Clinical failures of endovascular abdominal aortic aneurysm repair: Incidence, causes, and management

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Objective: **Despite well-documented good early results and benefits of endoluminal stent graft repair of abdominal aortic aneurysm (J Vasc Surg 2002;35:1137-44.)(AAA), the long-term outcome of this method of treatment remains uncertain. In particular, concern exists that late effectiveness and durability are inferior to that of open repair. To determine the incidence and causes of clinical failures of endovascular AAA repair, a 7-year experience with 362 primary AAA endografts was reviewed.**

Methods: **Clinical failures were defined as deaths within 30 days of the procedure, conversions (early and late) to open AAA repair, AAA rupture after endoluminal treatment, or AAA sac growth of more than 5 mm in maximal diameter despite endograft repair. Endoleak status per se was not considered unless it resulted in an adverse event. If clinical problems arose but could be corrected with catheter-based therapies or limited surgical procedures, thereby maintaining the integrity of successful stent graft treatment of the AAA, such cases were considered as *primary assisted success* and not classified as clinical failures.**

Results: **The average follow-up period was 1.5 years. Six deaths (1.6%) occurred after the procedure, all in elderly patients or patients at high risk. Five patients (1.4%) needed early conversion (immediate, 2 days) to open repair for access problems or technical difficulties with deployment, resulting in an implantation success rate of 98.6%. Eight patients (2.2%) underwent late conversion for a variety of problems, including AAA expansion (n = 4), endograft thrombosis (n = 1), secondary graft infection (n = 2), and rupture at 3 years (n = 1). Rupture occurred in an additional two patients for a total incidence rate of 0.8%. AAA sac growth of greater than 5 mm was observed in 20 patients (5.6%), four of whom have undergone successful catheter-based treatments to date. Overall, 39 patients (10.7%) needed catheter-based (n = 45) or limited surgical (n = 4) reinterventions for a variety of late problems that were successful in 92%.**

Conclusion: **In our 7-year experience, one or more clinical failures of endovascular AAA repair were observed in 31 patients (8.3%). Reinterventions were necessitated in a total of 10.7% of patients but were usually successful in maintaining AAA exclusion and limiting AAA growth. These results emphasize that endovascular repair provides good results and many benefits for most properly selected patients but is not as durable as standard open repair. (J Vasc Surg 2002;35:1137-44.)**

Standard open repair of abdominal aortic aneurysm (AAA) has been well documented as an effective and durable treatment that can be performed with highly acceptable morbidity and mortality rates in many experienced centers.[1-4] However, the risk of operation is reported as considerably higher (5% to 10% mortality rate) in numerous population-based reports, and many patients at high risk are often denied open surgical repair.[5-8] Major complications of open repair are experienced by at least 15% to 30% of patients after conventional open graft repair, and morbidity and mortality rates increase substantially in elderly patients or in those patients with pulmonary, cardiac, or renal comorbities.[9-11] In addition, long hospital stays, extensive rehabilitation, and prolonged convalescence are commonplace, and many elderly or frail patients never quite return to baseline function.[12] Furthermore, traditional open repair can lead to prolonged intensive care stays, extended hospitalizations, and other excessive resource use, which can become a strain fiscally to many hospitals in this current cost-conscious era.[13,14]

Since the introduction of endoluminal repair in 1991,[15] this technique has been eagerly accepted and used with rapidly increasing frequency in a growing number of centers within recent years. The potential advantages of reduced risk, quicker recovery, and possibly diminished care costs have generated intense interest on the part of patients, physicians, and industry alike. In September 1999, two devices were granted Food and Drug Administration (FDA) approval, and several devices currently in clinical trials will likely become commercially available within the next several years.

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Table I. Type and configuration of endograft device

	MGH	*Gore*	*Vanguard*	*EVT*	*AneuRx*	*Cook*	*Hybrid*	*Total*
ABI	59	50	29	38	76	10	5	267
AUNI	42			23				65
Tube	4		8	18				30
	105	50	37	79	76	10	5	362

MGH, Massachusetts General Hospital custom-made grafts; *hybrid*, custom cuff with commercial endograft in combination; *ABI*, aortobiiliac; *AUNI*, aortouniiliac.

Review of the current literature clearly documents the early feasibility and efficacy of endovascular repair of AAA.[16-21] However, some reports of mid-term experience have described a somewhat disturbing incidence of complications related to device failures, endoleaks, or other examples of treatment shortcoming, including continued AAA growth or even rupture despite endoluminal therapy.[22-27]

For these reasons, uncertainty remains regarding the long-term effectiveness of endoluminal AAA repair and its proper role in the management of patients with AAA. In particular, debate continues as to whether or not younger patients at good risk should be treated in this fashion or whether small aneurysms should be treated at an earlier interval in a more aggressive approach with stent grafts. To examine outcome data that might impact decisions on these issues, we reviewed a 7-year experience with 362 primary AAA endografts to determine the frequency of beneficial outcome and the incidence and causes of clinical failures of endovascular repair.

METHODS

Endograft repair of aneurysms or other vascular lesions began at the Massachusetts General Hospital in January 1994. Retrospective review up to January 2001 identified a total of 424 endovascular graft procedures during this 7-year experience. Sixty-two patients who underwent endoluminal repair of thoracic, iliac, or subclavian aneurysms or exclusion of various anastomotic aneurysms were excluded from consideration, leaving a total of 362 consecutive patients in whom treatment of primary AAAs by means of stent grafts was performed. These 362 patients treated during the 7-year period comprise the study group.

During the 7-year study period, a variety of endovascular grafts were used for aneurysm repair. Types and configurations of the devices are detailed in Table I. Five commercial endografts were used, as were a considerable number of custom-made devices. The custom-made devices were fabricated by our group from standard prosthetic graft materials (woven Dacron or polytetrafluoroethylene) and available intravascular stents (Gianturco Z-shaped self-expanding stents, Cook, Inc, Bloomington, Ind) as described originally by Dake and coworkers[28] and subsequently refined and improved by our group at the Massachusetts General Hospital.[29] These custom devices were most often used in patients with anatomic or clinical characteristics that made them ineligible for FDA protocols with commercially manufactured endograft devices. These features included short or ectatic (>28 mm) proximal necks, technically difficult or extensively aneurysmal iliac anatomy, prohibitive operative risk, renal insufficiency, or other adverse characteristics. In addition, custom devices were implanted at various intervals during the 7-year study period during which no FDA protocol studies were active and thus no commercial devices available.

Patient selection for the procedure and decisions regarding devices used were based on radiologic imaging studies. All patients underwent contrast enhanced computerized tomography (CT) scans with 3.0-mm cuts and three-dimensional reformatting. If initial measurements and morphology of the aneurysm were favorable for endovascular repair, multiplanar contrast angiography was performed with a special catheter with radiopaque markers at 1-cm intervals (Cook, Inc) to allow for precise length measurements and assessment of pelvic anatomy, particularly in regard to device access.

All procedures were performed in the operating room. The patients were prepared and draped for open repair in case this became necessary. Most patients underwent epidural anesthesia, and some needed general anesthesia. Open common femoral artery exposure by means of small groin incision was used for access. All patients underwent systemic heparinization. Radiologic imaging was performed with a high-quality portable C-arm fluoroscopic unit with digital imaging and road mapping capability on a radiolucent operating room table with movable top. A completion angiogram was obtained in all patients. The endoluminal device protocols were approved by the Institutional Review Board and Ethics Committee at the Massachusetts General Hospital.

Contrast enhanced CT scans and plain films were obtained in all patients before discharge and again according to protocol of the graft manufacturer, usually at 6 and 12 months and annually thereafter. If the CT scan was not done at the Massachusetts General Hospital, then films were obtained and reviewed by our staff.

Clinical failures of endovascular AAA repair were defined as the following events: periprocedural death (≤30 days), early (<30 days) or late conversion of endograft repair to conventional open surgical repair, increase in maximal AAA sac diameter of 5 mm or greater after endograft exclusion, and AAA rupture after endoluminal aneurysm treatment. Endoleak status itself was not considered, unless the endoleak resulted in an adverse event such as sac growth, conversion to open repair, or rupture.

Table II. Patient demographics

Total no. of patients	362
Age (years)	74.6 (44-93)
Male gender	302
Female gender	60
Average sac diameter (cm)	5.79 (3.0-10.2)
HTN	229 (63.6%)
CAD	208 (57.7%)
Tobacco use	188 (52.2%)
Diabetes	54 (15.0%)
Hyperlipidemia	117 (32.5%)
COPD	80 (22.2%)
Renal insufficiency	26 (7.2%)

HTN, Hypertension; *CAD*, coronary artery disease; *COPD*, chronic obstructive pulmonary disease.

In addition to these clinical endpoints defining failure of the endovascular repair, the frequency of subsequent secondary interventions to investigate, repair, or otherwise maintain the endograft was examined and the clinical problems needing such reintervention were documented. If clinical problems arose but could be corrected with catheter-based therapies or limited surgical procedure, thereby maintaining the integrity of successful stent graft treatment, such cases were considered *primary-assisted successes* and were not classified as clinical failures.

RESULTS

Eighty-three percent of treated patients were men. The mean age was 74.6 years (range, 44 to 93 years), and the mean AAA sac maximal diameter was 5.8 cm (range, 3.0 to 10.2 cm). Risk factors and comorbidities were typical of patients undergoing vascular surgical procedures. Patient demographics are displayed in Table II. The mean follow-up period for the study group was 18 months, with 219 of the procedures (60.5%) performed within the last 2 years of the study period. Follow-up of more than 4 years was available in nine patients, 66 patients had 3-year to 4-year follow-ups, and 68 patients had surveillance for 2 to 3 years after endograft repair.

In the 7-year experience, one or more clinical failures, as defined previously, were observed in a total of 31 patients (8.3%). Because some patients had more than one adverse event denoting failure, the number of such events (n = 42) exceeded the number of patients (n = 31) sustaining them.

Operative deaths. Six periprocedural deaths (1.5%) occurred in the total of 362 AAA stent graft repairs (Table III). One death occurred as the result of an acute myocardial infarction on day 2 after implant in a cardiac patient at high risk with known extensive coronary artery disease. The procedure had been prolonged with a series of technical deployment difficulties. Although eventually a technically successful implant was achieved, in retrospect, more prompt conversion to standard open repair would probably have been advisable.

Diffuse atheroembolization was the cause of another patient death after endoluminal stent graft placement. The patient had severe multisystem organ failure. The family refused dialysis and withdrew supportive care 2 weeks after surgery.

Table III. Perioperative deaths (<30 days)

Cause
MI after surgery
Massive atheroemboli
Died at home 5 days after discharge
Limb thrombosis-rhabdomyolysis
Avulsion EIA
Rupture 2 days after surgery

MI, Myocardial infarction; *EIA*, external iliac artery.

Table IV. Early conversions (n = 5)

Complication	*Operative correction*	*Time*
1 EVT limb twist	Aortobiiliac graft	Immediate
2 Failed insertions (small iliac artery)	Aorto bifemoral graft	Immediate
	Aortic tube; aortofemoral bypass	Immediate
1 Graft fell into sac	Aortic tube	Immediate
1 Early acute rupture	Aortobiiliac graft	48 hours

One sudden early death occurred at home 5 days after discharge and was presumed to be the result of rupture of a large symptomatic AAA. A proximal attachment leak had been suspected on predischarge CT scanning, but it had been elected to observe this for possible spontaneous resolution for a period of several weeks. Although no autopsy was performed to confirm rupture, the patient apparently had back and abdominal pain before collapse, and rupture seems the likely cause.

Another patient became hypotensive in hospital 2 days after apparently uneventful endoluminal repair. Emergency CT scan showed a large retroperitoneal hematoma suggestive of aneurysm rupture and a large endoleak believed to possibly represent a type 2 or 4 endoleak. An endoleak of indeterminate origin also had been observed on completion aortography after endograft implantation. Emergent open repair was carried out, but the patient died of renal and pulmonary insufficiency after a prolonged and complex 6-week postoperative course. In retrospect, possible vascular injury during endograft insertion and deployment was suspected, although rupture may have been caused by failure to exclude the AAA. The remaining two early deaths occurred after early complications of endograft implantation, one involving acute thrombosis of a graft limb and one iliac artery avulsion occurring on withdrawal of the delivery system, resulting in severe retroperitoneal hemorrhage. A common thread in most of these cases was technical complications occurring in patients who were elderly, fragile, and at high risk.

Early conversions. During the 7-year study period, five patients (1.4%) needed early conversion to open repair within the first 48 hours after the procedure (Table IV). One of the patients had a 360-degree twist in the limbs of

Table V. Late conversions (n = 8)

Complication	*Time*	*Intervention*
Sac Growth (4)	34 months	Infrarenal aortounilateral graft
	6 months	Infrarenal tube graft
	18 months	Infrarenal aortounilateral graft
	2 months	Juxtarenal repair AAA
Infected grafts (2)	2.5 years	Extraanatomic BPG
	2 years	Extraanatomic BPG; resection of old aorta, graft; insertion-PTFE aortic graft; L1,2 corpectomy
Bilateral kink and thrombosis (1)	26 months	Extraanatomic BPG
Rupture (1)	3 years	Infrarenal tube graft

BPG, Bypass graft; *PTFE*, polytetrafluoroethylene.

an unsupported Ancure bifurcated device (Guidant, Santa Clara, Calif) that could not be corrected. Two patients treated early in our experience had small iliac arteries that sustained severe traumatic injury during attempted passage of large-caliber devices. In retrospect, these cases are examples of poor patient selection and inappropriate judgments typical of an early "learning curve." Another patient needed immediate conversion when acute migration of the proximal attachment system occurred, resulting in the endograft falling into the AAA sac. The final patient in this subgroup had acute rupture of his aneurysm on postoperative day 2, as already described in the previous section detailing periprocedural deaths. Successful endograft implantation was achieved in the remaining 357 patients, for an overall procedural technical success rate of 98.6%.

Late conversions. Eight patients (2.2%) needed late (>30 days) conversion to standard open graft repair for a variety of reasons (Table V). Conversions were performed at a mean of 22 months after the original endograft procedure (range, 2 to 36 months).

Conversion was believed to be necessary in four patients because of continued AAA growth. Endoleak was present in all patients in this subgroup. Two patients had persistent proximal type 1 attachment leak, one a persistent type 2 leak from lumbar arteries, and one patient an acute late type 3 endoleak caused by a fabric hole erosion in a Vanguard endograft (Boston Scientific, Natick, Mass) implanted 6 months previously. The patient had done well, with sac shrinkage and no endoleak, up to the 6-month interval. The patient then was seen with sudden back pain, and CT scanning showed a type 3 leak with acute sac reexpansion. No rupture was found, however, at emergent open operation. No conversion in our series was caused by AAA sac growth without demonstrated endoleak ("endotension").

Two endoluminal grafts needed late conversion as the result of graft infection. Both were presumed to be caused by septicemic seeding of the endoluminal device. Although these cases might conceivably be the result of primary endograft infections, this is much less likely in our opinion. One patient did well until 18 months after the original procedure when he was seen at an outside facility with a septic left knee joint. The patient underwent treatment with drainage and antibiotics. Within several months, however, the patient returned again with bilateral septic knees and septic shoulder joint. Blood cultures grew methicillin-resistant *Staphylococcus aureus.* Again, the patient underwent treatment with drainage and antibiotics. Despite this, a left psoas muscle abscess developed that was drained percutaneously. However, subsequent CT scans showed communication with the endograft and the area of abscess. Further, evidence of bone destruction developed of vertebral bodies lumbar 1 and 2. Subsequently, the patient underwent a staged procedure of axillobifemoral bypass grafting followed by removal of the infected endograft with radical debridement of vertebral body osteomyelitis. The patient was discharged from the hospital after a prolonged hospital course. The second patient in whom infection developed had a somewhat similar history. The patient did well for 2.5 years after the original procedure, when he was seen at an outside facility with a septic left knee joint. This was attributed to infection of a subcutaneous vascular port device, which had been inserted several years previously for chronic treatment of hemochromatosis. The port was removed, and antibiotic treatment was instituted. However, several weeks later the patient returned with back pain. A CT scan revealed an increase in the size of the AAA with an inflammatory "rind" around it. On exploration, the graft was found to be purulent. The patient underwent removal of the endograft, aortic ligation, and extraanatomic bypass with a good outcome.

One patient needed late conversion at 27 months for acute thrombosis of the entire endograft, resulting in severe lower extremity ischemia. Prior follow-up CT scans had shown good AAA exclusion, with sac shrinkage and no endoleak. However, kinking of the endograft limbs was noted and believed attributable to the morphologic sac changes caused by its shrinkage. The patient was asymptomatic with intact pulses, so observation was elected. Presumably, limb kinking increased, resulting in thrombosis. Emergent axillobifemoral bypass grafting was performed, with a satisfactory result.

The final patient in this subgroup underwent conversion at 36 months for acute AAA rupture. This case will be described subsequently.

Sac growth. In the series, follow-up CT imaging revealed 20 patients with sac growth of the aneurysm of 5 mm or greater, despite endoluminal repair of the AAA. All of these patients had at least 90 days of follow-up. Of the 20 patients, four thus far have undergone successful treatment with endovascular therapies. Further secondary interventions are planned for most of the remaining patients with AAA sac growth but had not yet been performed when the study period was terminated. Such secondary procedures included a variety of catheter-based therapies, such as insertion of proximal or distal extender cuffs, branch or sac embolization, or related interventions as deemed appropri-

Table VI. Problems needing secondary interventions

Problem	*n*
Limb kinks/occlusions	
Supported devices	4
Unsupported devices	11
Attachment leaks/slippage	11
Type II leaks with growth	11
Endotension	1
Type III leaks	5
Native vessel stenosis	2
Total	45

ate with angiography or other diagnostic methods. None of the patients with sac growth are symptomatic, and no ruptures have occurred in this cohort.

Ruptures. During the 7-year experience, three patients (0.8%) are believed to have had AAA rupture after their endoluminal repair. Two patients, one with unproven but presumed rupture at home 5 days after surgery and the second with in-hospital rupture on postoperative day 2 (possibly traumatic), have already been described previously in the sections detailing periprocedural deaths and early conversions.

A final rupture occurred at 3 years after the original endovascular procedure, which was a tube endoprosthesis. The patient had undergone five interval CT scans that had shown no leak and a decrease in maximal AAA diameter from 5 to 3.6 cm. Shortly after the most recent follow-up CT scan, the patient was seen acutely with abdominal and back pain. Emergency CT scan showed a large endoleak and acute reexpansion of the AAA sac to the original 5-cm diameter, with an adjacent retroperitoneal hematoma. At emergency operation, acute detachment of the distal stent attachment mechanism of the tube endograft was found, with the distal endograft lying free in the AAA sac. We presumed the shrinking AAA sac wall had become atretic and ruptured when acutely repressurized. The patient survived operative conversion to open repair. After a prolonged hospitalization, the patient was discharged to a rehabilitation facility. Despite often extensive and emergent operations needed for late conversion of endovascular to open repairs, no deaths occurred as a result of such procedures in our series.

Secondary reinterventions. A variety of problems after endograft repair were identified at various intervals during clinical and radiologic postimplant follow-up surveillance (Table VI). These included persistent primary endoleaks, late secondary leaks, instances of graft migration, kinking, or thrombosis, and other problems that were believed to threaten endoluminal repair and expose the patient to possible conversion or rupture or both. For this reason, reinterventions were believed necessary.

During the 7-year study period, 39 patients (10.7%) needed a total of 49 secondary procedures. The vast majority of these were catheter-based reinterventions (n = 45), including reballooning, insertion of additional vascular stents in native vessels, proximal or distal extensions of the original stent graft device, embolization of branch vessels or the AAA sac itself, or similar related procedures. These were judged clinically effective in correcting or eliminating the problem needing reintervention in 92% of the 39 patients. Patients who underwent such success reinterventions were not classified as having clinical failures but rather as having assisted-primary successes.

DISCUSSION

This series summarizes the data from our initial 7-year experience with 362 patients with AAAs treated with endoluminal stent graft repair at the Massachusetts General Hospital. Our results confirm a growing number of reports from other centers that clearly document that endovascular AAA repair is safe and can be successfully performed in patients with suitable anatomy.[16-21] As in our series, the implant success in most centers is now approaching 98% to 99%, and this and other outcome parameters are likely to further improve with second-generation and third-generation devices.[30] In addition to low mortality and only a 1% early conversion rate, our results document quite effective treatment of the AAA relative to its anticipated natural history, albeit with a relatively short 1.5-year mean follow-up period. The AAA has remained stable in size or actually diminished in maximal diameter in 94% of cases, and serious late problems, such as conversion to open repair (2.2%) and AAA rupture (<1%), remain infrequent.

The less invasive characteristics of endoluminal repair are clearly reflected by the low morbidity and mortality rates reported in most series. The mortality rate of our series was 1.6%. Although this rate is not significantly different from results from several high-volume single institution reports involving traditional open repair, we believe many of the patients in our series who underwent endoluminal repair were truly high-risk patients, often with advanced cardiopulmonary problems or other comorbid problems, who would very likely have had considerably higher mortality rates if treated with conventional open operation. This contention remains unproven, of course, because no truly randomized prospective data exist in this regard. It is worthwhile emphasizing that most of the deaths in this series occurred after technical difficulties with the endovascular repair, usually in elderly, fragile patients with adverse challenging anatomic features. We believe this underscores the need for careful patient selection and adherence to accepted anatomic selection criteria.

Although *endoleak*, defined as a failure to totally exclude the AAA from continued perfusion and pressurization, remains a potential concern, we have not regarded this as a mode of clinical failure unless an adverse sequelae, such as continued AAA enlargement, AAA rupture, or other problems, resulted. This position may be challenged by some who believe that any demonstrated endoleak is a criteria of failure. However, the patient is unaware of an endoleak and not really concerned unless an undesirable outcome results. Indeed, the clinical significance of endoleak remains uncertain and poorly understood.[31-33] Several studies have shown poor correlation between endoleak

and outcome, and many authorities believe the most common variety of endoleak, type 2 retrograde branch leak, rarely causes clinical consequences.[34-36] However, it should be acknowledged that type I attachment leaks are well recognized as more hazardous in terms of AAA enlargement and rupture risk and that almost all type III endoleaks will need some form of reintervention or conversion. In addition, it should be noted that, in our series, all four patients with AAA sac growth resulting in conversion to open repair did have some type of endoleak, including one patient with a type II branch leak alone. Conclusion that endoleak is not a desirable or benign phenomenon seems justified, but we do not regard its presence alone as reliable prognostic predictor or a clear-cut indicator of clinical failure of endoluminal repair.

Similarly, in our opinion, the need for limited secondary reinterventions should not be considered an indicator of clinical failure of endoluminal AAA repair. As illustrated by our series in which 11% of patients needed secondary procedures, almost all catheter-based endovascular interventions rather than surgical procedures, the vast majority (92%) were believed clinically successful in correcting the presumed cause of sac growth or other clinical problems, thereby maintaining the integrity and success of the endograft repair. Similar success rates have been reported by other investigators with respect to secondary interventions.[37,38] We believe the concept of primary-assisted success, achieved by means of such limited reinterventions, is valid and well accepted by patients as long as successful endovascular treatment of their aneurysm can be maintained and major surgical repair avoided.

Although our reintervention rate was a relatively modest 11%, our relatively short mean follow-up period of 18 months must be recognized. In the large European collaborative registry (Eurostar) experience of more than 1000 patients followed for 12 or more months, 18% have needed secondary interventions at a mean follow-up interval of 20 months.[37] With life-table analysis, cumulative freedom from reintervention at 1, 3, and 4 years was 89%, 67%, and 62%. Thus, it appears reasonable to assume that secondary interventions will be necessary at a cumulative rate of approximately 10% per year. Similar Eurostar data have emphasized the ongoing and cumulative incidence of both late conversions and aneurysm rupture, noting cumulative rates of approximately 2%/year for conversion to open repair and a rupture risk of approximately 1%/year.[39]

The less invasive nature of the procedure, and the generally good and beneficial early results of treatment, clearly have made endovascular AAA repair an appealing, if not compelling, therapeutic alternative to many patients with AAA. Application of this method has increased rapidly in many centers around the world, and many investigators now urge more widespread use. Some regard it as the procedure of choice for all AAAs that are anatomically suitable and believe it is reasonable to use even in young patients at good risk. Other advocates urge prophylactic repair of small (<5 cm) AAA, with the belief that the safer and less invasive treatment would justify earlier treatment and potentially improve long-term outcomes. However, we believe our results, and mid-term results reported by other investigators,[40,41] should give some thoughtful concern in this regard. The long-term effectiveness and durability of endovascular repair clearly appears to be less than that anticipated by most surgeons after standard open operative repair.[2,4] Although it must be acknowledged that conventional surgical repairs are rarely subjected to the intense scrutiny and postimplant surveillance common to endovascular repair, nonetheless long-term effectiveness of endograft repair as we now know cannot match the late outcome and reliability of standard AAA operative repair. Second-generation and third-generation devices may improve endoluminal outcomes—most series to date, including this report, are dominated by results of earlier, first-generation endografts.[42] This remains to be established, however. In addition, future device advances and improvements may reduce device structural failures and may enable the endoluminal grafts to better accommodate to morphologic AAA sac changes that have been recognized by many authorities and that contribute to late failures by causing endograft kinking, migration, component separation, and other adverse consequences.[43,44]

We believe the clinical implications of our data are several. First, endoluminal AAA repair has clearly been a major advance. Its feasibility, safety, and generally good early and mid-term results have been well shown in our series and many other published reports. It appears particularly beneficial to more elderly patients at high risk, many of whom may have previously been denied repair. In such patients with suitable anatomy, it is reasonable and appropriate in our opinion to consider endovascular repair the procedure of choice.[45] However, it should be recognized that the actual definition of "high-risk" is open to some debate and not well defined in the literature. Similarly, endoluminal repair seems advantageous in patients with a "hostile" abdomen because of a variety of factors and also an appealing and likely beneficial option in patients with other unusual conditions that may cause technical difficulties and challenges for conventional open repair, such as paraanastomotic aneurysms after previous aortic surgery, AAA in the presence of a horseshoe kidney, and AAA in patients with prior renal transplants.

Secondly, because of current concerns related to device structural stability and long-term reliability of this form of repair, in our opinion, more widespread use of endografts to repair small AAA cannot be supported.[46] Similarly, because endoleak, graft migration, and other failure modes of endoluminal repair are much more frequent in patients with adverse anatomy, this procedure should not be used overaggressively in patients who do not have well-defined appropriate aneurysmal anatomic features. This is particularly true in patients at very high risk because the need for conversion in these circumstances is likely to be associated with truly excessive morbidity and mortality rates.[39,47,48] One cannot overemphasize the importance of proper and appropriate patient selection.

Whether or not the procedure should be recommended to younger patients at good risk remains unclear.[45] Given current technology, the increasing evidence of problems related to device durability, more frequent need for reinterventions, and the small but definite continued risk of AAA rupture, standard open repair still seems best for such patients. It is mandatory, in our opinion, that vascular surgeons properly inform patients of options, risks, and potential benefits and shortcomings of both open and endoluminal repair, so that they may make a truly informed decision. Although many of these patients may still opt for the less invasive repair even if fully advised, at least they will be aware of the "tradeoffs" involved and perhaps better committed to the need for repeated imaging and closer long-term follow-up. Patient interest and demand for less invasive treatment and societal pressures propelling quicker, more simplified, and less costly procedures will surely accelerate. Recognition of such forces serves to reemphasize the need for continued scientific scrutiny and evaluation of endograft repair.

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DISCUSSION

Dr Frank LoGerfo (Boston, Mass). I just have one question about endoleaks. I think you said that only sac growth, conversion, or rupture were regarded as failures. You could have an endoleak, have a pulsatile aneurysm, and it could essentially be no different from the aneurysm without the endograft present, but that would be considered a success. Is that correct?

Dr Jeffery Dattilo. That is correct. We think that the correlation between leak and eventual abdominal aortic aneurysm growth or rupture is unclear in the literature. This phenomenon, as you know, is controversial. However, in cases with persistent endoleak, we usually institute more frequent follow-up imaging and clinical examinations.

Dr Gregorio Sicard (St Louis, Mo). It was a wonderful presentation of one of the biggest experiences in the country, and I will share with you some of our own experience, which is similar, in St Louis.

I do have a question regarding your third conclusion. I think we are getting used to saying endovascular repair cannot match open repair. Obviously, without a randomized trial, we will not be able to know the final answer. During the 7-year period you report at the Massachusetts General Hospital, there were a large number of open aneurysm repairs performed. Did you look at those numbers and compare them with endoluminal repair? Something that has changed with endovascular repair is patient surveillance. Close surveillance has become mandatory. We do not have that same approach to open repair, and very few series have looked in a longitudinal manner at the graft-related complications of open repair. I will show you some data in my lecture that it is not as low as we think it is. So, could you give us in that similar 7-year period the graft-related complications with open repair, what was the follow-up, and what is the follow-up surveillance protocol used in your service for open repair? How often do you get CT scans to see if you have a paraanastomotic aneurysm? Very few services do that. Thank you.

Dr Dattilo. Thank you, sir. Those are very interesting questions. To address the open aneurysm follow-up in our practice, we have not involved the current open repairs into our database with similar veracity as we do with the endoluminal repairs. We do collect data on the open repairs, but as you well know, open repair dates back to the 1960s and the data collection at that period of time was not as large in scope as the endoluminal repair databases that we are collecting now. I find that comparing those two groups is going to be difficult, and we are not comparing in any kind of randomized prospective way endoluminal or open repair at our institution.

Dr Jack Cronenwett (Lebanon, NH). I have two questions. First, did you find as you went through your study longitudinally that the incidence rate of these complications decreased? As you know, the Sydney group has reported in their sequential analysis that they did not see the decrease in complications they expected, perhaps because they expanded and enlarged the population and the indications to include higher risk patients.

My second question relates to your incidence rate of reintervention of approximately 10%. Could you tell us what that looks like with life-table analysis? In other words, is it a relatively linear curve over time, and if so, given that, you have done most of these patients within the past year or 2, wouldn't you expect that this number is going to increase with longer follow-up?

Dr Dattilo. I am going to answer the second question first. Clearly that is the case. Life table analysis would not offer much value in our current data set. Perhaps with longer follow-up this tool would be useful. Our data tend to mirror the Eurostar data, which demonstrate that reinterventions on a cumulative basis are approximately 8% to 10% per year. Does the incidence rate over time change? For each subset of our clinical failures, it does not correlate on a time-related basis, meaning two of our deaths were in fact recent deaths. So that does not correlate with previous work in the literature suggesting once you get over the steep learning curve or rely upon device improvements you could obviate such adverse outcomes.