose? *Radiology* 2006; 241:908–914.
 129. Bley TA, Chase PJ, Reeder SB, et al. Endovascular abdominal aortic aneurysm repair: nonenhanced volumetric CT for follow-up. *Radiology* 2009; 253:253–262.
 130. van der Laan MJ, Bartels LW, Bakker CJ, Viergever MA, Blankensteijn JD. Suitability of 7 aortic stent-graft models for MRI-based surveillance. *J Endovasc Ther* 2004; 11:366–371.
 131. Cejna M, Loewe C, Schoder M, et al. MR angiography vs CT angiography in the follow-up of nitinol stent grafts in endoluminally treated aortic aneurysms. *Eur Radiol* 2002; 12:2443–2450.
 132. Insko EK, Kulzer LM, Fairman RM, Carpenter JP, Stavropoulos SW. MR imaging for the detection of endoleaks in recipients of abdominal aortic stent-grafts with low magnetic susceptibility. *Acad Radiol* 2003; 10:509–513.
 133. Ayuso JR, de Caralt TM, Pages M, et al. MRA is useful as a follow-up technique after endovascular repair of aortic aneurysms with nitinol endoprostheses. *J Magn Reson Imaging* 2004; 20:803–810.
 134. Pitton MB, Schweitzer H, Herber S, et al. MRI versus helical CT for endoleak detection after endovascular

- aneurysm repair. *AJR Am J Roentgenol* 2005; 185:1275–1281.
135. van der Laan MJ, Bartels LW, Viergever MA, Blankensteijn JD. Computed tomography versus magnetic resonance imaging of endoleaks after EVAR. *Eur J Vasc Endovasc Surg* 2006; 32:361–365.
 136. Wicky S, Fan CM, Geller SC, Greenfield A, Santilli J, Waltman AC. MR angiography of endoleak with inconclusive concomitant CT angiography. *AJR Am J Roentgenol* 2003; 181:736–738.
 137. Weigel S, Tombach B, Maintz D, et al. Thoracic aortic stent graft: comparison of contrast-enhanced MR angiography and CT angiography in the follow-up. Initial results. *Eur Radiol* 2003; 13:1628–1634.
 138. Lookstein RA, Goldman J, Pukin L, Marin ML. Time-resolved magnetic resonance angiography as a noninvasive method to characterize endoleaks: initial results compared with conventional angiography. *J Vasc Surg* 2004; 39:27–33.
 139. Cohen EI, Weinreb DB, Siegelbaum RH, et al. Time-resolved MR angiography for the classification of endoleaks after endovascular aneurysm repair. *J Magn Reson Imaging*. 2008; 27:500–503.
 140. Ersoy H, Jacobs P, Kent CK, Prince MR. Blood pool MR angiography of aortic stent-graft endoleak. *AJR Am J Roentgenol* 2004; 182:1181–1186.
 141. Sato DT, Goff CD, Gregory RT, et al. Endoleak after aortic stent graft repair: Diagnosis by color duplex ultrasound scan versus computed tomography scan. *J Vasc Surg* 1998; 28:657–663.
 142. Wolf YG, Johnson BL, Hill BB, Rubin GD, Fogarty TJ, Zarins CK. Duplex ultrasound scanning versus computed tomographic angiography for postoperative evaluation of endovascular abdominal aortic aneurysm repair. *J Vasc Surg* 2000; 32:1142–1148.
 143. d'Audiffret A, Desgranges P, Kobeiter DH, Becquemin JP. Follow-up evaluation of endoluminally treated abdominal aortic aneurysms with duplex ultrasonography: validation with computed tomography. *J Vasc Surg* 2001; 33:42–50.
 144. Raman KG, Missig-Carroll N, Richardson T, Muluk SC, Makaroun MS. Color-flow duplex ultrasound scan versus computed tomographic scan in the surveillance of endovascular aneurysm repair. *J Vasc Surg* 2003; 38:645–651.
 145. Sprouse LR II, Meier GH III, Parent FN, DeMasi RJ, Glickman MH, Barber GA. Is ultrasound more accurate than axial computed tomography for determination of maximal abdominal aortic aneurysm diameter? *Eur J Vasc Endovasc Surg* 2004; 28:28–35.
 146. Elkouri S, Panneton JM, Andrews JC, et al. Computed tomography and ultrasound in follow-up of patients after endovascular repair of abdominal aortic aneurysm. *Ann Vasc Surg* 2004; 18:271–279.
 147. McLafferty RB, McCrary BS, Mattos MA, et al. The use of color-flow duplex scan for the detection of endoleaks. *J Vasc Surg* 2002; 36:100–104.
 148. Golzarian J, Murgio S, Dussaussois L, et al. Evaluation of abdominal aortic aneurysm after endoluminal treatment: comparison of color doppler sonography with biphasic helical CT. *AJR Am J Roentgenol* 2002; 178:623–628.
 149. AbuRahma AF, Welch CA, Mullins BB, Dyer B. Computed tomography versus color duplex ultrasound for surveillance of abdominal aortic stent-grafts. *J Endovasc Ther* 2005; 12:568–573.
 150. Ashoke R, Brown LC, Rodway A, et al. Color duplex ultrasonography is insensitive for the detection of endoleak after aortic endografting: a systematic review. *J Endovasc Ther* 2005; 12:297–305.
 151. Heilberger P, Schunn C, Ritter W, Weber S, Raithel D. Postoperative color flow duplex scanning in aortic endografting. *J Endovasc Surg* 1997; 4:262–271.
 152. McWilliams RG, Martin J, White D, et al. Use of contrast-enhanced ultrasound in follow-up after endovascular aortic aneurysm repair. *J Vasc Interv Radiol* 1999; 10:1107–1114.
 153. Bendick PJ, Bove PG, Long GW, Zeleznock GB, Brown OW, Shanley CJ. Efficacy of ultrasound scan contrast agents in the noninvasive follow-up of aortic stent grafts. *J Vasc Surg* 2003; 37:381–385.
 154. Napoli V, Bargellini I, Sardella SG, et al. Abdominal aortic aneurysm: contrast-enhanced US for missed endoleaks after endoluminal repair. *Radiology* 2004; 233:217–225.
 155. Sun Z. Diagnostic value of color duplex ultrasonography in the follow-up of endovascular repair of abdominal aortic aneurysm. *J Vasc Interv Radiol* 2006; 17:759–764.
 156. Ten Bosch JA, Rouwet EV, Peters CT, et al. Contrast-enhanced ultrasound versus computed tomographic angiography for surveillance of endovascular abdominal aortic aneurysm repair. *J Vasc Interv Radiol* 2010; 21:638–643.
 157. Sarlon G, Lapierre F, Sarlon E, Bartoli MA, Magnan PE, Branchereau A. Endovascular aneurysm repair follow-up by unenhanced and contrast-enhanced duplex ultrasound. *J Mal Vasc* 2009; 34:34–43.
 158. Chaer RA, Gushchin A, Rhee R, et al. Duplex ultrasound as the sole long-term surveillance method post-endovascular aneurysm repair: a safe alternative for stable aneurysms. *J Vasc Surg* 2009; 49:845–849.
 159. Collins JT, Boros MJ, Combs K. Ultrasound surveillance of endovascular aneurysm repair: a safe modality versus computed tomography. *Ann Vasc Surg* 2007; 21:671–675.
 160. Sandford RM, Bown MJ, Fishwick G, et al. Duplex ultrasound scanning is reliable in the detection of endoleak following endovascular aneurysm repair. *Eur J Vasc Endovasc Surg* 2006; 32:537–541.
 161. Manning BJ, O'Neill SM, Haider SN, Colgan MP, Madhavan P, Moore DJ. Duplex ultrasound in aneurysm surveillance following endovascular aneurysm repair: a comparison with computed tomography aortography. *J Vasc Surg* 2009; 49:60–65.
 162. Bargellini I, Cioni R, Napoli V, et al. Ultrasonographic surveillance with selective CTA after endovascular repair of abdominal aortic aneurysm. *J Endovasc Ther* 2009; 16:93–104.
 163. Sternbergh WC III, Greenberg RK, Chuter TA, Tonnessen BH; Zenith Investigators. Redefining postoperative surveillance after endovascular aneurysm repair: recommendations based on 5-year follow-up in the US zenith multicenter trial. *J Vasc Surg* 2008; 48:278–284.
 164. Tomlinson J, McNamara J, Matloubieh J, et al. Intermediate follow-up after endovascular aneurysm repair: can we forgo CT scanning in certain patients? *Ann Vasc Surg* 2007; 21:663–670.
 165. Corriere MA, Feurer ID, Becker SY, et al. Endoleak following endovascular abdominal aortic aneurysm repair: implications for duration of screening. *Ann Surg* 2004; 239:800–805.
 166. Hirsch AT, Haskal ZJ, Hertzler NR, et al. ACC/AHA Guidelines for the Management of Patients with Peripheral Arterial Disease (Lower Extremity, Renal, Mesenteric, and Abdominal Aortic): a collaborative report from the American Associations for Vascular Surgery/Society for Vascular Surgery, Society for Cardiovascular Angiography and Interventions, Society for Vascular Medicine and Biology, Society of Interventional Radiology, and the ACC/AHA Task Force on Practice Guidelines (writing committee to develop guidelines for the management of patients with peripheral arterial dis-

- ease)—summary of recommendations. *J Vasc Interv Radiol* 2006; 17:1383–1397.
167. Alsac JM, Zarins CK, Heikkinen MA, et al. The impact of aortic endografts on renal function. *J Vasc Surg* 2005; 41:926–930.
 168. Waasdorp E, van't Hullenaar C, van Herwaarden J, et al. Renal function after endovascular aortic aneurysm repair: a single-center experience with transrenal versus infrarenal fixation. *J Endovasc Ther* 2007; 14:130–137.
 169. Brenner DJ, Hall EJ. Computed tomography—an increasing source of radiation exposure. *N Engl J Med* 2007; 357:2277–2284.
 170. Brenner DJ, Doll R, Goodhead DT, et al. Cancer risks attributable to low doses of ionizing radiation: assessing what we really know. *Proc Natl Acad Sci U S A* 2003; 100:13761–13766.
 171. Noll RE Jr, Tonnessen BH, Mannava K, Money SR, Sternbergh WC III. Long-term postplacement cost after endovascular aneurysm repair. *J Vasc Surg* 2007; 46:9–15.
 172. Brewster DC, Jones JE, Chung TK, et al. Long-term outcomes after endovascular abdominal aortic aneurysm repair: the first decade. *Ann Surg* 2006; 244:426–438.
 173. Greenberg RK, Chuter TA, Sternbergh WC III, Fearnot NE; Zenith Investigators. Zenith AAA endovascular graft: intermediate-term results of the US multicenter trial. *J Vasc Surg* 2004; 39:1209–1218.
 174. Waasdorp EJ, de Vries JP, Hobo R, et al. Aneurysm diameter and proximal aortic neck diameter influence clinical outcome of endovascular abdominal aortic repair: a 4-year EUROSTAR experience. *Ann Vasc Surg* 2005; 19:755–761.
 175. Lange C, Leurs LJ, Buth J, Myhre HO; EUROSTAR Collaborators. Endovascular repair of abdominal aortic aneurysm in octogenarians: an analysis based on EUROSTAR data. *J Vasc Surg* 2005; 42:624–630.
 176. Peppelenbosch N, Buth J, Harris PL, van Marrewijk C, Fransen G; EUROSTAR Collaborators. Diameter of abdominal aortic aneurysm and outcome of endovascular aneurysm repair: does size matter? A report from EUROSTAR. *J Vasc Surg* 2004; 39:288–297.
 177. Drury D, Michaels JA, Jones L, Ayiku L. Systematic review of recent evidence for the safety and efficacy of elective endovascular repair in the management of infrarenal abdominal aortic aneurysm. *Br J Surg* 2005; 92: 937–946.
 178. Katzen BT, MacLean AA. Complications of endovascular repair of abdominal aortic aneurysms: a review. *Cardiovasc Intervent Radiol* 2006; 29:935–946.
 179. Liaw JV, Clark M, Gibbs R, Jenkins M, Cheshire N, Hamady M. Update: complications and management of infrarenal EVAR. *Eur J Radiol* 2008; 71: 541–551.
 180. Sharif MA, Lee B, Lau LL, et al. Prosthetic stent graft infection after endovascular abdominal aortic aneurysm repair. *J Vasc Surg* 2007; 46:442–448.
 181. Tannouri E, Golzarian J, Struyven J. Aortic prosthetic graft infection. *JBR-BTR*. 2002; 85:158–159.
 182. Vogel TR, Symons R, Flum DR. The incidence and factors associated with graft infection after aortic aneurysm repair. *J Vasc Surg* 2008; 47:264–269.
 183. van den Berg HR, Leijdekkers VJ, Vahl A. Aortic stent-graft infection following septic complications of a kidney stone. *Cardiovasc Intervent Radiol* 2006; 29:443–445.
 184. Bergqvist D, Bjorck M, Nyman R. Secondary aortoenteric fistula after endovascular aortic interventions: a systematic literature review. *J Vasc Interv Radiol* 2008; 19:163–165.
 185. Parra JR, Lee C, Hodgson KJ, Perler B. Endograft infection leading to rupture of aortic aneurysm. *J Vasc Surg* 2004; 39:676–678.
 186. Baker M, Uflacker R, Robison JG. Stent graft infection after abdominal aortic aneurysm repair: a case report. *J Vasc Surg* 2002; 36:180–183.
 187. Ducasse E, Calisti A, Speciale F, Rizzo L, Misuraca M, Fiorani P. Aortoiliac stent graft infection: current problems and management. *Ann Vasc Surg* 2004; 18:521–526.
 188. Fiorani P, Speciale F, Calisti A, et al. Endovascular graft infection: preliminary results of an international enquiry. *J Endovasc Ther* 2003; 10:919–927.
 189. Becquemin JP, Kelley L, Zubilewicz T, Desgranges P, Lapeyre M, Kobeiter H. Outcomes of secondary interventions after abdominal aortic aneurysm endovascular repair. *J Vasc Surg* 2004; 39:298–305.
 190. Perry RJ, Martin MJ, Eckert MJ, Sohn VY, Steele SR. Colonic ischemia complicating open vs endovascular abdominal aortic aneurysm repair. *J Vasc Surg* 2008; 48:272–277.
 191. Zhang WW, Kulaylat MN, Anain PM, et al. Embolization as cause of bowel ischemia after endovascular abdominal aortic aneurysm repair. *J Vasc Surg* 2004; 40:867–872.
 192. Berg P, Kaufmann D, van Marrewijk CJ, Buth J. Spinal cord ischaemia after stent-graft treatment for infra-renal abdominal aortic aneurysms. analysis of the EUROSTAR database. *Eur J Vasc Endovasc Surg* 2001; 22:342–347.
 193. Garcia ND, Tehrani H, Morasch M, Pearce W, Matsumura J. Spinal cord ischemia following endovascular repair of an infrarenal aortic aneurysm. *Ann Vasc Surg* 2002; 16:509–512.
 194. Fortes DL, Atkins BZ, Chiou AC. Delayed paraplegia following infrarenal abdominal aortic endograft placement: case report and literature review. *Vascular* 2004; 12:130–135.
 195. Rockman CB, Riles TS, Landis R. Lower extremity paraparesis or paraplegia subsequent to endovascular management of abdominal aortic aneurysms. *J Vasc Surg* 2001; 33:178–180.
 196. Reid JA, Mole DJ, Johnston LC, Lee B. Delayed paraplegia after endovascular repair of abdominal aortic aneurysm. *J Vasc Surg* 2003; 37:1322–1323.
 197. Bajwa A, Davis M, Moawad M, Taylor PR. Paraplegia following elective endovascular repair of abdominal aortic aneurysm: reversal with cerebrospinal fluid drainage. *Eur J Vasc Endovasc Surg* 2008; 35:46–48.
 198. Kalliafas S, Albertini JN, Macierewicz J, et al. Incidence and treatment of intraoperative technical problems during endovascular repair of complex abdominal aortic aneurysms. *J Vasc Surg* 2000; 31:1185–1192.
 199. van Dijk LC, van Sambeek MR, Cademartiri F, Pattynama PM. Partial blockage of the renal artery ostium after stent-graft placement: detection and treatment. *J Endovasc Ther* 2003; 10:684.
 200. Gorich J, Kramer S, Rilinger N, et al. Malpositioned or dislocated aortic endoprostheses: repositioning using percutaneous pull-down maneuvers. *J Endovasc Ther* 2000; 7:123–131.
 201. Maleux G, Koolen M, Heye S, Nevelsteen A. Limb occlusion after endovascular repair of abdominal aortic aneurysms with supported endografts. *J Vasc Interv Radiol* 2008; 19:1409–1412.
 202. Carroccio A, Faries PL, Morrissey NJ, et al. Predicting iliac limb occlusions after bifurcated aortic stent grafting: anatomic and device-related causes. *J Vasc Surg* 2002; 36:679–684.
 203. Maldonado TS, Rockman CB, Riles E, et al. Ischemic complications after endovascular abdominal aortic aneurysm repair. *J Vasc Surg* 2004; 40:703–709.
 204. White GH, May J, Waugh RC, Yu W. Type I and type II endoleaks: a more useful classification for reporting results of endoluminal AAA repair. *J Endovasc Surg* 1998; 5:189–191.
 205. Carpenter JP, Baum RA, Barker CF, et al. Durability of benefits of endovas-

- cular versus conventional abdominal aortic aneurysm repair. *J Vasc Surg* 2002; 35:222–228.
206. Resch T, Ivancev K, Lindh M, et al. Persistent collateral perfusion of abdominal aortic aneurysm after endovascular repair does not lead to progressive change in aneurysm diameter. *J Vasc Surg* 1998; 28:242–249.
 207. Makaroun M, Zajko A, Sugimoto H, Eskandari M, Webster M. Fate of endoleaks after endoluminal repair of abdominal aortic aneurysms with the EVT device. *Eur J Vasc Endovasc Surg* 1999; 18:185–190.
 208. Torella F. Effect of improved endograft design on outcome of endovascular aneurysm repair. *J Vasc Surg* 2004; 40:216–221.
 209. Zarins CK, Bloch DA, Crabtree T, Matsumoto AH, White RA, Fogarty TJ. Aneurysm enlargement following endovascular aneurysm repair: AneuRx clinical trial. *J Vasc Surg* 2004; 39:109–117.
 210. Politz JK, Newman VS, Stewart MT. Late abdominal aortic aneurysm rupture after AneuRx repair: A report of three cases. *J Vasc Surg* 2000; 31:599–606.
 211. Sahgal A, Veith FJ, Lipsitz E, et al. Diameter changes in isolated iliac artery aneurysms 1 to 6 years after endovascular graft repair. *J Vasc Surg* 2001; 33:289–294.
 212. Baum RA, Carpenter JP, Golden MA, et al. Treatment of type 2 endoleaks after endovascular repair of abdominal aortic aneurysms: comparison of transarterial and translumbar techniques. *J Vasc Surg* 2002; 35:23–29.
 213. Baum RA, Cope C, Fairman RM, Carpenter JP. Translumbar embolization of type 2 endoleaks after endovascular repair of abdominal aortic aneurysms. *J Vasc Interv Radiol* 2001; 12:111–116.
 214. Liewald F, Ermis C, Gorich J, Halter G, Scharrer-Pamler R, Sunder-Plassmann L. Influence of treatment of type II leaks on the aneurysm surface area. *Eur J Vasc Endovasc Surg* 2001; 21:339–343.
 215. Stavropoulos SW, Carpenter JP, Fairman RM, Golden MA, Baum RA. Inferior vena cava traversal for translumbar endoleak embolization after endovascular abdominal aortic aneurysm repair. *J Vasc Interv Radiol* 2003; 14:1191–1194.
 216. Kasthuri RS, Stivaros SM, Gavan D. Percutaneous ultrasound-guided thrombin injection for endoleaks: An alternative. *Cardiovasc Intervent Radiol* 2005; 28:110–112.
 217. Boks SS, Andhyiswara T, de Smet AA, Vroegindewij D. Ultrasound-guided percutaneous transabdominal treatment of a type 2 endoleak. *Cardiovasc Intervent Radiol* 2005; 28:526–529.
 218. Goodney PP, Fillinger MF. The effect of endograft relining on sac expansion after endovascular aneurysm repair with the original-permeability gore excluder abdominal aortic aneurysm endoprosthesis. *J Vasc Surg* 2007; 45:686–693.
 219. Fisher RK, Brennan JA, Gilling-Smith GL, Harris PL. Continued sac expansion in the absence of a demonstrable endoleak is an indication for secondary intervention. *Eur J Vasc Endovasc Surg* 2000; 20:96–98.
 220. Krajcer Z, Gilbert JH, Dougherty K, Mortazavi A, Strickman N. Successful treatment of aortic endograft thrombosis with rheolytic thrombectomy. *J Endovasc Ther* 2002; 9:756–764.
 221. Bockler D, Probst T, Weber H, Raithel D. Surgical conversion after endovascular grafting for abdominal aortic aneurysms. *J Endovasc Ther* 2002; 9:111–118.
 222. Greenberg RK, Lawrence-Brown M, Bhandari G, et al. An update of the zenith endovascular graft for abdominal aortic aneurysms: Initial implantation and mid-term follow-up data. *J Vasc Surg* 2001; 33 (suppl):S157–S164.
 223. Lyden SP, McNamara JM, Sternbach Y, Illig KA, Waldman DL, Green RM. Technical considerations for late removal of aortic endografts. *J Vasc Surg* 2002; 36:674–678.
 224. Conner MS III, Sternbergh WC III, Carter G, Tonnessen BH, Yoselevitz M, Money SR. Secondary procedures after endovascular aortic aneurysm repair. *J Vasc Surg* 2002; 36:992–996.

SIR DISCLAIMER

The clinical practice guidelines of the Society of Interventional Radiology attempt to define practice principles that generally should assist in producing high quality medical care. These guidelines are voluntary and are not rules. A physician may deviate from these guidelines, as necessitated by the individual patient and available resources. These practice guidelines should not be deemed inclusive of all proper methods of care or exclusive of other methods of care that are reasonably directed towards the same result. Other sources of information may be used in conjunction with these principles to produce a process leading to high quality medical care. The ultimate judgment regarding the conduct of any specific procedure or course of management must be made by the physician, who should consider all circumstances relevant to the individual clinical situation. Adherence to the SIR Quality Improvement Program will not assure a successful outcome in every situation. It is prudent to document the rationale for any deviation from the suggested practice guidelines in the department policies and procedure manual or in the patient's medical record.