Nature and significance of endoleaks and endotension: Summary of opinions expressed at an international conference

Frank J. Veith, MD, Richard A. Baum, MD, Takao Ohki, MD, Max Amor, MD, Mohan Adiseshiah, MD, Jan D. Blankensteijn, MD, Jacob Buth, MD, Timothy A. M. Chuter, MD, Ronald M. Fairman, MD, Geoffrey Gilling-Smith, MD, Peter L. Harris, MD, Kim J. Hodgson, MD, Brian R. Hopkinson, MD, Krassi Ivancev, MD, PhD, Barry T. Katzen, MD, Michael Lawrence-Brown, MD, George H. Meier, MD, Martin Malina, MD, PhD, Michel S. Makaroun, MD, Juan C. Parodi, MD, Götz M. Richter, MD, Geoffrey D. Rubin, MD, Wolf J. Stelter, MD, Geoffrey H. White, MD, Rodney A. White, MD, Willem Wisselink, MD, and **Christopher K. Zarins, MD,** *New York, NY and Philadelphia, Pa*

Objective: **Endoleaks and endotension are critically important complications of some endovascular aortic aneurysm repairs (EVARs). For the resolution of controversial issues and the determination of areas of uncertainty relating to these complications, a conference of 27 interested leaders was held on November 20, 2000.**

Methods: **These 27 participants (21 vascular surgeons, five interventional radiologists, one cardiologist) had previously answered 40 key questions on endoleaks and endotension. At the conference, these 40 questions and participant answers were discussed and in some cases modified to determine points of agreement (consensus), near consensus (prevailing opinion), or disagreement.**

Results: **Conference discussion added two modified questions for a total of 42 key questions for the participants. Interestingly, consensus was reached on the answers to 24 of 42 or 57% of the questions, and near consensus was reached on 14 of 42 or 33% of the questions. Only with the answers to four of 42 or 10% of the questions was there persistent controversy or disagreement.**

Conclusion: **The current endoleak classification system with some important modifications is adequate. Types I and II endoleak occur after 0 to 10% and 10% to 25% of EVARs, respectively. Many (30% to 100%) type II endoleaks will seal and have no detrimental effect, which never or rarely occurs with type I endoleaks. Not all endoleaks can be visualized with any technique, and increased pressure (endotension) can be transmitted through clot. Aneurysm pulsatility after EVAR correlates poorly with endoleaks and endotension. An enlarging aneurysm after EVAR mandates surgical or interventional treatment. These and other conclusions will help to resolve controversy and aid in the management of these vexing complications and should also point the way to future research in this field. (J Vasc Surg 2002;35:1029-35.)**

Endovascular abdominal aortic aneurysm repair (EVAR) has been performed since 1990.[1] A variety of endograft devices have been used at centers around the world.[2-6] Although many dramatically successful early results have been achieved and many advantages are claimed, the mid-term and long-term durability of these grafts and their effectiveness in prevention of rupture remain questionable.[7-10] One of the principal reasons for failure after the 1st year is the occurrence of *endoleaks*, defined as persistent blood flow outside the graft and within the aneurysm sac.[11] Another reason is the presence of *endotension*, defined as a state of elevated pressure within the aneurysm sac.[12]

That endoleaks and endotension are critically important complications of many EVARs is widely but not universally agreed. However, many aspects of the nature, incidence, and significance of these problems are highly controversial. Some investigators believe that most endoleaks do not influence the outcome of EVARs,[13] whereas others consider most to be serious and the Achilles' heel of the procedure.[10,11] In addition, wide differences of opinion exist regarding the best treatment for endoleaks and endotension. Even the classification systems for these problems and the relationship between them remain subjects of controversy.[14,15]

To resolve some of these controversies and the conflicting recommendations for management related to endoleaks and endotension, a conference was held and included experts from several disciplines interested in EVAR in an attempt to reach agreement on as many key issues related to endoleaks and endotension as possible. Another purpose was to define areas of uncertainty or unanswered questions regarding these two entities. This article summa-

From the Montefiore Medical Center/Albert Einstein College of Medicine; and the Hospital of the University of Pennsylvania.

Supported in part by grants from the William J. von Liebig Foundation, the Guidant Corporation, Medtronic Inc, the James Hilton Manning and Emma Austin Manning Foundation, and the Anna S. Brown Trust.

Competition of interest: nil.

Reprint requests: Frank J. Veith, MD, Montefiore Medical Center, 111 E 210th St, New York, NY 10467.

0741-5214/2002/$35.00 + 0 **24/1/123095**
doi:10.1067/mva.2002.123095

Table I. Consensus participants

Vascular surgeons		*Interventional radiologists*	*Interventional cardiologist*
Veith	Meier	Baum	Amor
Adisehsiah	Malina	Ivancev	
Blankensteijn	Makaroun	Katzen	
Buth	Ohki	Richter	
Chuter	Parodi	Rubin*	
Fairman	Stelter		
Gilling-Smith	White, G.		
Harris	White, R.		
Hodgson	Wisselink		
Hopkinson	Zarins		
Lawrence-Brown			

*Expert in vascular imaging.

rizes the results of this consensus-seeking process and provides an overview of the current state of knowledge concerning endoleaks and endotension. This information should be helpful in guiding patient management decisions and may also prove useful in evaluating the overall effectiveness of EVAR and in guiding future investigations relating to these two important complications.

METHODS

To provide the most informed yet balanced overview of the endoleak/endotension field, the conference organizers (F.J.V., R.A.B.) selected as participants physicians who, irrespective of the specialty they represented, had the widest experience and greatest interest in endoleaks and endotension and who had studied these processes and published most extensively on them. Twenty-seven participants were chosen from three different specialties (Vascular Surgery, Interventional Radiology, and Interventional Cardiology) and nine different countries (United States, United Kingdom, France, Germany, Denmark, Sweden, Australia, Argentina, and the Netherlands). As shown in Table I, 20 of these participants were vascular surgeons, four were interventional radiologists, one was an expert in vascular imaging techniques, and one was an interventional cardiologist. All of the participants had extensive experience with EVAR or evaluation of patients for EVAR before or after the procedure. All had lectured and written widely about the diagnoses or management of endoleaks and endotension. All 27 of the selected participants agreed to take part in the process and its subsequent documentation.

Before the oral conference. All participants were sent a questionnaire requesting their responses to 40 key questions relating to endoleaks and endotension. These questions related to the timing, incidence, and nature of endoleaks and endotension, how they should be classified, how they should be diagnosed and managed, and how they are related to each other and to the outcomes of EVAR. These questions were designed to evaluate current points of agreement or disagreement about various critical issues regarding these complications. The answers to these questions plus discussion at the oral session served as the basis for the written documentation of the process. The questionnaire had room for comments in addition to the "agree," "disagree," or "uncertain" answers or percentage answers that were possible for most of the questions.

Oral consensus conference. The conference was held in New York City on November 20, 2000. A summary and analysis of the participant answers to each of the 40 original questions was presented. Discussion of each question and its answers followed. Two questions were modified slightly to permit answers to which most participants could agree. In other instances, participant answers were changed as the question was clarified with the discussion. After free discussion of each question and issue, a summary statement was made regarding each.

Written documentation. This article summarizes the participant answers to the most important and controversial questions posed and will also provide a prevailing opinion overview summarizing current thinking regarding endoleaks and endotension. It will highlight areas of uncertainty and directions for future investigation. In addition, a book will be published representing the results of the conference process in greater detail, with articles from most participants (Marcel Dekker, unpublished data).

Definitions. Answers to all 42 questions were analyzed and interpreted according to the following definitions. If 18 or more of the 26 responding participants agreed on a response to a question, that answer was considered to represent *consensus* or *general agreement.* If 14 to 17 of the 26 respondents agreed, that response was considered to represent *near consensus* or *prevailing opinion.* If less than 14 of the 26 participants agreed on a response to a question, that response was considered an area of *divided opinion, disagreement,* or *uncertainty.*

RESULTS

Overall results

Of the 40 questions originally posed to the conference participants, consensus or agreement with regard to the answer was attained on 22, and near consensus or prevailing opinion on an additional 14. The answers to the four remaining original questions reflected divided opinion or wide ranges of opinion, on the basis of conflicting views, uncertainty, or actual disagreement. In addition, at the oral conference, two derivative or altered questions were posed. With these modified questions, the answers were converted from prevailing opinion to consensus. Thus, consensus was reached on 24 of 42 or 57% of the answers to key questions posed, and near consensus was reached on 14 of 42 or 33% of the answers. Only with the answers to four of the 42 or 10% of the questions was there a wide range of divergent opinions.

The important areas of **agreement** included the following questions and answers.

Agreement Relating to Nature or Behavior of Endoleaks and Endotension

If a type I or type III endoleak is detected and then sealed (cannot be visualized on computerized tomographic scans), it will have no bad consequence? Of the 26 respondents, 20 disagreed, although one respondent stated that it could happen and another indicated that it could if abdominal aortic aneurysm (AAA) shrinkage occurred. Thus, consensus was reached that type I and type III endoleaks will have serious consequences, even if sealing appears to have occurred. The implication is that pressure can be transmitted through clot.

If a type II endoleak is detected and then sealed (cannot be visualized on computed tomographic scans), it will usually have no bad consequences? Twenty-one of the 26 respondents agreed with some qualifications. One commented "unless the AAA enlarges." Thus, consensus was reached that sealed type II endoleaks are usually benign. This question without the word "usually" elicited agreement by only 17 participants, indicating that not all type II endoleaks are benign.

Agreement Relating to Incidence of Endoleaks and Endotension

What is the total percentage of type I endoleaks at your institution (after 24 hours)? The answers ranged from 0 to 30%, with a mean of 7.5%. Twenty of the 26 responses (consensus) ranged from 0 to 10%. One comment indicated that the incidence rate was dependent on patient selection.

What is the total percentage of type II endoleaks at your institution (after 24 hours)? The answers ranged from 5% to 40%, with a mean of 17%. Eighteen of the 26 responses (consensus) ranged from 10% to 25%.

What percentage of type II endoleaks seal and have no detrimental effect? The answers ranged from 13% to 100%, with a mean of 53%. Eighteen of the 26 responses (consensus) ranged from 30% to 90%. One comment indicated that most type II endoleaks that seal are those detected at the original procedure. Thus, again consensus was reached that most but not all type II endoleaks will seal and behave benignly.

What percentage of patients in your institution will have delayed endoleaks develop within 3 years of endovascular abdominal aortic aneurysm repair? Answers ranged from 0 to 30%, with a mean of 10%. Eighteen of the 26 responses (consensus) ranged from 4% to 30%. Although two respondents answered 0%, another respondent commented that a delayed endoleak indicated a "missed" endoleak or device failure. However, the consensus was that delayed endoleaks do occur.

Agreement Relating to Diagnosis of Endoleaks and Endotension

Contrast computed tomographic scans with delayed images are the best method to detect endoleaks? Twenty of the 26 respondents agreed. Thus, consensus was reached that computed tomographic (CT) scans are currently the best method for detecting endoleaks, although comments were made that this method is not sensitive enough and that angiography determines the etiology more specifically.

Endoleaks can always be detected with appropriately delayed computed tomographic scans? Twenty-two of the 26 respondents disagreed. Thus, consensus was reached that some endoleaks could not be detected with even optimal CT scanning.

Simple diameter measurements are an adequate method to follow abdominal aortic aneurysm size and to document enlargement? Eighteen of the 26 respondents agreed. Thus, consensus was reached that simple diameter measurements could document AAA enlargement. However, comments were made that careful comparative measurements must be made and that focal enlargement could be missed.

Duplex scans are the best method to detect endoleaks? Eighteen of the 26 respondents disagreed. Consensus was, therefore, reached that duplex ultrasonography, although useful, was not the best method for detection of endoleaks. A comment was made that it was not accurate in detection of type II endoleaks.

Enlargement of an abdominal aortic aneurysm by more than 0.5 cm is indicative of endotension or an endoleak? With some qualifications, 19 of the 26 respondents agreed (consensus). Three believed an 8-mm increase is required because of measurement difficulties, and one was more comfortable with volume measurement changes.

Agreement Relating to Prevention and Treatment of Endoleaks and Endotension

Patent lumbar and inferior mesenteric branches should be treated in some way before endovascular abdominal aortic aneurysm repair? Twenty-three respondents disagreed. Thus, consensus was reached that such pretreatment should not be performed.

Patent hypogastric branches that can reflux into common iliac aneurysms giving rise to endoleaks should be treated in some way before endovascular abdominal aortic aneurysm repair? Nineteen of the 26 respondents agreed. Thus, consensus was reached on this issue, although five disagreed and two were uncertain.

Coils placed within the area of contrast visualization within an excluded abdominal aortic aneurysm sac after endovascular abdominal aortic aneurysm repair will result in thrombosis of any endoleak and elimination of endotension? Twenty-one of the respondents disagreed. Thus, consensus was reached that such sac coiling would be ineffective in treatment of particularly a type I or type III leak. Even if clot were induced, pressure would be transmitted to the sac wall. A possible exception was thought by some to be a type II leak in which thrombosis of a long branch might lead to decreased sac pressure.

An enlarging abdominal aortic aneurysm after endovascular abdominal aortic aneurysm repair without evidence of an endoleak should usually be repaired surgically or with a new endograft? Twenty of 26 respondents agreed. Thus, consensus was reached on the requirement for aggressive treatment in this circumstance. Two participants commented with the reservation that the AAA must be threateningly large and the patient suitably fit to withstand the procedure.

If a type II endoleak is to be repaired, what is your primary method of choice to do so? Twenty-two of the 26 respondents indicated that transarterial coil embolization was their preferred method via the hypogastric or superior mesenteric arteries. Several commented that it was essential to get as close to the AAA sac as possible. Several also commented that this method may be ineffective. Two respondents commented that laparoscopic branch clipping was their primary option, although it was the opinion of others that translumbar approaches will gain increasing recognition and use.[16]

Agreement Relating to Abdominal Aortic Aneurysm Pulsatility and Endoleaks and Endotension

If an abdominal aortic aneurysm is nonpulsatile on physical examination after endovascular abdominal aortic aneurysm repair, it indicates there is no endoleak or endotension? Eighteen of the 26 participants disagreed. Thus, consensus was reached that decreased AAA pulsatility on physical examination was not a good method for assuring freedom from an endoleak or endotension. One comment indicated that type II endoleaks rarely cause AAA pulsatility.

Prevailing opinion was reached on answers to the following questions.

Classification of Endoleaks and Endotension and Relationship Between the Two

The current endoleak classification system (types I to IV) is adequate? Seventeen of the 26 respondents answered that it is, although with some qualifications. Thus, the prevailing opinion was that, with some modifications, the current classification system for endoleaks and endotension is adequate. The modified classification system is detailed in Table II. Additional classifiers or descriptors are included in the footnote to Table II.

Finally, endoleaks and endotension may be associated with AAA enlargement, stability, or shrinkage. Also, the prevailing opinion was that some endoleaks could not be classified despite all efforts to do so and that AAA enlargement could sometimes occur in the absence of an endoleak and AAA shrinkage could occasionally occur despite the presence of an endoleak.

The presence of endotension without a visualized endoleak indicates the presence of an endoleak that is sealed (clotted) or otherwise not visualized? Sixteen of the 26 respondents agreed. Thus, the prevailing opinion was that endotension indicated the presence of a sealed or clotted endoleak (ie, a "virtual endoleak"). One comment indicated that pressure can be transmitted through clot, a point on which general agreement existed. Another comment suggested that pressure could be transmitted to the AAA sac through an intact graft, and another comment indicated that infection in the excluded AAA sac could result in endotension without an endoleak.

Type II endoleaks after endovascular abdominal aortic aneurysm repair usually produce systemic pressures within the abdominal aortic aneurysm sac? Fourteen of 26 respondents agreed. Thus, prevailing opinion was that type II endoleaks could produce systemic pressures within the AAA sac. However, 10 participants disagreed and two were uncertain, indicating some disagreement on this important point.

Table II. Classification scheme for endoleaks and endotension

Endoleaks (type)*	*Description source of perigraft flow*
I	Attachment site leaks†
A	Proximal end of endograft
B	Distal end of endograft
C	Iliac occluder (plug)
II	Branch leaks‡ (without attachment site connection)
A	Simple or to-and-fro (from only 1 patent branch)
B	Complex or flow-through (with 2 or more patent branches)
III	Graft defect†
A	Junctional leak or modular disconnect
B	Fabric disruption (midgraft hole)
	Minor (<2 mm; eg, suture holes)
	Major (≥2 mm)
IV	Graft wall (fabric) porosity (<30 days after graft placement)
Endotension§	
Type	
A	With no endoleak
B	With sealed endoleak (virtual endoleak)
C	With type I or type III leak‖
D	With type II leak‖

*Endoleaks also can be classified on basis of the time of first detection as: *perioperative*, within 24 hours of EVAR; *early*, 1-90 days after EVAR; and *late*, after 90 days. In addition, they can be described as *primary*, from time of EVAR; *secondary*, appearing only after not being present at time of EVAR; and *delayed*, occurring after prior negative CT scan results. Endoleaks also can be described as *persistent*, *transient* or *sealed*, *recurrent*, *treated successfully*, or *treated unsuccessfully*. Endoleaks and endotension may be associated with AAA enlargement, stability, or shrinkage.

†Some type I and type III leaks also may have patent branches opening from AAA sac and providing outflow for leak.

‡From lumbar, inferior mesenteric, hypogastric, renal, or other arteries.

§Endotension (*strict* definition) is defined here as increased intrasac pressure after EVAR without visualized endoleak on delayed contrast CT scans. In *generic* sense, endotension is any elevation of intrasac pressure and occurs with type I, type III, and most type II leaks and endotension in strict sense.

‖Detectable only on opening aneurysm sac.

Prevailing Opinion Relating to the Behavior or Diagnosis of Endoleaks and Endotension

What percentage of type II endoleaks produce bad outcomes? Answers ranged from 0 to 50%, with a mean of 16%. Fifteen of 26 respondents (prevailing opinion) believed that 2% to 15% of type II endoleaks were associated with bad outcomes. However, two participants commented that this may be because of an unrecognized type I leak.

Stability or absence of shrinkage in abdominal aortic aneurysm diameter after endovascular abdominal aortic aneurysm repair is indicative of endotension or endoleak or both? Fifteen of the 26 respondents disagreed. Thus, the prevailing opinion was that stability of AAA size does not necessarily indicate the presence of an endoleak or endotension. Comments were that large aneurysms (>6 cm in diameter) may not shrink and that AAA shrinkage may take years and depends on the type of endograft used.

Prevailing Opinion Relating to Abdominal Aortic Aneurysm Pulsatility and Endoleaks and Endotension

If an abdominal aortic aneurysm is pulsatile after endovascular abdominal aortic aneurysm repair, it is evidence of an endoleak? Fifteen of the 26 respondents disagreed. Nine agreed, and two were uncertain. Thus, prevailing opinion existed that pulsatility after EVAR was not evidence of an endoleak. Two comments indicated that such AAA pulsatility after EVAR was worrisome but inconclusive.

Divided opinions or **disagreement** existed on the answers to the following questions.

What percentage of type I endoleaks seal and have no detrimental effect? Answers ranged from 0 to 50%, with a mean of 15.5%. A clearly bimodal distribution of answers was seen, with 10 participants answering that none sealed and that immediate treatment was necessary. Nine other respondents thought that 10% to 50% could seal but rarely after 2 weeks. One comment indicated that all sealed type I leaks will recur.

Occlusion of both hypogastric arteries when necessary is a reasonably safe procedure and carries an acceptable risk? Twelve of 26 respondents agreed, nine disagreed, and five were uncertain. Thus, an obvious difference of opinion existed on this issue.

DISCUSSION

Endoleaks are a major unsolved problem when aortoiliac aneurysms are repaired with endovascular grafts. Yet the nature and significance of these endoleaks in individual patients remain unclear. Moreover, the definition and consequences of endotension and the exact relationship between endotension and endoleaks continue to be controversial. This conference process was designed to clarify the nature and significance of these endoleaks and endotension, to elucidate the relationship between these two complications, and to provide an overview of current knowledge and opinion regarding these two vexing problems that impact on the rapidly advancing field of EVAR. That many of the consensus opinions expressed within this process will change as new knowledge accumulates is recognized. As one participant (Wolf Stelter) commented, "Truth in science cannot be found by voting for it." Nevertheless, it is likely that the conclusions of this conference process will provide useful information on the incidence of endoleaks and methods to diagnose and treat them. Hopefully, these conclusions will also be helpful to others in the management of specific patient problems and in pointing the way toward investigations designed to advance knowledge and to clarify uncertainties.

Despite the potential limitations of conference conclusions, several areas of current agreement deserve emphasis. That in some circumstances elevated pressure can be transmitted through clot now appears likely.[17-20] This explains why coil embolization of type I or type II endoleaks may be ineffective. This also explains why some AAAs enlarge even when no endoleak can be detected and why endotension may occur without an endoleak.[12] However, other as yet unproven mechanisms may contribute to endotension and AAA enlargement in the absence of an endoleak.[15]

That type II endoleaks, although often benign and associated with AAA stability or shrinkage,[21] can lead to AAA enlargement and rupture and that this can occasionally occur when the leak appears to have been sealed by clot is now clear.[7,22,23] It is also becoming increasingly well recognized that type II endoleaks can produce intrasac pressures in the systemic range and that translumbar embolization is a more effective method for diagnosis and treatment.[16] However, this remains to be proven, and laparoscopic clipping of branches remains a popular alternative.[24]

The conference produced general agreement that simple CT scan diameter measurements were adequate to determine AAA size changes after EVAR. Volume measurements, although superior and a valuable research tool, were believed to be too cumbersome for general usage. However, several participants commented on difficulties that could occur with making accurate AAA diameter measurements on CT scan and that efforts should be made to avoid these pitfalls when determining diameter changes.

Several past concepts were clarified or corrected with the conference process. AAA pulsatility after EVAR was agreed to be a poor index of the presence or absence of an endoleak and endotension.[15,25] Furthermore, although some investigators in the past have believed that failure of AAA shrinkage or size stability after EVAR was evidence of an endoleak or endotension, the current prevailing opinion was that this was not so, particularly with large AAAs. Moreover, CT scanning was generally agreed to be a better diagnostic method for endoleak detection than duplex ultrasonography.

General agreement existed on several items that point the way toward future research requirements. Improved techniques for diagnosis of endoleaks and particularly endotension are needed. Presently no method exists to measure intrasac pressure noninvasively. Also agreed was that

hypogastric arteries or branches should not be allowed to retain open communication with the sac of aortoiliac or iliac aneurysms and that unilateral hypogastric embolization was justified. However, bilateral hypogastric occlusion, although sometimes justified, was believed to be best avoided. How often this was justified or what would be the best method to avoid it (hypogastric revascularization or branched endografts) was not certain at present.

The modified classification scheme (Table II) was introduced to permit a more detailed categorization of endoleaks and endotension. With endoleaks, this system is consistent with the classification proposed by The Society for Vascular Surgery/American Association for Vascular Surgery ad hoc committee on standardized reporting practices for EVAR,[26] although it does further subclassify type II leaks in a way that may explain why some behave differently. This will hopefully facilitate communication between investigators and lead to more accurate analyses of the natural history and the effectiveness of treatment methods. The Society for Vascular Surgery/American Association for Vascular Surgery classification includes, in addition to types I to IV, " endoleaks of undefined origin" explained as "flow visualized but source undetermined."[26] The classification does not classify endotension under endoleaks, but we have added a classification of endotension in the second part of Table II. This underscores our definition of endotension. Obviously, pressure is elevated within an aneurysm sac after EVAR with all types of endoleaks (endotension in the generic sense). However, the strict usage of the term endotension within our classification scheme is reserved for circumstances in which the intrasac pressure is elevated without a demonstrable endoleak on a delayed contrast CT scan. This definition and classification scheme will permit a more accurate categorization of the circumstances in individual patients. This in turn will permit more accurate determination of natural history and prognosis and more precise application of the most appropriate treatment.

The classification scheme presented in Table II includes a category for endotension without any endoleak (endotension, type A) even at open operation, and we and others have described such cases. It also includes a category (endotension, type B) with a sealed or clotted endoleak. This would be considered a virtual endoleak because there is no blood flow outside the grafts in the sac. Only when clot is removed from the branch orifice at operation does the leak become apparent, and we have had such a case.[23] In addition, patients with a type I or type III endoleak may not have it visualized on a CT scan but still have a high intrasac pressure (endotension, type C); and patients with a type II endoleak may not have a leak visualized but still have a high intrasac pressure (endotension, type D). In these latter two categories, the endoleak only becomes apparent when the aneurysm sac is opened at operation. Whether patients with type C and type D endotension behave like those with type I and III leaks and type II leaks, respectively, remains to be determined.

In conclusion, the conference process achieved remarkable agreement among leaders in the field from many countries. Hopefully, this agreement will be helpful in guiding current patient management. In addition to reaching agreement or near agreement on a number of complex and controversial issues, all the participants agreed that much remained to be learned about endoleaks and endotension and how they may be prevented and treated. These perplexing problems will continue to be a challenge to the success of EVAR and will have to be addressed aggressively by those interested in the field. Until solutions of these problems are found, EVAR will remain an imperfect long-term treatment and continued periodic follow-up examination to detect and treat these problems will be mandatory.

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Submitted Jun 20, 2001; accepted Nov 26, 2001.