

REPORTING STANDARDS

Reporting standards for endovascular aortic aneurysm repair

Elliot L. Chaikof, MD, PhD, Jan D. Blankensteijn, MD, Peter L. Harris, MD, Geoffrey H. White, MD, Christopher K. Zarins, MD, Victor M. Bernhard, MD, Jon S. Matsumura, MD, James May, MD, Frank J. Veith, MD, Mark F. Fillinger, MD, Robert B. Rutherford, MD, and K. Craig Kent, MD, for the Ad Hoc Committee for Standardized Reporting Practices in Vascular Surgery of The Society for Vascular Surgery/American Association for Vascular Surgery

In 1997, an initial set of reporting standards for studies directed at the endovascular repair of the infrarenal aortic aneurysm was introduced by the Ad Hoc Committee for Standardized Reporting Practices in Vascular Surgery of the Society for Vascular Surgery/International Society for Cardiovascular Surgery.¹ Since this initial report, the rapid evolution of the field has led to new concepts and insights into factors that define success and clinicopathologic features that have a measurable impact on outcome. Thus, a revised set of recommended standards is presented to accommodate these and other developments in this emerging discipline.

CLASSIFICATION CRITERIA FOR ARTERIAL ANEURYSMS

Recommendations regarding the definition and classification of arterial aneurysm have been previously published.² As noted in this initial document, precise nomenclature is desirable, but a risk of overclassification exists that may result in small patient subgroups that preclude meaningful data analysis. Moreover, no classification of aneurysms on the basis of a single factor has proved to be entirely satisfactory. Therefore, classification of aneurysm is recommended with respect to a combination of factors, including: (1) site; (2) etiology; and (3) clinicopathologic mani-

festations. In any one specific report, selection of only one of these factors as the basis for classification may be appropriate.

Anatomic classification. All reports should classify aortic aneurysms on the basis of site and extent of disease (Table I). Respective definitions and recommended classification schemes for nonaortic arterial aneurysms have been described in detail elsewhere.²

Etiologic classification. It is recommended that reports identify arterial aneurysm etiology. Specifically, distinction should be made between degenerative (arteriosclerotic), anastomotic, infectious, inflammatory (noninfectious), traumatic, and congenital aneurysms. Although dissection may be associated with any of a number of underlying causes, such as Marfan's syndrome or hypertension, it represents a distinct pathologic entity that should be noted accordingly in clinical reports.

Clinical classification. Aneurysms should be categorized by clinical presentation as asymptomatic or symptomatic. Specific symptoms and their time course should be documented, including those related to compression or erosion into neighboring structures, thrombosis, or embolization. Rupture should distinguish between a free rupture, fistulization into an adjacent organ, and a contained rupture. In this regard, central hemodynamic status should be reported, including blood pressure and response to initial resuscitation (stable, unstable, cardiac arrest).

OUTCOME CRITERIA AND DEFINITIONS

The motivation for aneurysm treatment is elimination of the risk of rupture and death. Therefore, by definition, the *primary outcome criteria* for endovascular aneurysm repair include the prevention of: (1) aneurysm rupture; (2) death from aneurysm rupture; and (3) aneurysm-related death that may result from primary or secondary treatment. Nonetheless, the mere presence of a device does not necessarily prohibit aneurysm rupture and death. Thus, surrogate markers that suggest a continuing or increasing risk of rupture, such as aneurysm enlargement or endoleak, although designated as *secondary outcome criteria*, play a

Competition of interest: ELC has been paid a consulting fee by and received clinical research funding from Guidant. MFF has received a speaking fee from Medtronic. JSM has been paid a consulting fee by and has received clinical research funding from Guidant, Medtronic, and WL Gore. He has also received research support from Boston Scientific. VMB is a consultant to and owns stock in Guidant. JM has been paid a consulting fee by Medtronic. CZ has been paid a consulting fee by and owns shares in Medtronic.

Additional material for this article may be found online at www.mosby.com/jvs.

Reprint requests: Elliot L. Chaikof, MD, PhD, 1639 Pierce Dr, Rm 5105, Emory University, Atlanta, GA 30322 (e-mail: echaiko@emory.edu).

J Vasc Surg 2002;35:1048-60.

Copyright © 2002 by The Society for Vascular Surgery and The American Association for Vascular Surgery.

0741-5214/2002/\$35.00 + 0 24/1/123763

doi:10.1067/mva.2002.123763

Table I. Classification of aortic and iliac aneurysms by anatomic site

Aorta
Ascending thoracic
Arch
Descending thoracic
Thoracoabdominal (types I to IV)
Abdominal
Suprarenal
Splanchnic and renal arterial involvement
Only renal artery involvement
Juxtarenal or pararenal (no normal aorta between upper extent of aneurysm and renal arteries)
Infrarenal
Iliac
Common
External
Internal

critical role in the overall assessment of the effectiveness of endovascular treatment strategies. All told, significant value exists in reporting an accepted and unifying measure of clinical success that combines the most significant of primary and secondary outcome criteria, which reflect the goals of this treatment method. The following section provides recommendations for the definition and reporting of clinical success and associated outcome criteria that impact on the clinical effectiveness of endovascular repair.

Definition of success

Defining the success of endovascular aneurysm repair remains dependent on a consideration of both clinical and radiographic criteria within the context of a historic standard established by open surgical repair in which the aneurysmal segment is treated with in situ prosthetic graft replacement. In this regard, a similar result can only be accomplished with an endograft, if complete exclusion of the aneurysm from the circulatory system is achieved. Some investigators, however, have suggested that the presence of a persistent type II endoleak after endovascular repair may not be an absolute predictor of late aneurysm expansion and rupture.³ Moreover, it has also been argued that a measured reduction in rupture risk may be possible even in the presence of a type II endoleak. In the absence of definitive data, the relative predictive value for this and other outcome criteria with respect to the risk of aneurysm rupture remains incompletely defined. Thus, future investigations may lead to further refinement of the recommended definitions of success presented herein.

Definition of technical success. *Technical success* relates to periprocedural events that occur from the initiation of the procedure and extend through the first 24-hour postoperative period. *Primary technical success* is defined on an intent-to-treat basis and requires the successful introduction and deployment of the device in the absence of surgical conversion or mortality, type I or III endoleaks, or graft limb obstruction. A technical success thus implies the following qualifying details:

1. Successful access to the arterial system using a remote site (ie, the femoral, external iliac, common iliac, or brachiocephalic arteries with or without use of a temporary or permanent prosthetic conduit to access these arteries);
2. successful deployment of the endoluminal graft with secure proximal and distal fixation;
3. absence of either a type I or III endoleak;
4. patent endoluminal graft without significant twist, kinks, or obstruction (>30% luminal stenosis or a pressure gradient >10 mm Hg) by intraoperative measurements.

Primary technical success can include the use of additional modular components, stents, or angioplasty, and adjunctive surgical procedures. However, if unplanned endovascular or surgical procedures are necessitated, the terms *assisted primary* or *secondary technical success*, respectively, should be used.

Secondary endpoints should be reported, such as procedure time, blood loss, blood transfusion, fluoroscopy time, contrast load, recovery time, range and average number of days in an intensive care unit, and hospital length of stay. However, these parameters do not enter into the consideration of technical success rates.

Definition of clinical success. Clinical success should be reported on an intent-to-treat basis and requires successful deployment of the endovascular device at the intended location without death as a result of aneurysm-related treatment, type I or III endoleak, graft infection or thrombosis, aneurysm expansion (diameter \geq 5 mm, or volume \geq 5%), aneurysm rupture, or conversion to open repair. Moreover, the presence of graft dilatation of 20% or more by diameter, graft migration, or a failure of device integrity classifies a case as a clinical failure. Clinical success can be claimed for those cases with a type II endoleak only in the absence of aneurysm expansion. As long as the significance of a type II endoleak and its implication as a marker for late clinical failure remains an area of active investigation, it is recommended that reports clearly indicate the proportion of patients classified as clinical successes that harbor a type II endoleak.

The presentation of clinically meaningful success rates mandates that the data are statistically valid for the time period in question. Specifically, the standard deviation of life table or Kaplan-Meier estimates should not exceed 10%. The following temporal characterization of clinical success is offered with this consideration in mind. *Initial* or *30-day clinical success* encompasses 30-day data. *Short-term clinical success* includes outcome measures reported within a 30-day to 6-month time frame. *Mid-term clinical success* refers to all outcome measures that are statistically significant up to 5 years after endograft implantation. *Long-term clinical success* includes all outcome measures that are statistically significant beyond 5 years.

Primary clinical success is clinical success without the need for an additional or secondary surgical or endovascular procedure. *Assisted primary clinical success* is clinical

success achieved with the use of an additional or secondary endovascular procedure. *Secondary clinical success* is clinical success obtained with the use of an additional or secondary surgical procedure (eg, the performance of a femoral-femoral bypass for treatment of a unilateral limb occlusion of a bifurcated endograft). Conversely, *clinical failure* includes a failure to deploy the endovascular device at the intended location, the presence of a type I or III endoleak, graft thrombosis or infection, aneurysm expansion (diameter ≥ 5 mm, or volume $\geq 5\%$), aneurysm rupture, conversion to open repair, or death as a result of aneurysm rupture or aneurysm-related treatment. Moreover, the presence of graft dilatation of 20% or more by diameter, graft migration, or a failure of device integrity classifies a case as a clinical failure. Aneurysm rupture should be reported as either a *procedure-related aneurysm rupture* (ie, perforation of the aneurysm during the course of the implantation procedure) or as a *late aneurysm rupture* that follows device deployment.

Longitudinal reporting of clinical outcome measures. The following parameters are recommended for inclusion in any comprehensive report of endovascular aneurysm repair: survival; rupture-free survival; prevalence of aneurysm rupture, death from aneurysm rupture, and aneurysm-related death; freedom from aneurysm expansion; freedom from type I and III endoleaks; prevalence of type II endoleak; prevalence of secondary endoleak; endograft patency; and technical and clinical success rates.

Life tables or Kaplan-Meier curves should be calculated for presentation of survival and rupture-free survival, maintenance of clinical success, risk of aneurysm-related death, as well as for freedom from aneurysm expansion and type I or III endoleaks. *Endograft patency* should be reported in life table or Kaplan-Meier format as primary, assisted primary, or secondary depending on the use of additional endovascular or surgical procedures.

Changes in renal function may occur as a result of suprarenal fixation of an endograft. Therefore, for those patients in whom an endograft extends above the renal artery orifices, a separate analysis should consider reporting parameters relevant to long-term renal function, including renal artery patency, incidence of segmental renal infarcts, estimated glomerular filtration rates, hypertension, and dialysis-free survival.

Comparing the clinical success of endovascular and open surgical repair. Investigations that compare open surgical and endovascular repair should report primary outcome criteria for both treatment groups, as previously defined. However, the use of a related although distinct definition of clinical success is necessary for patients treated with open surgery. *Primary technical success for open surgical repair* should be reported on an intent-to-treat basis and should require replacement or bypass of the aneurysmal segment with a prosthetic graft in the absence of mortality or graft thrombosis either during surgery or during the initial 24-hour postoperative period. If an unplanned surgical procedure is necessitated, such as a splenectomy or

reexploration for bleeding, the term *secondary technical success* should be used.

The definition of *clinical success for open surgical repair* includes the absence of death as the result of aneurysm-related treatment, graft infection or thrombosis, failure of device integrity, including graft dilatation 20% or more by diameter, and paraanastomotic aneurysm formation. Should open repair consist of aneurysm exclusion and bypass grafting, aneurysm expansion (diameter ≥ 5 mm, or volume $\geq 5\%$) or rupture would classify a case as a clinical failure. Definitions of *initial (30-day)*, *short-term*, *mid-term*, and *long-term clinical success* and of *primary*, *assisted primary*, and *secondary clinical success* otherwise remain unchanged, as do recommendations for longitudinal reporting of clinical data. Other significant outcome variables, such as device integrity, quality of life, and cost effectiveness can be compared with guidelines outlined below. Likewise, grading schemes for reporting complications and their severity, although primarily focused in this report on endovascular treatment, can be adapted with little modification for open repair. Finally, in comparison of two or more patient populations treated with open surgery and endovascular approaches, adjusting for case severity mix, particularly with respect to comorbid medical conditions, can be performed with schemes described elsewhere.⁴

Aortic and iliac artery remodeling

Because aneurysm size is the predominant factor determining risk of aneurysm rupture,⁵ changes in aneurysm dimension have been used as a surrogate marker for clinical efficacy after endovascular repair. Other morphologic changes, including progressive iliac angulation and aortic neck enlargement, may occur in response to either aneurysm exclusion or associated degenerative changes in adjacent segments, respectively. These late changes in aortoiliac anatomy have not been used in the definition of clinical success after endovascular aneurysm repair. Nonetheless, they may have a significant impact on late device performance and treatment durability. Thus, characterizing morphologic responses to endovascular intervention is useful both in defining clinical outcome and in serving to suggest mechanisms and engineering remedies in the event of a device failure.

Changes in dimension of the aortic aneurysm. In endovascular repair, the aneurysm sac is left intact and, as a consequence, this feature plays an important role in outcome assessment. Principally, changes in the dimension of the residual sac assist in defining the success or failure of aneurysm exclusion. Specifically, clinical correlation suggests that aneurysm growth after endovascular repair is an indicator of incomplete aneurysm exclusion, continued risk of aneurysm rupture, and a presumed treatment failure.⁶ In addition, secondary changes in the configuration of the aortoiliac segment, in response to a reduction in aneurysm size, may jeopardize the integrity or function of the endovascular graft.

Because variations in size occur in three dimensions, both sac volume and diameter are relevant parameters for defining changes in aneurysm size. It is noteworthy, however, that relatively small diameter shifts of 1 to 2 mm, that may otherwise be difficult to accurately measure with conventional imaging techniques, may be correlated with a significant change in aneurysm volume.⁷⁻⁹

Methodology for measuring changes in aneurysm diameter, volume, and length. Aneurysm size should be expressed as either maximum diameter or volume. Modality, method, and definitions should be clearly described, and comparisons should only be made between identical sources. Preferably, *maximum aneurysm diameter* should be measured perpendicular to the flow line of the vessel with three-dimensional reconstructed computed tomographic (CT) scan images. Because the aneurysm cross section often appears elliptic on axial images, the minor axis of the ellipse (smaller diameter) is generally a closer approximation of true maximum aneurysm diameter.¹⁰ The intraobserver and interobserver variability of diameter measurements obtained from CT scan images range between 2 and 5 mm or 5% and 15%.^{11,12} Therefore, a diameter change of 5 mm or more is considered significant. Blinding of observers and data sets are recommended to minimize bias.

Total aneurysm volume is defined as the volume within the native aortic wall, which extends from the level of the most caudal renal artery to a reproducible distal landmark, such as the aortic or iliac bifurcation. *Luminal aneurysm volume* is defined as the volume circumscribed by the endograft, while *nonluminal aneurysm volume* is comprised of thrombus and, if present, endoleak. Endograft dilatation may be associated with an increase in luminal volume, while reduction in aneurysm size is principally related to a decrease in nonluminal volume. The term *complete aneurysm resolution* should be used if the nonluminal aneurysm volume is less than 10% of the original nonluminal volume noted after endograft implantation. The intraobserver and interobserver variability for volume measurements have ranged between 3% and 5%. Therefore, a volume change of 5% or more is considered significant.⁸

A reduction in aortic length has been noted in association with a reduction in aneurysm size and may be associated with predisposition for endograft buckling, kinking, and component dislocation.¹³ Aortic length is recommended to be measured from the most caudal renal artery to a reproducible distal endpoint, such as the aortic bifurcation, along the flow line axis, as depicted in three-dimensional reconstructed spiral CT scan images.^{14,15} Of note, in the absence of three-dimensional reconstruction, aneurysm length is underestimated as determined from axial CT scan images.¹⁶⁻¹⁸ Moreover, limitations also exist for angiography performed with a calibrated marker catheter.^{14,19}

Changes in dimension of the aortic neck. Long-term aneurysm exclusion and device stabilization is dependent on the maintenance of an effective attachment, connection, or seal between the endograft and the host aorta. Therefore, dilatation of the aorta at the site or sites intended for primary endograft fixation may lead to treat-

ment failure either with device migration or via the occurrence of a new endoleak with aneurysm expansion.²⁰⁻²³

Methodology for measuring changes in neck dimension. Both diameter and cross-sectional area of the aortic neck at the sites intended for sealing or graft attachment are reportable parameters, and measurement perpendicular to the flow line is recommended. If the aortic segment does not run perpendicular to the plane of measurement, the smallest diameter (minor axis of the elliptical cross section) is an appropriate approximate of the true neck diameter.²⁰ Notably, the outer perimeter of the aortic neck wall should be used as the reference point for all measurements. Modality, method, and definitions should be clearly described, and comparisons should only be made between identical sources.

Changes in dimension of the iliac arteries. Progressive angulation of the aortoiliac segment leading to distortions of the endograft can accompany reductions in aneurysm size. This may result in endograft disruption or limb occlusion. Likewise, progressive dilatation of an iliac artery may also contribute to device instability or loss of effective aneurysm exclusion. Accordingly, alterations in iliac artery angulation and size after endovascular grafting and the consequent responses of the prosthesis may be important determinants of outcome.

Methodology for measuring changes in iliac artery diameter and tortuosity. Three-dimensional image analysis with appropriate anatomic referencing is recommended for accurate determination of iliac angulation and tortuosity. A second option is the presentation of measures derived from oblique, anteroposterior, and lateral plain abdominal x-rays that provide a simpler, albeit more approximate, approach. Admittedly, if the latter approach is used, variations in position, angulation, and equipment may have a significant impact on the reproducibility of derived measures. Definitions and categorization of iliac tortuosity and angulation have been detailed elsewhere.⁴

Methodology for reporting sequential changes in aortoiliac morphology. After placement of an endovascular prosthesis, the local biomechanical and hemodynamic environments of the native aorta are altered. As a consequence, dynamic changes in aortoiliac morphology are observed over time. The rate, magnitude, and direction of this response will be influenced by properties inherent to the chosen device and host aorta. Defining, with assuredness, the onset and durability of an unqualified treatment success will require capturing the time course and the direction of these associated morphologic changes.

Reporting morphologic changes that occur during a study period. Changes in aneurysm dimension should be reported in terms of prevalence (%) with the number of available subjects at each time period clearly stated. In this regard, the investigator should report the following outcome measures: (1) reduction in abdominal aortic aneurysm (AAA) diameter or volume by 5 mm or more or 5% or more, respectively; (2) enlargement in AAA diameter or volume by 5 mm or more or 5% or more, respectively; and (3) absence of significant change either in AAA diameter or volume. In characterizing the absence of aneurysm expan-

Table II. Classification of endoleak

Type	Cause of perigraft flow
I	a) Inadequate seal at proximal end of endograft b) Inadequate seal at distal end of endograft c) Inadequate seal at iliac occluder plug
II	Flow from visceral vessel (lumbar, IMA, accessory renal, hypogastric) without attachment site connection
III	a) Flow from module disconnection b) Flow from fabric disruption Minor (< 2 mm) Major (\geq 2 mm)
IV	Flow from porous fabric (<30 days after graft placement)
Endoleak of undefined origin	Flow visualized but source unidentified

sion at any time during a study period, life table or Kaplan-Meier analysis is an appropriate form for data presentation, as in freedom from aneurysm expansion.

Long-term studies are anticipated to provide data for two additional outcome measures of increasing interest: (1) complete aneurysm resolution; and (2) the rate of change of aneurysm diameter or volume, with a distinction between population subgroups that show either an enlargement or reduction in aneurysm size. Measurable changes in aneurysm dimensions have been reported immediately after endograft deployment. Therefore, changes in aneurysm size should be referenced to those measurements obtained from the first set of postoperative images. Likewise, similar methodologic approaches can be adopted for presenting the changes of other morphologic features, such as aortic neck diameter, aortic length, or iliac angulation.

Endoleak and endotension

Endoleaks, including their detection, potential clinical significance, and treatment, remain an active area of investigation. However, although it is now evident that an endoleak may resolve spontaneously, a proportion of those that do persist have been associated with late aneurysm rupture.²⁴⁻²⁷ Thus, endoleak remains an important measure of clinical outcome.

Definition of endoleak. *Endoleak* is defined by the persistence of blood flow outside the lumen of the endoluminal graft but within the aneurysm sac, as determined by an imaging study.^{28,29} An endoleak is evidence of incomplete exclusion of the aneurysm from the circulation and may be the result of an incomplete seal between the endograft and the wall of the blood vessel, an inadequate connection between components of a modular prosthesis, fabric defects or porosity, or retrograde blood flow from patent aortic side branches.^{29,30} Although intrasac pressure may approach systemic arterial pressure in the presence of an endoleak, some type II endoleaks have been associated with shrinking aneurysms and intrasac pressures that are substantially less than systemic values.³¹

Classification of endoleak. An endoleak can be classified according to time of occurrence relative to the oper-

ative procedure and site of origin.³² An endoleak first observed during the perioperative (≤ 30 days) period is defined as a *primary endoleak*, and initial detection thereafter is termed a *secondary endoleak*.²⁹ The reappearance of an endoleak either after spontaneous resolution or after an intervention that was considered successful is defined as a *recurrent endoleak*. Further categorization requires precise information regarding the course of blood flow into the aneurysm sac (Table II). A *Type I endoleak* is indicative of a persistent perigraft channel of blood flow caused by inadequate or ineffective seal at either the proximal or distal graft ends or attachment zones. Use of subscripts *a* or *b* indicate a type I endoleak originating at the proximal or distal ends of the endograft, respectively. In the case of an aortoiliac prosthesis, a type I endoleak may also refer to blood flow around an iliac occluder plug and should be indicated with use of subscript *c*. A *type II endoleak* is attributed to retrograde flow from lumbar arteries, the inferior mesenteric artery (IMA), or other collateral vessels. Origin and outflow sources of a type II endoleak should be specified, such as lumbar-lumbar, lumbar-IMA, accessory renal-lumbar/IMA, hypogastric-lumbar/IMA, or undefined. It should be emphasized that any connection of flow to proximal or distal graft ends or attachment zones, even in the presence of retrograde flow from a lumbar or IMA vessel, classifies an endoleak as type I. An endoleak caused by fabric tears or disruption, component disconnection, or graft disintegration is classified as a *type III endoleak*. Distinction between modular disconnection and fabric tear can be indicated through the use of subscripts *a* and *b*, respectively. A type III*b* endoleak can be further stratified with respect to the extent of fabric disruption as *major* (≥ 2 mm) or *minor* (< 2 mm). An example of the latter is late "microleaks," which can develop at the site of suture attachment of a fabric to supporting elements. Blood flow through an intact but otherwise porous fabric, observed during the first 30 days after graft implantation, is termed a *type IV endoleak*.³² This designation is not applicable to fabric-related endoleaks observed after the first 30-day period. If an endoleak is visualized on imaging studies but the precise source cannot be determined, the endoleak is categorized as an *endoleak of undefined origin*.

Endotension. It is now appreciated that an AAA may continue to enlarge after endovascular repair, even in the absence of a detectable endoleak, and that this enlargement may lead to aneurysm rupture.^{31,33-36} It has been proposed that this condition be classified as endoleak-related.³² Explanations for persistent or recurrent pressurization of an aneurysm sac include blood flow that is below the sensitivity limits for detection with current imaging technology^{32,34,37} or pressure transmission through thrombus^{38,39} or endograft fabric. On physical examination, the aneurysm may be pulsatile and intrasac measurements may reveal pressures that approach or are equal to systemic values.

At this time, endotension remains an investigative term for which some disagreement persists regarding its precise definition, appropriate usage, and current applicability. Endotension has been variably used in the literature to refer to

aneurysm enlargement in the presence or absence of an endoleak and to persistent or recurrent pressurization of an aneurysm sac, as determined with direct intrasac measurement.³⁶ It is anticipated that future research may lead to the development of a surrogate marker for sac pressurization, allowing more precise identification and definition of this phenomenon. However, in the interim, the majority view of this committee is that the term *endotension* be confined to instances of aneurysm enlargement after endovascular repair in the absence of a detectable endoleak. As such, endotension should not be classified as an endoleak of undefined origin.

Methodology for measuring the presence, source, magnitude, and physiologic significance of endoleak and endotension. Although conflicting studies exist, most recent investigations suggest that the sensitivity of contrast-enhanced CT scan imaging is superior to that of other noninvasive techniques, such as duplex ultrasonography, for endoleak detection.⁴⁰ Nonetheless, CT scan imaging may fail to identify an endoleak if delayed images are not obtained after infusion of contrast medium.⁴¹ Furthermore, the sensitivity and specificity of available imaging methods for endoleak detection have not been characterized. This may be particularly significant because of varying degrees of metal-induced scattering artifact that may obscure the identification of a small or subtle endoleak. Modality and method, including the use of angiography, should be clearly stated, and comparisons should only be performed with identical sources. Likewise, details should be provided regarding techniques used for direct pressure measurements of the aortic sac. Recently, specialized ultrasound scan techniques have been used to characterize the elastic modulus or stiffness of the aortic wall as a potential marker of intrasac pressure.⁴²⁻⁴⁴ Validation studies are not yet complete, and conflicting results have been reported.⁴⁵

Characterizing the frequency of endoleak in a study population during a defined observation period. The presence or absence of an endoleak is an important component in the definition of both early technical and late clinical success. Therefore, the initiation of postimplant examinations for endoleak identification should begin within 1 month after graft implantation. Although recommendations exist with respect to the timing of postoperative imaging studies, the optimal frequency of follow-up examinations has not been defined with prospective analysis. Current data do suggest, however, that secondary or recurrent endoleaks appear at an annual rate of approximately 10%, without evidence of a significant decrease in prevalence over time. Therefore, studies aimed at defining long-term success will continue to require late imaging data.

Reports are recommended to include the prevalence (%) of endoleak over the duration of the implant period. If bar graphs are used to represent each time interval, the data can be further stratified by: (1) time of endoleak occurrence (primary, secondary, recurrent); or (2) site of endoleak origin (types I to V), to provide additional perspective. Similarly, endoleak data can be correlated with aneurysm enlargement by plotting: (1) the proportion of patients

with a demonstrated endoleak; (2) the proportion of patients with a detectable endoleak and confirmed aneurysm expansion; and (3) the proportion of patients without detectable endoleak with demonstrable aneurysm expansion (ie, possible endotension). The number of patients available for analysis should be specified at each time point.

In characterizing the absence of endoleak at any time during a study period, life table or Kaplan-Meier analysis is an appropriate form for data presentation, as in freedom from endoleak or endoleak-free survival. In this regard, primary, assisted primary, and secondary curves, which are distinguished on the basis of whether or not a secondary surgical or catheter-based intervention was performed, can be used to assess the role of endoleak-directed intervention.

Device integrity

Despite premarketing fatigue testing of endografts, reports of structural failure throughout a wide range of device types continue to be noted with some frequency, including fractures of nitinol frames, elgiloy hooks, and disruption of endograft fabric.⁴⁶⁻⁵¹ To date, the total number of published reports that document a serious adverse clinical event as the result of a structural failure remains relatively small. However, as a new class of implantable medical prosthesis, the impact of material fatigue and failure on long-term durability and clinical efficacy remains an issue of significance. As endograft technology evolves, new concepts or design modifications will require validation in a clinical setting. A standard system for monitoring and reporting device integrity will facilitate this process.

Methodology for documenting the integrity of an endoprosthesis. The integrity of the endovascular device may be compromised at the time of deployment (*early device failure*) or at some late date after graft implantation (*late device failure*). If disruption of a component of the prosthesis is observed at the time of deployment, the role of operator error, including inappropriate patient selection or technical misjudgment, should be noted. Furthermore, reports should distinguish between failure in the delivery catheter, endograft, or related systems. A recommended system for grading late deformation of endografts is proposed. *Grade 0* indicates no device deformation. *Grade I* is used for devices with induced curvature, without acute angulation. *Grade II* refers to the presence of an angulation more than 30 degrees, and *Grade III* refers to angulation more than 30 degrees in association with device obstruction (*IIIa*) or component module disconnection (*IIIb*). The site of angulation should be specified.

Plain radiographs obtained in the anteroposterior, lateral, and oblique positions can be used for noninvasive assessment of the integrity of metallic graft components. High-resolution spiral CT scan imaging provides an additional approach, although all imaging methods remain limited in their ability to detect fabric failure. Investigations aimed at characterizing long-term device durability should contain late imaging data, which have been closely analyzed for device integrity. The prevalence of a structural failure in a carefully studied, unselected, and numerically significant

subset of implanted devices can serve as a proper estimate of device failure. However, it remains inappropriate to estimate the prevalence of a given structural failure mode based on the total number of implanted devices, unless all devices have been properly imaged with the specific purpose of detecting identifiable markers of the type of failure under investigation.

The *clinical significance of reported device failures* should be stratified as: 0, not associated with an adverse clinical event or necessitating increased surveillance or intervention; 1, necessitating increased surveillance but without clinical event; 2, necessitating intervention to control or manage; and 3, resulting in conversion, rupture, major complication (see section on Complications) or death. Reports of late device failure should also include potentially relevant clinical, anatomic, and hemodynamic parameters, such as implant duration, associated changes in aortoiliac morphology, and blood pressure.

Quality of life and cost-effectiveness studies

At a time of finite societal resources, the benefit provided by new interventions must be weighed against their expense. Therefore, studies designed to evaluate aortic endograft technology should include an assessment of both the cost of this technology and the quality of life of the treated patient. Formal Markov decision analysis models are recommended for application in studies designed to evaluate the cost effectiveness of endografts. The value of these models resides in their ability to incorporate intervention-related immediate-term and long-term expenditures and clinical outcome. In this regard, an effective analysis of the costs/benefits of aortic endografts will require determination of the impact of this technology on a large number of clinical outcome variables. Examples of variables that would favor the use of endografts with respect to open surgery include reduced hospital stay, diminished mortality and morbidity, improved quality of life in the perioperative period and over the long term, and reduced psychologic stress. These benefits, however, may be offset by high initial costs, expenditures related to monitoring and reintervention, and psychologic stress associated with the potential for aneurysm recurrence.

The effect of either open surgery or endovascular repair on a patient's quality of life can be prospectively evaluated with a variety of available instruments.⁵²⁻⁵⁴ For an economic analysis, cost rather than charge data should be gathered, analyzed, and include as many of the total costs as possible, including preadmission imaging, postimplantation surveillance, and late secondary procedures. In regional or national population-based economic studies, investigators should consider the additional costs of endovascular intervention for patients who would not otherwise be candidates for open surgery.

CATEGORIZATION OF OPERATIONS AND PROCEDURES

Operations and procedures determine the magnitude, complexity, and expense of endovascular aneurysm repair,

including all the additional procedures necessary to maintain a clinically durable result. Therefore, careful reporting will assist in efforts designed to enhance treatment efficacy either with revision of patient selection criteria, improvement of critical intraoperative adjuncts, such as imaging, anesthesia, and pharmacologic therapy, or refinement of device design. In all cases, identification of procedural goals and maneuvers and device components and configurations is important.

Categorization and definitions of endografting procedures, device configurations, and components

Precise description of the configuration, modularity, fabric, support, and fixation structures of the endografting system should be provided, if not detailed elsewhere, in addition to an accounting of all adjunctive components, devices, and maneuvers. Recommendations regarding uniform reporting of device configuration and components follow.

Configuration. Endograft configuration can be categorized either as a straight tubular aortic or an iliac graft, as an aortoiliac or an aortofemoral graft combined with contralateral iliac occlusion and femorofemoral bypass, or as a bifurcated or inverted Y-shaped graft. The prefix *branched* or *nonbranched* can be used to indicate the presence of side arms to visceral, brachiocephalic, or hypogastric vessels.

Modularity. Endografts should be categorized as comprised of a single unibody graft or modular components, which are assembled in situ within the vascular tree of the patient. Modular grafts must have overlapping junctions between components.

Endograft fabric. The nature of the graft material should be identified. For example, this material may be expanded polytetrafluoroethylene, knitted or woven polyester fabric (Dacron), or some other material or combination thereof. Reports should provide details regarding unique textile structures and whether the fabric is of standard thickness (ie, equivalent to that used in open repair). If fabric thickness is less than that commonly used for prostheses in open repair, the thickness should be specified.

Support system. The nature of the support skeleton of the device also should be defined. Reports should state whether the system fully or partially supports the graft, whether it is balloon expandable or self-expanding, and whether the supporting framework is fixed to the graft with stitches or otherwise incorporated within the graft. Likewise, the geometric configuration and the material composition also should be specified. For example, the support skeleton may be a braided or nonbraided framework and may be comprised of stainless steel, elgiloy, nitinol, or some other metal or plastic struts.

Fixation components and techniques. Reports should specify whether graft fixation is achieved with a component that is an integral part of the support skeleton or a separate or unique element of the endograft system. The geometric configuration of these components should be described, including whether hooks, pins, scales, barbs, or other means are used and whether fixation is to be

achieved with balloon or self-expansion or by a means separate from endograft deployment. The material composition and the degree to which the fixation component extends above or below the graft material should be specified.

In addition to characterizing the actual fixation components, the percent of graft oversizing relative to the host artery diameter at the intended fixation sites should be reported, because this may affect attachment and sealing. Finally, the intended placement of a fixation system below or above renal artery or other visceral or pelvic vessel orifices should be noted.

Endograft extensions and intraluminal stents. The nature of adjunctive devices to assure proper patency and positioning of the endograft should be described. For example, reports should include details of graft extensions and the use of adjunctive intraluminal balloon expandable or self-expanding stents.

Adjunctive maneuvers: preoperative, intraoperative, and postoperative

Definitions. A *principal procedure* is one that the surgeon believes to contribute most to aneurysm treatment, such as placement of endograft components. An *adjunctive procedure* is any other procedure that is designed to augment the effects of the principal procedure, such as angioplasty of an iliac artery stenosis or placement of a bypass graft between an external iliac and hypogastric artery. Such procedures may occur in the preoperative, intraoperative, or postoperative periods and should be designated in like manner. An *ancillary procedure* is one that does not contribute to the overall treatment of the aneurysm, such as the simultaneous repair of an inguinal hernia. *Primary procedures* refer to all interventions performed at the time of initial endovascular repair. *Secondary procedures* include all operations or endovascular interventions performed at a later date.

Preoperative adjunctive maneuvers. Categorization of preoperative adjunctive procedures is based on a consideration of unique sets of common objectives that are most often related to treatment of concomitant arterial occlusive disease to facilitate access or a prophylactic intervention to reduce the likelihood of endoleak or ischemia after endograft deployment. For example, the treatment of concomitant arterial occlusive disease may be necessary to improve access through the iliac arteries or to allow the treatment of renal or visceral disease, which may be rendered more difficult if performed after the principal procedure. In addition, the management of collateral arteries originating within the aneurysm may be necessary to prevent type II collateral endoleak after endoluminal repair of the aneurysm. The hypogastric arteries may require embolization when an aneurysm involves the abdominal aorta and the common iliac artery. The IMA and lumbar arteries may require embolization in AAAs, and the celiac artery may require embolization in thoracoabdominal aneurysms in which the superior mesenteric and renal arteries do not arise from the aneurysm. Finally, adjunctive procedures may be necessary in anticipation of ischemia that may result

Table III. Preoperative adjunctive maneuvers

Management of concomitant iliac/renal/visceral artery occlusive disease
Balloon dilatation
Stent deployment
Management of collateral arteries originating within the aneurysm
Embolization of hypogastric arteries
Embolization of IMA
Embolization of lumbar arteries
Embolization of visceral arteries (celiac)
Management of arterial branches to be deliberately rendered ischemic by the proposed principal procedure
Carotid/subclavian transposition or interposition graft for aneurysm disease adjacent to subclavian artery
Spleno-renal anastomosis
Hypogastric to external iliac artery bypass graft

Table IV. Intraoperative adjunctive maneuvers: Planned procedures

Management of access site
Percutaneous arterial access closure
Modified access sites
Iliac artery/aorta
Direct
Via prosthetic tube graft sutured end-to-side to iliac artery/aorta
Carotid artery
Repair of femoral aneurysm at access site by open technology
Management of collateral arteries within aneurysm
Embolization of hypogastric arteries
Embolization of IMA
Management of arterial tortuosity
Brachiofemoral wire
Direct or indirect manual deformation of artery
“Pull-down” maneuver
Management of occlusive disease of iliac arteries
“Sizing” with arterial dilator
Balloon dilatation

from the principal procedure, thereby necessitating prophylactic treatment. With this in mind, a categorization scheme for preoperative adjunctive maneuvers is provided in Table III.

Intraoperative adjunctive maneuvers. It is recommended that intraoperative adjunctive maneuvers be classified as planned procedures or unplanned procedures. *Planned procedures* comprise techniques that are part of a preformulated operative strategy, and *unplanned procedures* are necessary for management of unintended complications or an otherwise unsatisfactory outcome. Categorization schemes for planned and unplanned intraoperative maneuvers are provided in Tables IV and V, respectively.

Postoperative adjunctive maneuvers. Postoperative procedures should be categorized with respect to treatment objectives, including local wound management, endoleak, and graft limb occlusion. In addition to reporting procedures used in the management of graft limb obstruction, outcomes should be described in accordance with published standards for reports dealing with lower extremity

Table V. Intraoperative adjunctive maneuvers:
Unplanned procedures

Management of perforation of iliac arteries/aorta
Endovascular repair
Bypass graft
Conversion to open repair
Management of obstructed blood flow to lower extremity with thrombolysis, thrombectomy, endarterectomy, and patch graft or bypass graft†
Management of endoleak identified on table†
Balloon dilatation at anchor zones
Open mesh stent deployment at anchor zones
Proximal aortic cuff deployment
Distal limb extension deployment
“Deflector” graft to convert bifurcated to aortouniiliac graft
Instillation of thrombin, gelatin sponge, coils, or other thrombotic agents into aneurysm sac
Maneuvers designed to move an endovascular graft inferiorly
Traction on balloon catheter inflated within endograft
Traction on femorofemoral guidewire across prosthetic bifurcation
Traction on graft with bronchoscopy forceps within sheath
Conversion from endoluminal to open repair

†Placement of additional stents, extender cuffs, or instillation of thrombotic agents at time of primary endovascular aneurysm repair is not absolutely indicative of “unplanned” procedure.

Table VI. Postoperative adjunctive maneuvers

Management of wound complications at access site
Evacuation of hematoma
Operation for lymph fistula/lymphocele
Repair of false aneurysm
Management of ischemia of lower limbs
Balloon catheter thrombectomy/embolectomy at sites remote from access site
Thrombectomy/endarterectomy at access site
Bypass graft
Femorofemoral
Femorodistal
Thrombolysis
Procedures that may be required for endoleak management
Endovascular procedures
Balloon dilatation at anchor zones
Open mesh stent deployment at anchor zones
Secondary endoluminal repair with aortic cuffs, extensions involving deployment of secondary endoluminal prosthesis within primary prosthesis
Coil embolization of patent collateral channels
Laparoscopic procedures
Clip ligation of IMA
Banding of anchor zones
Open procedures
Hand sewn anastomosis of endolimb to native iliac artery
Banding of anchor zones
Open conversion

ischemia.⁵⁵ A categorization scheme for planned intraoperative maneuvers is provided in Table VI.

Conversion to open surgical repair. Conversion from endovascular to open repair may be required either at the original operation (*primary conversion*) or on a subsequent occasion (*secondary conversion*). Secondary conversion also should be classified as *urgent* or *elective*. For

example, secondary conversion for persistent endoleak in an asymptomatic aneurysm is an elective procedure, and conversion precipitated by aneurysm rupture or arterial occlusion is an urgent intervention. Details of conversion should be reported, including indication, site of aortic control, and other relevant operative information. Additional imaging studies, such as angiography or intravascular ultrasound, may be conducted in the operating room to assist in a final determination of aneurysm suitability for endovascular repair. *Therefore, intent-to-treat should be considered initiated by any maneuver directed at treating the aneurysm with an endovascular approach that follows the completion of intraoperative preprocedural aneurysm imaging.*

REPORTING DEATHS AND COMPLICATIONS

Standardized reporting of deaths and complications is necessary to establish endograft exclusion as safe and effective therapy for aortic aneurysm. These standards are necessary to compare endovascular procedures with other minimally invasive techniques and conventional surgery for patients at low and high risk. Thus, reports that carefully compile these data will lead to the development of criteria for selecting the most appropriate procedure for a given patient and provide scientifically valid information for advances in the technology of endograft exclusion.

Deaths

All deaths that occur within 30 days of the operative procedure should be categorized as *operative deaths* and classified as either *procedure-related* or *device-related*. Deaths that occur after 30 days should be considered *late deaths*. The cause of late death and its relationship to the implanted device or procedure should be noted. *Aneurysm-related deaths* should be reported explicitly and are defined as all deaths due to aneurysm rupture, a primary or secondary procedure, or surgical conversion. Because these issues are frequently difficult to determine in the absence of an autopsy or direct assessment at surgery, the reliability of the information used to make these determinations should be stratified. The cause of death should be classified as *verified* if on the basis of autopsy findings, direct surgical observation that defines the status of the aneurysm, or definitive imaging studies of the endograft obtained during the patient's terminal illness. When this level of information is unavailable, the cause and its relationship to the procedure and device should be classified as *probable* if the clinical picture is consistent and documented with reliable observations during the terminal illness. When these criteria cannot be met, the cause of death should be considered *indeterminate*.

Complications

Complications should be reported with standardized definitions and severity scoring to make appropriate comparisons with other methods of therapy and to objectively discern improvements or failures accruing from advancements in device design. Difficulties may arise in determin-

Table VII. Classification and grading of complications

<i>Complication</i>	<i>Grading</i>
Deployment-related complications	
Failed deployment with or without conversion	<ol style="list-style-type: none"> 1 No complications from attempted endovascular procedure, hospital stay not prolonged after endovascular repair 2 Conversion to open repair, no permanent disability 3 Significant permanent disability that impairs employment, function, or ability to live independently or death
Operative bleeding	<ol style="list-style-type: none"> 1 Autotransfusion < 2 units, no homologous transfusion 2 >2 units autologous, < 3 units homologous, limited incision for control 3 > 3 units homologous, laparotomy, thoracotomy, or necessitated exposure in addition to initial vessel cutdown to control bleeding
Aortic dissection (within 30 days of AAA repair)	<ol style="list-style-type: none"> 1 Incidentally noted, asymptomatic 2 Resolved with endovascular repair 3 Open repair or fatal
Arterial perforation or rupture	<ol style="list-style-type: none"> 1 Spontaneous closure 2 Stent graft or limited retroperitoneal iliac repair at primary procedure 3 Laparotomy/thoracotomy
Access artery dissection or thrombosis	<ol style="list-style-type: none"> 1 Incidentally noted, non-flow limiting dissection, local repair/prophylactic patch closure of access artery 2 Stent, limited retroperitoneal bypass, or necessitated return to operating room for thrombosis 3 Conversion to open AAA repair
Peripheral microembolization	<ol style="list-style-type: none"> 1 Resolution without tissue loss 2 Minor tissue loss, including toe or ray amputation 3 Major amputation or significant tissue loss
Peripheral macroembolization	<ol style="list-style-type: none"> 1 Resolution with intraoperative embolectomy at primary procedure 2 Embolectomy or other minor secondary operation, minor tissue loss 3 Arterial bypass or major arterial repair, major amputation
Access site hematoma	<ol style="list-style-type: none"> 1 Spontaneous resolution 2 Surgical evacuation 3 Nerve compression or associated arterial repair
Access site false aneurysm	<ol style="list-style-type: none"> 1 Resolved spontaneously, with compression, or thrombin therapy 2 Surgical repair 3 Ruptured
Access site lymphocele, lymphorrhea, lymphedema	<ol style="list-style-type: none"> 1 Resolution with or without aspiration, minor edema easily controlled with elastic support 2 Open drainage or repair 3 Permanent debilitating edema
Access site infection	<ol style="list-style-type: none"> 1 Resolved with oral antibiotics 2 Operative drainage, intravenous antibiotics 3 Major debridement, artery repair
Fever of unknown origin	<ol style="list-style-type: none"> 1 Prolongation of hospital stay
Implant-related complications	
Ruptured aneurysm	<ol style="list-style-type: none"> 2 Endovascular repair with survival and no permanent morbidity 3 Open surgical repair or fatal outcome
Endograft migration (> 10 mm relative to anatomical landmarks or any migration leading to symptoms or requiring therapy)	<ol style="list-style-type: none"> 1 No evidence of endoleak, graft obstruction, or aneurysm expansion 2 Secondary endovascular treatment 3 Explantation necessitated, AAA rupture, death
Endograft infection	<ol style="list-style-type: none"> 2 Apparently resolved or controlled with antibiotics 3 Endograft removal with extraanatomic or in situ repair
Device erosion through aortic or iliac wall	<ol style="list-style-type: none"> 2 Successful endovascular repair 3 Surgical intervention or fatal outcome
Intraoperative endograft limb obstruction	<ol style="list-style-type: none"> 1 Resolved at primary procedure and not associated with thrombosis 2 Limited retroperitoneal repair or thrombectomy 3 Bypass or conversion
Postoperative endograft limb obstruction	<ol style="list-style-type: none"> 2 Resolved with endovascular repair, minor tissue loss, including toe or ray amputation 3 Lytic therapy or open operative repair, major amputation
Buttock/leg claudication/ischemia	<ol style="list-style-type: none"> 1 Transient 2 Persistent but not disabling (controlled with exercise or pharmacotherapy) 3 Sufficiently disabling to necessitate intervention

(continued on next page)

Table VII. Continued.

Complication	Grading
Systemic complications	
Cardiac	1 Little or no hemodynamic consequence 2 Symptomatic necessitating intravenous medication, percutaneous transluminal coronary angioplasty/stent therapy 3 Severe hemodynamic dysfunction necessitating resuscitation, cardiac arrest, or fatal outcome
Pulmonary	1 Prompt recovery with medical treatment 2 Prolonged hospitalization or intravenous antibiotics 3 Prolonged intubation, tracheostomy, deterioration in pulmonary function, O ₂ dependence, or fatal outcome
Renal insufficiency	1 No dialysis 2 Temporary dialysis, prolonged hospitalization, permanently reduced renal function 3 Permanent dialysis, transplant, or fatal outcome
Cerebrovascular	1 Temporary deficit with recovery within 24 hours 2 Delayed recovery, infarct on CT or magnetic resonance, permanent deficit with mild impairment 3 Severe impairment or fatal outcome
Deep venous thrombosis	2 Anticoagulation therapy, inferior vena cava filter 3 Surgical or lytic therapy
Pulmonary embolism	2 Anticoagulation therapy, inferior vena cava filter 3 Hemodynamic instability, endovascular or surgical therapy, or fatal outcome
Coagulopathy	2 Transfusion therapy 3 Surgical intervention or fatal outcome
Bowel ischemia	1 Recovered without intervention 2 Recovered with intravenous antibiotics or total parenteral nutrition 3 Bowel resection, or fatal outcome
Spinal cord ischemia	1 Resolution within 24 hours 2 Resolution within 1 month or minor permanent deficit, able to walk without support 3 Major permanent deficit
Erectile dysfunction	1 Spontaneous recovery to preoperative status within 6 months 2 Functional with medical or injection therapy 3 No return of function or implant necessitated

ing precise gradations of severity. Nevertheless, complications should be assigned a severity score so that degrees of morbidity can be assessed and compared. The following scoring system has been modified from Rutherford et al.⁵⁵ *Mild* (1) indicates that the complication has occurred but resolved spontaneously or with nominal intervention, did not prolong hospital stay, and did not cause permanent impairment. *Moderate* (2) indicates the need for significant intervention, prolongation of hospitalization more than 24 hours, and at most, minor permanent disability that does not preclude normal daily activity. A *severe* complication (3) necessitates major surgical or medical intervention, may be associated with prolonged convalescence, is usually accompanied by prolonged or permanent disability, and may result in death.⁵⁶

Complications are presented in Table VII. Column 1 lists individual complications with specific definitions as needed and details for inclusion. Column 2 provides additional specifications for severity grading beyond the general criteria described previously. All complications should be classified as procedure-related or device-related, and anatomic site and presumed etiology should be reported where appropriate. All complications graded as moderate (2) or severe (3) are considered *major complications*, and those graded as mild (1) can be considered *minor complications*.

INVESTIGATOR DISCLOSURE AND CONFLICT OF INTEREST

Most publications relating to endovascular aneurysm repair will be based on information acquired during industry-sponsored clinical trials. In varying degrees, most authors and their institutions will have direct or indirect monetary relationships with these sponsors or their competitors. Although other factors have significant impact on author bias, financial issues are considered the most relevant. Disclosure does not in itself resolve the problem; however, it places this information before readers who can draw their own conclusions. Disclosure also may enhance authors' efforts to recognize and avoid bias in their presentations.

The International Committee of Editors of Biomedical Journals has established guidelines for disclosure of conflicts of interest.⁵⁷ On the basis of these guidelines, the Journal of Vascular Surgery has published its formal requirements for all authors of a submitted article.^{58,59} These requirements include disclosure of: (1) university, institutional, and corporate relationships; (2) a list of sources of funding received for the reported investigation and other investigations supported within 3 years of manuscript acceptance by organizations that may have a competitive or

antagonistic interest; and (3) potential financial conflicts, including consulting arrangements, board membership or employment, royalties, stock holdings, or option agreements with the company relating to the publication or its competitors. In addition to the admission of financial relationships, authors should describe within the Methods section of a manuscript the respective roles of study sponsors and investigators in: (1) study design; (2) conduct of the study; (3) data collection; (4) data analysis; (5) data interpretation; (6) writing of the manuscript; and (7) the decision regarding where and when to submit the report for publication. In this regard, clear relationships should be established between the investigator and company before the initiation of the investigation to avoid the potential of manuscript rejection by the Journal of Vascular Surgery or by other scholarly publications. As a final note, disclosure also offers an opportunity to reveal any further personal, academic, or other conflict that might introduce bias. These guidelines should be followed by all listed authors with a standard form provided by the publisher or as a formal statement in the body of the text or a footnote.

REFERENCES

- Ahn SS, Rutherford RB, Johnston KW, May J, Veith FJ, Baker JD, et al. Reporting standards for infrarenal endovascular abdominal aortic aneurysm repair. *J Vasc Surg* 1997;25:405-10.
- Johnston KW, Rutherford RB, Tilson MD, Shah DM, Hollier L, Stanley JC. Suggested standards for reporting on arterial aneurysms. *J Vasc Surg* 1991;13:452-8.
- Resch T, Ivancev K, Lindh M, Nyman U, Brunkwall J, Maline 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;28:242-9.
- Chaikof EL, Fillingim MF, Matsumura JS, Rutherford RB, White GH, Blankensteijn JD, et al. Identifying and grading factors that modify the outcome of endovascular aortic aneurysm repair. *J Vasc Surg* 2002;35:1060-6.
- Vardulaki KA, Prevost TC, Walker NM, Day NE, Wilmsink AB, Quick CR, et al. Growth rates and risk of rupture of abdominal aortic aneurysms. *Br J Surg* 1998;85:1674-80.
- Matsumura JS, Moore WS. Clinical consequences of periprosthetic leak after endovascular repair of abdominal aortic aneurysm. *J Vasc Surg* 1998;27:606-13.
- Singh-Ranger R, McArthur T, Corte MD, Lees W, Adiseshiah M. The abdominal aortic aneurysm sac after endoluminal exclusion: a medium term morphologic follow-up based on volumetric technology. *J Vasc Surg* 2000;31:490-500.
- White RA, Donayre C, Walot I, Stewart M. Abdominal aortic aneurysm rupture following endoluminal graft deployment: report of a predictable event. *J Endovasc Ther* 2000;7:257-62.
- Wever JJ, Blankensteijn JD, Th M Mali WP, Eikelboom BC. Maximal aneurysm diameter follow-up is inadequate after endovascular abdominal aortic aneurysm repair. *Eur J Vasc Endovasc Surg* 2000;20:177-82.
- Ouriel K, Green RM, Donayre C, Shortell CK, Elliott J, DeWeese JA. An evaluation of new methods of expression aortic aneurysm size: relationship to rupture. *J Vasc Surg* 1992;15:12-20.
- Lederle FA, Wilson SE, Johnson GR, Reinke DB, Littooy FN, Acher CW, et al. Variability in measurement of abdominal aortic aneurysms. *J Vasc Surg* 1995;21:945-52.
- Aarts NJ, Schurink GW, Schultze Kool LJ, Bode PJ, van Baalen JM, Hermans J, et al. Abdominal aortic aneurysm measurements for endovascular repair: intra- and interobserver variability of CT measurements. *Eur J Vasc Endovasc Surg* 1999;18:475-80.
- Harris P, Brennan J, Martin J, Gould D, Bakran A, Gilling-Smith G, et al. Longitudinal aneurysm shrinkage following endovascular aortic aneurysm repair: a source of intermediate and late complications. *J Endovasc Surg* 1999;6:11-6.
- Armon MP, Whitaker SC, Gregson RHS, Wenham PW, Hopkinson BR. Spiral CT angiography versus aortography in the assessment of aortoiliac length in patients undergoing endovascular abdominal aortic aneurysm repair. *J Endovasc Surg* 1998;5:222-7.
- Wever JJ, Blankensteijn JD, Broeders IA, Eikelboom BC. Length measurements of the aorta after endovascular abdominal aortic aneurysm repair. *Eur J Vasc Endovasc Surg* 1999;18:481-6.
- Beebe HG, Jackson T, Pigott JP. Aortic aneurysm morphology for planning endovascular aortic grafts: limitations for conventional imaging methods. *J Endovasc Surg* 1995;2:139-48.
- Resch T, Ivancev K, Lindh M, Nirhov N, Nyman U, Lindblad B. Abdominal aortic aneurysm morphology in candidates for endovascular repair evaluated with spiral computed tomography and digital subtraction angiography. *J Endovasc Surg* 1999;6:227-32.
- Shin CK, Rodino W, Kirwin JD, Wisselink W, Abruzzo FM, Panetta TF. Can preoperative spiral CT scans alone determine the feasibility of endovascular AAA repair? A comparison to angiographic measurements. *J Endovasc Ther* 2000;7:177-83.
- Diethrich EB. Will contrast aortography become obsolete in the preoperative evaluation of abdominal aortic aneurysm for endovascular exclusion? *J Endovasc Surg* 1997;4:5-12.
- Matsumura JS, Chaikof EL. Continued expansion of aortic necks after endovascular repair of abdominal aortic aneurysms. *J Vasc Surg* 1998;28:422-31.
- Sonesson B, Malina M, Ivancev K, Lindh M, Lindblad B, Brunkwall J. Dilatation of the infrarenal aneurysm neck after endovascular exclusion of abdominal aortic aneurysm. *J Endovasc Surg* 1998;5:195-200.
- Resch T, Ivancev K, Brunkwall J, Nirhov N, Malina M, Lindblad B. Midterm changes in aortic aneurysm morphology after endovascular repair. *J Endovasc Ther* 2000;7:279-85.
- Wever JJ, de Nie AJ, Blankensteijn JD, Broeders IA, Mali WP, Eikelboom BC. Dilatation of the proximal neck of infrarenal aortic aneurysms after endovascular AAA repair. *Eur J Vasc Endovasc Surg* 2000;19:197-201.
- Parodi JC. Endovascular repair of abdominal aortic aneurysms and other arterial lesions. *J Vasc Surg* 1995;21:549-55.
- Lumsden AB, Allen RC, Chaikof EL, Resnikoff M, Moritz MW, Gerhard H, et al. Delayed rupture of aortic aneurysms following endovascular stent grafting. *Am J Surg* 1995;170:174-8.
- White GH, Yu W, May J, Waugh R, Chaufour X, Harris JP, et al. Three-year experience with the White-Yu endovascular GAD graft for transluminal repair of aortic and iliac aneurysms. *J Endovasc Surg* 1997;4:124-36.
- Torsello GB, Klenk E, Kasprzak B, Umscheid T. Rupture of abdominal aortic aneurysm previously treated by endovascular stentgraft. *J Vasc Surg* 1998;28:184-7.
- White GH, Yu W, May J. "Endoleak": a proposed new terminology to describe incomplete aneurysm exclusion by an endoluminal graft. *J Endovasc Surg* 1996;3:124-5.
- White GH, Yu W, May J, Chaufour X, Stephen MS. Endoleak as a complication of endoluminal grafting of abdominal aortic aneurysms: classification, incidence, diagnosis and management. *J Endovasc Surg* 1997;4:152-68.
- Malina M, Ivancev K, Chuter TAM, Lindh M, Lanne T, Lindblad B, et al. Changing aneurysmal morphology after endovascular grafting: relation to leakage or persistent perfusion. *J Endovasc Surg* 1997;4:23-30.
- Gilling-Smith GL, Martin J, Sudhindran S, Gould DA, McWilliams RG, Baskran A, et al. Freedom from endoleak after endovascular aneurysm repair does not equal treatment success. *Eur J Vasc Endovasc Surg* 2000;19:421-5.
- White GH, May J, Petrask P, Waugh RC, Yu W. Type III and type IV endoleak: toward a complete definition of blood flow in the sac after endoluminal repair of AAA. *J Endovasc Surg* 1998;5:305-9.
- Stelter W, Umscheid T, Ziegler P. Three-year experience with modular stent-graft devices for endovascular AAA treatment. *J Endovasc Surg* 1997;4:362-9.

34. White GH, May J, Petrasck P, Waugh R, Stephen M, Harris J. Endotension: an explanation for continued AAA growth after successful endoluminal repair. *J Endovasc Surg* 1999;6:308-15.
35. White GH, May J. How should endotension be defined? History of a concept and evolution of a new term. *J Endovasc Ther* 2000;6:435-40.
36. Gilling-Smith G, Brennan J, Harris PL, Bakran A, Gould D, McWilliams R. Endotension after endovascular aneurysm repair: definition, classification and implications for surveillance and intervention. *J Endovasc Surg* 1999;6:305-7.
37. Schurink GW, Aarts NJ, Wilde J, van Baalen JM, Chuter TA, Schultze Kool LJ, et al. Endoleakage after stent-graft treatment of abdominal aneurysm: implications on pressure and imaging: an in-vitro study. *J Vasc Surg* 1998;28:234-41.
38. Faries PL, Sanchez LA, Marin ML, Parsons RE, Lyon RT, Oliveri S, et al. An experimental model for the acute and chronic evaluation of intra-aneurysmal pressure. *J Endovasc Surg* 1997;4:290-7.
39. Schurink GW, van Baalen JM, Visser MJ, van Bockel JH. Thrombus within an aortic aneurysm does not reduce pressure on the aneurysmal wall. *J Vasc Surg* 2000;31:501-6.
40. Sato DT, Goff CD, Gregory RT, Robinson KD, Carter KA, Herts BR, et al. Endoleak after aortic stent graft repair: diagnosis by color duplex ultrasound scan versus computed tomography scan. *J Vasc Surg* 1998;28:657-63.
41. Schurink GW, Aarts NJ, Wilde J, van Baalen JM, Chuter TA, Schultze Kool LJ, et al. Endoleakage after stent-graft treatment of abdominal aneurysm: implications on pressure and imaging: an in vitro study. *J Vasc Surg* 1998;28:234-41.
42. Malina M, Lanne T, Ivancev K, Lindblad B, Brunkwall J. Reduced pulsatile wall motion of abdominal aortic aneurysms after endovascular repair. *J Vasc Surg* 1998;27:624-31.
43. Wilson K, Bradbury A, Whyman M, Hoskins P, Lee A, Fowkes G, et al. Relationship between abdominal aortic aneurysm wall compliance and clinical outcome: a preliminary analysis. *Eur J Vasc Endovasc Surg* 1998;15:472-7.
44. Wilson KA, Hoskins PR, Lee AJ, Fowkes FG, Ruckley CV, Bradbury AW. Ultrasonic measurement of abdominal aortic aneurysm wall compliance: a reproducibility study. *J Vasc Surg* 2000;31:507-13.
45. Sonesson B, Sandgren T, Lanne T. Abdominal aortic aneurysm wall mechanics and their relation to risk of rupture. *Eur J Vasc Endovasc Surg* 1999;18:487-93.
46. Umscheid T, Stelter WJ. Time-related alterations in shape, position, and structure of self-expanding, modular aortic stent-grafts: a 4-year single-center follow-up. *J Endovasc Surg* 1999;6:17-32.
47. Riepe G, Heilberger P, Umscheid T, Chakfe N, Raithel D, Stelter W, et al. Frame dislocation of body middle rings in endovascular stent tube grafts. *Eur J Vasc Endovasc Surg* 1999;17:28-34.
48. Matsumura JS, Ryu RK, Ouriel K. Identification and implications of transgraft microleaks after endovascular repair of aortic aneurysms. *J Vasc Surg* 2001;34:190-7.
49. Najibi S, Steinberg J, Katzen BT, Zemel G, Lin PH, Weiss VJ, et al. Detection of isolated hook fractures 36 months after implantation of the Ancure™ endograft: a cautionary note. *J Vasc Surg* 2001;34:353-6.
50. Device Alert. AneuRx stent graft system: nitinol frame fracture after implantation. Available at: www.medical-devices.gov.uk/mda/mdawebsitev2.ns4, DA 2000(6). Accessed November 2000.
51. Device Alert. Vanguard endoprosthesis: upper stent row separation with or without nitinol wire fracture. Available at: www.medical-devices.gov.uk/mda/mdawebsitev2.ns4, DA 2001(1). Accessed January 2001.
52. Ware JE Jr, Sherbourne CD. The MOS 36-item short-form health survey (SF-36): I. Conceptual framework and item selection. *Med Care* 1992;30:473-83.
53. The EuroQol Group. EuroQol—a new facility for the measurement of health-related quality of life. *Health Policy* 1990;16:199-208.
54. Ruta DA, Garratt AM, Leng M, Russell IT, MacDonald LM. A new approach to the measurement of quality of life. The patient-generated index. *Med Care* 1994;11:1109-26.
55. Rutherford RB, Baker JD, Ernst C, Johnston KW, Porter JM, Ahn S, et al. Recommended standards for reports dealing with lower extremity ischemia: revised version. *J Vasc Surg* 1997;26:517-38.
56. Sacks D, Marinelli DL, Martin LG, Spies JB. Reporting standards for clinical evaluation of new peripheral arterial revascularization devices. *J Vasc Interv Radiol* 1997;8:137-49.
57. Editors. Uniform requirements for manuscripts submitted to biomedical journals. *JAMA* 1996;277:927-34.
58. Johnston KW, Rutherford RB. Disclosure of competition of interest. *J Vasc Surg* 1999;30:200-2.
59. Johnston KW, Rutherford RB. Policy on declaring conditions of funding for research studies. *J Vasc Surg* 2002;35:197.

Submitted Jan 15, 2002; accepted Feb 13, 2002.

Additional material for this article may be found online at www.mosby.com/jvs.