Recommended Reading | VASCULAR SURGERY, PART I
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SPECIAL COMMUNICATION

Screening for abdominal aortic aneurism: A consensus statement

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THE PROBLEM

Treatment of abdominal aortic aneurism (AAA) with minimally invasive techniques has recently gained tremendous national and international attention. However, enthusiasm for this new technique has diverted attention from an equally important issue, that of early detection or screening for aneurysms. Over the past 20 years, despite advances in diagnostic imaging and in general medical care of patients, there has been essentially no change in the number of patients seen in US hospitals with ruptured AAA.1 Approximately 15,000 persons die of ruptured AAA and dissections each year.2 However, this may be the tip of the iceberg. It is estimated that 300,000 persons per year die suddenly without receiving medical care.3 Furthermore, studies have shown that the incidence of ruptured AAA in cases of sudden death ranges from 4% to 5%.4-6 Thus the yearly death rate from ruptured AAA could be as high as 30,000. This is comparable to a yearly mortality of 32,000 for prostate cancer and 42,000 for breast cancer.2 The foregoing data strongly emphasize the increasingly recognized7 need for a strategy that will enable early detection of aneurysms.

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COST AND EFFICACY OF SCREENING

When evaluating the cost and effectiveness of screening programs, four important issues must be considered: cost, invasiveness, and accuracy of the screening test; prevalence of the disease; efficacy of interventions to treat the disease; and cost of these interventions. Screening for AAA can be performed with a simple noninvasive ultrasound study. It is well-documented that a limited ultrasound examination is extremely accurate in identifying the presence of AAA.8 The prevalence of AAA is quite high if selected populations are screened. For example, the incidence of AAA larger than 3 cm in all men older than 60 years is 4% to 8%.9-14 If patients have cardiovascular risk factors, such as smoking, hypertension, or history of peripheral arterial disease, the incidence of AAA increases two to five times.15 The prevalence of AAA larger than 3 cm in women older than 60 years is only 1.5%.16-19 However, in female patients with a family history of aneurysm or with multiple cardiovascular risk factors the incidence of AAA is also two to three times higher than in those without these factors.20 The efficacy of treatment of large aneurysms is profound. The yearly incidence of rupture and death in patients with AAA larger than 5.5 cm is 16%, compared with perioperative mortality of 2% to 6% for open repair.1,10,13,14,19,21-24 Moreover, recent data suggest that the mortality rate for endovascular AAA repair may be as low as 1%.25 Thus patients with large aneurysms clearly benefit from repair.

As of yet, there is no definitive treatment for “small” aneurysms, and a screening program will identify many of these. Nevertheless, rate of growth of small AAs is relatively predictable. With appropriate surveillance, early identification of small aneurysms is quite beneficial for those patients with aneurysms that enlarge and reach treatment thresholds. In addition, emerging data suggest that medicines such as doxycycline, and risk factor modification may retard aneurysm expansion.26-29 Early identification of aneurysms will enable application of these treatments and analysis of their efficacy.

Although AAA repair with open or endovascular techniques is expensive, the cost more than doubles if repair is performed emergently.1 When these various factors were
incorporated into a Markov decision analysis model, AAA screening was found to be cost-effective.\(^8\) The cost per quality-adjusted life year saved for screening men older than 60 years was $11,285. This number compares favorably with the cost-effectiveness of other well-accepted interventions, such as coronary artery bypass grafting ($26,117)\(^9\) or hemodialysis ($54,400).\(^3\) Of interest, the cost-effectiveness of AAA screening appears to be similar to that of screening mammography ($16,000-$20,000).\(^2\) As might be anticipated, AAA screening is not cost-effective in patients older than 84 years.\(^8\)

**PROSPECTIVE STUDIES**

The benefit of screening for AAA has been demonstrated in six prospective randomized studies.\(^10,11,13,14,19,21-23\) Although these studies were performed in multiple countries, with variable patient cohorts, the findings are surprisingly similar. Male patients of various ages were invited to participate in ultrasound screening, and subsequently aneurysm-related mortality rates in the screened and unscreened populations were compared. Patient response to the request for screening was high (74%-84%), and follow-up ranged from 4 to 10 years.\(^10,13,14,19,21-23\) In screened patients the authors observed a remarkable 45% to 49% reduction in incidence of ruptured AAA\(^10,13\) and a 21% to 68% decrease in aneurysm-related deaths.\(^10,13,14,19,21\) The largest of these studies was a recently published randomized trial in the United Kingdom that involved 70,495 men ages 65 to 74 years.\(^10\) Eighty percent of patients responded to the request for screening. Mortality associated with elective AAA repair was 6%. At 4 years the authors found a 42% reduction in deaths from AAA in the invited group. Moreover, the mortality curves for screened and unscreened patients in this trial continue to diverge after 4 years.

**CONTROVERSIAL ISSUES**

Several concerns have been raised about the utility of population-based screening for AAA. It has been proposed that patients who are found to have “small” aneurysms will experience a diminished quality of life related to concern about rupture.\(^33,35\) Level of anxiety, however, appears to diminish when a prudent plan of treatment is provided.\(^35,36\) As with any screening program, there will be patients who do not participate. However, similar screening programs within and outside the United States enjoy acceptance rates that range from 75% to 88%.\(^10,14,37,38\) Moreover, very little cost is incurred for patients who do not participate in screening. Aortic aneurysm disease is one of the least-known killers in American society. Initiation of an educational program to inform seniors and their physicians of this disease will increase the rate of response to screening and constitute an important step in a strategy to prevent death from aneurysm rupture. Last, critics have suggested that screening may identify a large number of patients who are unfit for surgery.\(^33\) However, Irvine et al.\(^23\) found that patients identified through screening were healthier than those in whom aneurysms were discovered incidentally. Moreover, endovascular techniques will also likely reduce the percentage of patients who are unfit for aneurysm repair.

**RECOMMENDATIONS**

On the basis of available data we recommend baseline ultrasound screening for AAA in the following patient cohorts:

- **All men aged 60 to 85 years**
- **Women aged 60 to 85 years with cardiovascular risk factors**
- **Men and women older than 50 years with a family history of AAA.**

Patients who appear unfit for any intervention should not be screened. On the basis of available data we recommend subsequent surveillance of screened patients as follows:

- **Aortic diameter less than 3 cm, no further testing**
- **AAA 3 to 4 cm in diameter, yearly ultrasound examination**
- **AAA 4 to 4.5 cm in diameter, ultrasound examination every 6 months**
- **AAA greater than 4.5 cm in diameter, referral to a vascular specialist.**

**CONCLUSIONS**

There are compelling data that in appropriately selected patient cohorts identification of AAA can save lives at a cost to society that compares favorably with other well-accepted interventions. Inasmuch as reimbursement remains the major impediment to acceptance of aneurysm screening, we strongly encourage that insurers adopt a policy that allows payment for this life-saving test.

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Medical Management of Small Abdominal Aortic Aneurysms
B. Timothy Baxter, Michael C. Terrin and Ronald L. Dalman

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Aneurysmal degeneration of the abdominal aortic and iliac arteries (referred to as abdominal aortic aneurysm, or AAA) is a common and frequently lethal age-related disease process. The prevalences of unsuspected, asymptomatic AAA in men and women over the age of 60 years are 4% to 8% and 0.5% to 1.5%, respectively. Advanced age, high total serum cholesterol, known coronary artery disease, hypertension, male gender, and family history have been the most frequently recognized AAA risk factors in prior screening studies. Of these, smoking is the most important. In a recent cohort study of >100 000 health maintenance organization participants, after a median 13-year follow-up, additional factors associated with subsequent AAA development include treated and untreated hypertension, high total serum cholesterol, known coronary artery disease, and intermittent claudication. Black race and Asian race are inversely associated with AAA risk in men. Ruptured AAA and complications after surgical treatment are responsible for at least 15 000 deaths per year in the United States, but some estimate the actual rate may be as high as 30 000 deaths per year, a mortality rate that approaches those associated with prostate and breast cancer. Open surgical repair for larger aneurysms is effective in preventing rupture but is morbid; newer endovascular exclusion strategies are less morbid but may require additional interventions and do not always prevent aneurysm rupture. Most investigators agree that AAA disease, in its initial stage, is an inflammatory condition associated with benign dilation. At some point, typically when the aneurysm enlarges beyond 5 cm, progressive degenerative changes predominate, which leads, in some cases, to mechanical failure. Although research involving genetically deficient mice has identified key proteins involved in experimental aneurysm formation, a detailed pathophysiologic understanding of human disease is still incomplete. Major clinical challenges in AAA disease include the absence of diagnostic biomarkers and effective nonsurgical therapies to prevent progression of early-stage disease. Biomarkers indicative of progression or efficient imaging indices to monitor metabolic
activity will be important in guiding suppressive medical therapies for small aneurysms.

The abdominal aorta between the renal arteries and the iliac bifurcation is the most common extracranial site of aneurysm formation. Although aneurysms are generally defined by a 50% increase in native vessel diameter, aortic diameter increases with age even in the absence of overt disease. Consequently, some controversy exists as to when a large infrarenal aorta becomes an AAA. Some investigators have used an absolute aortic diameter of 3 cm. This distinction has limitations, however, because it falls within the upper limits of the normal aortic diameter related to the body habitus and advancing age. Because of these limitations, others have used relative measures compared with nondiseased aortic segments or adjacent vertebral bodies. Relative aortic indices (eg, 1.5 or 2.0 ×) are, however, less useful in the setting of diffuse arteriomegaly or generalized aortic enlargement. An absolute diameter of 3.5 cm or greater represents a practical compromise, separating the large aorta with age-related changes that will not progress from frank aneurysmal disease.

**Current Treatment Modalities**

Mechanical intervention is currently the only treatment shown to be effective in preventing AAA rupture and aneurysm-related death; it is reserved for AAA ≥5.5 cm in diameter for men and ≥5.0 cm in women. Although smaller aneurysms do rupture, the likelihood of aneurysm-related death only exceeds treatment risks above these thresholds. This conclusion in men is based on data from 2 large-scale randomized clinical trials. These studies did not have a sufficient number of women to precisely identify the size threshold for repair. The threshold of 5 cm, used by most clinicians for women, is based on a higher rate of rupture of 5- to 5.5-cm aneurysms in women. In the United States, elective AAA repairs, generally performed for asymptomatic or medically stable patients, averaged 87.7 per 100 000 Medicare patients between 2000 and 2003. In the last decade, endovascular aneurysm repair has gained acceptance as an alternative to open surgical repair, with reduced periprocedural risks. Several Food and Drug Administration–approved commercial endovascular aneurysm repair devices are currently available and in use. A reflection of the impact of endovascular aneurysm repair strategies and devices is that perioperative (30 day) mortality for all elective AAA repairs declined from 5.0% to 3.7% (P < 0.001) between 2000 and 2003 with no change in outcome for open repairs alone. By 2003, endovascular repair accounted for 41% of elective AAA repairs.

Although treatment outcomes have clearly improved for surgical patients in the endovascular aneurysm repair era, evidence from prospective screening studies on 3 continents suggests that a substantial window of opportunity exists for earlier intervention in AAA disease. After congressional passage of the Screening for Abdominal Aortic Aneurysms Very Efficiently (SAAAVE) Amendment in 2006, the Centers for Medicare and Medicaid Services in the United States added a screening AAA ultrasound examination to the Initial Preventative Physical Examination, or IPPE, for new program enrollees as they turn 65 years of age. This benefit is extended to men between the ages of 65 and 75 years who have smoked at least 100 cigarettes in their lifetime and men and women in this age range with a family history of AAA disease. The IPPE must be completed within 6 months of Medicare eligibility. This benefit was justified on the basis of a review by the US Preventive Services Task Force that concluded that ultrasound screening may reduce AAA mortality by 43% in men aged 65 to 75 years. The potential utility of intervention in smaller (<5.5 cm) AAAs was considered, but the risks of surgical repair greatly outweighed the potential benefit of reduced AAA rupture, even when the likelihood that widespread screening would identify thousands of new patients with smaller AAAs was taken into account. In the largest previous US screening study, 90% of AAAs identified were <5.5 cm in diameter. In the next several years, increasing awareness of AAA disease driven by provider and patient education related to this new screening benefit could dramatically increase the pool of small AAA patients seeking treatment options for early-stage disease.

**Measuring Aneurysm Progression**

Traditionally, aneurysm diameter has been used as the principal surrogate marker for disease progression. For purposes of population-based disease screening studies and to determine the timing of surgical intervention, abdominal ultrasound imaging has proven accurate and reproducible. Although more is being learned about AAA biology and progression, aneurysm diameter remains the most important clinical determinant for risk of rupture. Interpretation of ultrasonic diameter data is frequently complicated by lengthening of and increased angulation from the axial plane during disease progression. This variability may be overcome in part in the course of serial examinations. CT imaging protocols may be reproducible to within a millimeter when a standardized technique is used in their interpretation. The computerized reconstruction available with CT images provides accurate 3D images that allow for planning of operative repair, but the cost, risk, and inconvenience associated with CT imaging do not lend themselves to screening and surveillance applications.

**Factors That Influence Aneurysm Progression**

The most common method of AAA detection is an abdominal imaging study obtained for an unrelated problem. The mean growth rate for small AAAs (≤5.5 cm) is 2.6 to 3.2 mm per year, which increases with aneurysm diameter. Studies of AAA expansion and the factors associated with expansion have been limited by sample size or a limited number of serial observations. In the United Kingdom Small Aneurysm Trial (UK SAT), AAA expansion in 1743 patients followed up for up to 7 years was most strongly associated with diameter at baseline. No association with growth rate was noted for age or gender. Self-reported cigarette smoking status was associated with an incrementally increased growth rate of 0.4 mm per year, which persisted after adjustment for potential confounding variables. Of other potential risk factors considered in the UK SAT, including hypertension, peripheral arterial occlusive disease, total or high-density lipoprotein
plasma cholesterol concentration, and diabetes mellitus, only the presence of peripheral arterial disease or diabetes influenced aneurysm growth, with peripheral arterial disease decreasing it by 0.2 mm per year for each 0.2 change in ankle brachial index (95% CI = 0.03 to 0.25) and diabetes reducing the growth rate by 0.79 mm per year (95% CI 0.27 to 1.33 mm). On the basis of these data, investigators calculated that screening intervals of 36, 24, 12, and 3 months for patients with AAA diameters of 35, 40, 45, and 50 mm, respectively, yielded less than a 1% chance of the AAA unexpectedly exceeding 55 mm in diameter between examinations. In clinical practice, examination intervals vary but rarely exceed more than 12 months, with increasing frequency associated with progressive enlargement.27 Part of the reason for the more frequent studies is reassurance for both the patient and physician. Quality-of-life surveys indicate that diagnosis without treatment of AAA can be associated with significant anxiety.28

Although not considered in the analyses of most AAA trials, lifelong patterns of lower-extremity exercise may provide some protection from AAA. Computational flow-modeling studies of hemodynamic conditions in the distal aorta suggest that the decreased flow from prolonged sedentary existence may promote aneurysmal disease.29 Indirect clinical evidence in support of this concept includes the fact that above-knee traumatic amputation30 and chronic spinal cord injury20 are associated with increased AAA risk independent of other risk factors, including cigarette smoking.

**Tobacco**

Tobacco smoking as a specific risk factor for AAA disease prevalence, incidence, and progression deserves special mention. The relative risk of AAA in individuals who have ever smoked is 2.5 times greater than the relative risk for coronary heart disease. AAA is more closely associated with cigarette smoking than any other tobacco-related disease except lung cancer.10 Nearly all AAA patients (>90%) relate a history of smoking; however, only about half of those continue to smoke at the time of diagnosis.11 Several small studies have associated continued cigarette smoking with more rapid aneurysm expansion. Chang and associates9 found a significant correlation with continued smoking and aneurysm expansion. MacSweeney et al11 monitored 43 patients with small (median size <4.0 cm) AAAs to assess active smoking (serum cotinine levels), blood pressure, cholesterol, and triglycerides. Only active smoking was associated with a small but significant increase in growth rate. Lindholt32 evaluated and prospectively followed up 117 AAA patients; he found a positive correlation between continued smoking and the rate of expansion.33 In the UK SAT itself, smoking and initial aneurysm size were the only 2 factors positively associated with aneurysm growth, although that study did not find a dose response between self-reported smoking habits or serum cotinine levels and aneurysm growth rate.27 Animal studies have confirmed accelerated aneurysm growth with smoking, although the mechanism for this effect does not appear to be related to a direct increase in matrix metalloproteinase (MMP)-9 levels.33 When the studies are considered together, continued smoking appears to be associated with a relatively small (15%) increase in growth rate that has important implications when compounded over several years. At the present time, smoking cessation should be considered one of the most certain approaches to decreasing the rate of aneurysm expansion.

**Statins**

Statin therapy reduces the progression of atherosclerosis and improves clinical outcomes in cardiovascular diseases. Although effective in reducing atherogenic lipoproteins, statins also demonstrate additional biological effects (ie, pleiotropic effects), including reduction of C-reactive protein levels, that may be relevant to the pathogenesis of AAA disease.14 Several studies have found an association between the presence of AAA and total cholesterol.8,12 There is, however, no clear relationship between total cholesterol and AAA expansion rate.27,31,32 Despite the absence of a relationship between cholesterol and growth rate, there is evidence from a number of studies to suggest that statins may influence aneurysm growth rate, presumably via these pleiotropic effects. Simvastatin therapy at 2 mg · kg⁻¹ · d⁻¹ reduces both aortic diameter and the percentage of mice with aneurysms after elastase infusion.35 No changes in effect size were noted when these experiments were repeated in hypercholesterolemic apolipoprotein E–deficient mice.

MMP-9 expression is closely linked to aneurysm formation in animal models of AAA. In human AAA specimens explanted for organ culture, addition of cervistan (0.001 to 0.1 μM/mL) significantly reduces tissue levels of both total and active MMP-9 in a concentration-dependent manner.36 Cervistan did not reduce the number of macrophages or neutrophils present in cultured aneurysms, which suggests that statin therapy inhibited inflammatory cell activation. In a prospective study by Evans et al,37 patients were randomized to a 3-week preoperative course of simvastatin versus placebo before open aneurysm repair. MMP-9 levels in excised aneurysm tissue were decreased in the simvastatin group. In 1 observational study of 130 patients followed up for 2 years, no aneurysm expansion was observed in the 75 patients taking statins, whereas the mean aneurysm size in the group not taking statins increased from 4.5 to 5.3 cm.38 Schouten et al39 monitored 150 patients for a minimum of 12 months with at least 3 measurements. Aneurysm expansion rate was decreased in the patients who were taking statins (2.0 mm per year) compared with those not taking statins (3.6 mm per year).39

Although these associative data are intriguing, there are many potential biases in these uncontrolled observational studies. They are reminiscent of similar analyses that suggested that β-blockers would inhibit aneurysm expansion, whereas randomized clinical trials showed propranolol to be ineffective. Because AAA, coronary artery disease, and peripheral vascular disease share common risk factors, such as tobacco use, there will be clear indications for statin use in many AAA patients related to coronary artery disease and peripheral vascular disease. The use of statins will become more common with efforts to meet the National Heart, Lung, and Blood Institute’s increasingly stringent adult treatment protocol guidelines. A high prevalence of statin use among
AAA patients will make it challenging to design trials to assess the specific role of statin therapy as an inhibitor of aneurysm expansion. Such studies will be important, however, because some guidelines such as those for the Women’s Health Initiative have made the leap to categorizing AAA as a peripheral vascular disease equivalent. At the present time, there does not appear to be sufficient evidence to recommend that statin therapy be initiated for the diagnosis of AAA alone.

β-Blockers

Several animal studies have indicated that propranolol might have beneficial effects on aneurysmal disease on the basis of both its hemodynamic properties and its biochemical effects on matrix proteins. Two clinical studies used retrospective analysis to assess the impact of β-blockers in aneurysm growth rates. Both identified a significant inhibitory effect of β-blockers. These studies provided the underpinning for 2 multicenter randomized trials that tested propranolol in aneurysm patients. Propranolol did not inhibit aneurysm expansion in a trial reported by Lindholt et al. These results were compromised by low compliance in the propranolol arm, because only 22% of patients continued the medication for 2 years. The mean growth rate was slightly (but not significantly) higher in the propranolol group. A Canadian trial that recruited 552 patients suffered similarly from compliance problems in that 42% of propranolol-treated participants discontinued the drug during the trial. The growth rate in the placebo group and in the propranolol group did not differ, although there was a slight trend in favor of propranolol. Quality of life, assessed by the short-form 36-item (SF-36) questionnaire, showed that propranolol had a significant negative effect, as one would anticipate from the low compliance rate.

Angiotensin-Converting Enzyme Inhibitors and Angiotensin Receptor Blockers

Angiotensin-converting enzyme (ACE) inhibitors have been shown to both stimulate and inhibit MMPs depending on cell type or animal model. Losartan does not appear to have a direct effect on MMPs. A number of animal experiments using different models of aneurysmal disease have suggested an important role for the angiotensin/rexin axis in aneurysm development. Captopril but not losartan, an angiotensin receptor blocker, prevents aneurysm formation in the rat elastase model of AAA. This model relies on infusion of elastase into the infrarenal aorta, which results in initial mechanical dilation followed by progressive enlargement. Another commonly studied aneurysm model is based on chronic infusion of angiotensin II into apolipoprotein E-deficient mice, which results initially in midaortic dilation and eventual rupture. Losartan prevents aneurysm formation in this model. This effect of losartan is consistent with observations in genetically engineered mice with Marfan syndrome. Work done in these mice has suggested that the inability of mutated fibrillin to sequester transforming growth factor-β plays a role in the progression of tissue changes associated with Marfan syndrome. In a series of studies, transforming growth factor-β antagonism by losartan was effective in preventing progressive matrix degradation. The reason for the discrepant effects of losartan—ineffective in the elastase aneurysm model and effective in the angiotensin and Marfan models—may relate to differences among the models. In the angiotensin infusion model, initial dissection of the upper abdominal aorta is followed by dilation. This process may have more similarities to the Marfan syndrome models, in which the thoracic aorta is affected. Clinical trials of losartan in Marfan syndrome have recently begun enrollment.

Hackam et al recently published results of an analysis of a linked administrative database from Ontario, Canada analyzing ruptured (n = 3379) and nonruptured (n = 11 947) aortic aneurysms from 1992 to 2002. ACE inhibitor use within the prior 3 to 12 months was less frequent among those admitted for aneurysm rupture (OR 0.82, CI 0.74 to 0.90). β-Blockers, lipid-lowering agents, and angiotensin receptor blockers showed no relationship to rupture. In a published response to that article, Lederle and Taylor noted that among those patients who discontinued ACE inhibitors within the past 3 to 12 months, there was a harmful effect in favor of aneurysm rupture. The case-control study by Schouten et al and post hoc analysis of the UK aneurysm trial data did not find a relationship between ACE inhibitors and aneurysm expansion rates. Most patients presenting with aneurysm rupture have large, undetected aneurysms, whereas patients with known aneurysms typically undergo repair long before their rupture risk becomes significant. Thus, this information regarding ACE inhibitors and rupture risk might find its most practical application among the small number of patients deemed unfit for repair.

Macrolides

A number of antibiotics have been proposed as a treatment for AAA with varying rationales. One line of reasoning is that AAA progression is enhanced by secondary infection within the aortic wall. Chlamydia pneumoniae has been found in atherosclerotic plaque and the wall of AAAs. There was once great enthusiasm for the hypothesis that treatment of the secondary chlamydial infection could slow progression of atherosclerosis. This has been diminished by subsequent prospective randomized trials that showed no cardiovascular benefit of a year of a treatment with azithromycin in patients with stable coronary artery disease. Similar negative results were found by Burkhardt et al. A small study by Lindholt et al suggested that serological evidence of a C pneumoniae infection was associated with an increased rate of aneurysm expansion. This led to a randomized clinical trial in which 43 patients received a 1-month course of roxithromycin, whereas 49 patients received placebo. Patients in the treatment arm had an expansion rate at the end of the study of 1.56 mm per year compared with a rate of 2.75 mm per year in the placebo-treated group. The inhibition was greater in the first year than the second year. The study did not clarify the mechanism of effect because there was no correlation between Chlamydia titers and roxithromycin ability to inhibit aneurysm expansion.
Tetracyclines
The tetracycline antibiotics have been studied because of their known inhibition of MMPs. Petrič et al\textsuperscript{61} were the first to demonstrate that doxycycline could suppress aortic wall MMP activity, elastin degradation, and aneurysm development in the elastase-induced rat model. They achieved similar results using nonantimicrobial (chemically modified) tetracyclines and nonselective hydroxamic acid derivatives as MMP inhibitors, which indicates that the aneurysm-suppressing effects of doxycycline are most likely related to its activity as an MMP inhibitor.\textsuperscript{62} Longo et al\textsuperscript{63} characterized a second murine aneurysm model using calcium chloride applied to the abluminal surface to induce the aneurysm. In this model, doxycycline demonstrates the same dose-dependent inhibition of aneurysm expansion.\textsuperscript{64} The plasma doxycycline levels achieved in these animal studies were in the same range as those seen in AAA patients receiving doxycycline (100 mg BID).\textsuperscript{65} These murine studies suggest that inhibition can still be achieved at plasma levels in the 1- to 2-μg/mL range.\textsuperscript{66}

A number of studies in patients have suggested that doxycycline can inhibit MMPs in aneurysm tissue. Curci et al\textsuperscript{66} treated a series of patients with a 3-week course of doxycycline before open aneurysm repair. Tissue levels of MMP-9 were significantly reduced by doxycycline compared with untreated patients. Baxter et al\textsuperscript{67} showed in a small series of 36 patients on a 6-month course of doxycycline that plasma MMP-9 levels decreased significantly compared with baseline levels. This work has been followed by a small, prospective, randomized trial of doxycycline in which 32 patients were randomized, with 17 receiving doxycycline (150 mg/d) for 3 months. Patients were followed up for 18 months.\textsuperscript{67} C pneumoniae titers were assessed but found not to be affected by doxycycline treatment. The calculated growth rate at the end of the 18-month period of observation was 1.5 mm per year in the doxycycline-treated group versus 3.0 mm per year in the placebo-treated group. This difference did not achieve statistical significance, but the 6- and 12-month time periods did show a significant difference in favor of doxycycline treatment. Level B evidence (from small randomized trials) suggests that roxithromycin or doxycycline will decrease the rate of aneurysm expansion.

Considerations for Evaluating Medical Therapies
There are 3 important features of AAA that lend themselves to medical treatment: (1) Inexpensive and accurate methods for detection; (2) long period of surveillance before intervention; and (3) life expectancy of the affected population. Increased public awareness and the availability of screening will lead to increased aneurysm detection in the next decade. Ninety percent of aneurysms detected at screening are below the threshold for immediate repair, and aneurysm expansion is gradual. A reduction of the expansion rate of a 4.0-cm AAA by 50% potentially increases the time before surgical intervention is required to >10 years, which exceeds the life expectancy of many aneurysm patients. The current standard of care for these small AAAs is “watchful waiting.” The provision of a relatively benign and efficacious medical therapy to these patients may reverse the diminished quality of life associated with detection of a potentially life-threatening condition for which no immediate treatment is offered. In the format of the American College of Cardiology/American Heart Association clinical practice guidelines,\textsuperscript{68} level A evidence (from large randomized trials) is available to indicate that observation of aneurysms in men is safe up to a size of 5.5 cm and that propranolol does not inhibit aneurysm expansion (Table).\textsuperscript{69}

### Sources of Funding
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### Disclosures
None.

### References

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<table>
<thead>
<tr>
<th>Intervention</th>
<th>Reference(s)</th>
<th>Effect on AAA Growth</th>
<th>Level of Evidence</th>
<th>Class of Recommendation</th>
</tr>
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<tbody>
<tr>
<td>Propranolol</td>
<td>46, 69</td>
<td>No inhibition</td>
<td>A</td>
<td>III</td>
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<td>Macrolides</td>
<td>60</td>
<td>Inhibition</td>
<td>B</td>
<td>IIA</td>
</tr>
<tr>
<td>Tetracycline*</td>
<td>67</td>
<td>Inhibition</td>
<td>B</td>
<td>IIA</td>
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<tr>
<td>Statins</td>
<td>38, 39</td>
<td>Inhibition</td>
<td>B and C</td>
<td>IIb</td>
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<td>ACE inhibitors</td>
<td></td>
<td>No inhibition</td>
<td>B and C</td>
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<td>AR blockers</td>
<td>48, 50</td>
<td>Animal data</td>
<td>C</td>
<td>IIb</td>
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</table>

*Inhibition at 6 and 12 months after 3 months of treatment.
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The impact of exposure technique on perioperative complications in patients undergoing elective open abdominal aortic aneurysm repair

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Sara L. Zettervall, MD, Marc L. Schermerhorn, MD, and Fred A. Weaver, MD, MMM, Austin Tex; Los Angeles and Loma Linda, Calif; and Boston, Mass

Objective: The objective of this study was to evaluate the impact of exposure technique on perioperative complications in patients undergoing elective open abdominal aortic aneurysm (AAA) repair.

Methods: Using the Society for Vascular Surgery Vascular Quality Initiative database, the study identified patients subjected to open AAA repair from January 2003 to July 2014 and divided them into two aortic exposure groups, retroperitoneal (RP) and transperitoneal (TP). Multivariable analysis was performed to compare the incidence of cardiac events (myocardial infarction, dysrhythmia, heart failure), prolonged intubation, renal dysfunction, and mortality, adjusting for between-group differences identified on univariate analysis.

Results: Open AAA repair was performed in 3530 patients, using RP in 26% and TP in 74%. The RP group had a higher rate of suprarenal aortic clamp (60.9% vs 30.2%; \( P < .001 \)), higher proportion of high-risk patients as stratified by the Vascular Study Group of New England Cardiac Risk Index (25.6% vs 22.2%; \( P = .038 \)), and lower rate of iliac aneurysms (18.0% vs 31.2%; \( P < .001 \)). After multivariable analysis, RP was associated with a lower incidence of cardiac events (12.2% vs 16.0%; adjusted odds ratio, 0.60; 95% confidence interval, 0.41-0.88; \( P = .009 \)) and renal dysfunction (13.3% vs 16.5%; adjusted odds ratio, 0.65; 95% confidence interval, 0.46-0.97; \( P = .001 \)). No difference in respiratory complications or mortality was identified.

Conclusions: Despite increased utilization of suprarenal aortic clamp during elective open AAA repair, the RP technique was associated with a lower risk-adjusted incidence of cardiac and renal complications compared with the TP technique. (J Vasc Surg 2016;63:1141-6.)

Since the initial description of the retroperitoneal (RP) approach to the abdominal aorta and the publication of an early series, the adoption of this approach has been variable, and the results compared with the transperitoneal (TP) approach have been conflicting. A recent meta-analysis summarizing the currently available data demonstrated that patients undergoing open aortic surgery through an RP approach had significantly lower rates of postoperative ileus, pneumonia, intensive care unit (ICU) stay, total hospital stay, and cost. Most of the literature investigating this issue predates the advent of endovascular techniques. With advances in endovascular techniques for the treatment of occlusive and aneurysmal aortoiliac disease, the open approach has been progressively reserved for a select group of patients with challenging anatomy who require a more extensive aortic procedure and a suprarenal aortic cross-clamp. The RP approach has been proposed as particularly advantageous for patients with complex anatomy, and an increase in the use of the RP approach compared with the TP exposure has occurred with the increased use of endovascular aortic aneurysm repair.

The open aortic aneurysm repair registry of the Society for Vascular Surgery Vascular Quality Initiative (VQI) captures the method of aortic exposure as well as other key preoperative, perioperative, and postoperative variables. The objective of this study was to evaluate the impact of exposure technique on perioperative complications in patients undergoing elective open abdominal aortic aneurysm (AAA) repair using data from the VQI.

METHODS

This study was approved by the Society for Vascular Surgery’s Patient Safety Organization Research Advisory Subcommittee. The open abdominal aortic registry of the VQI database was then queried for the period from January 2003 to July 2014. Data compiled under the auspices of the
Society for Vascular Surgery’s Patient Safety Organization are in compliance with the Patient Safety Act, which allows patient data to be captured within a quality improvement framework, foregoing the requirement of Institutional Review Board approval or patient consent. Nonidentifiable data can also be used by the participating centers for quality improvement projects and outcomes research. 

All patients who underwent open AAA repair in the VQI database were identified. Patients who underwent emergent aortic aneurysm repair were excluded from the analysis. The study population was then divided into two study groups according to the technique used for the exposure of the abdominal aorta (RP vs TP).

The primary outcome measures of the study were the incidence of cardiac events, defined as the composite outcome of myocardial infarction, dysrhythmia, or heart failure, and respiratory complications, defined as the composite outcome of postoperative pneumonia or respiratory failure (the latter defined as respiratory insufficiency requiring ventilator support). Secondary outcomes included mortality, renal insufficiency, hospital length of stay, and ICU length of stay.

The majority of the variables in the database were dichotomous. Continuous variables were dichotomized using clinically relevant cutoffs. Patients at high risk of cardiac complications according to previously published Vascular Society of New England Cardiac Risk Index (VSG-CRI) criteria were identified, and a new variable was created to identify those patients. The VSG-CRI is a validated scoring system capable of accurately predicting cardiac complications. The composite nature of this index allows an aggregate numbered score that summarizes several areas of the patient’s underlying comorbid condition, which may in tandem result in worse outcomes. The VSG-CRI score was calculated for each of the patients in the study using variables available in the database. For this calculation, each patient received a predefined number of points for each of the following variables: age, smoking, insulin-dependent diabetes, coronary artery disease, congestive heart failure, prior coronary artery bypass graft or percutaneous coronary intervention, long-term beta-blocker treatment, chronic obstructive pulmonary disease, and creatinine concentration >1.8 mg/dL.

Statistical comparison of the two study groups was then performed using univariate analysis with \( \chi^2 \) or Fisher exact test for proportions and t-test or Mann-Whitney U test for comparison of means to identify significant differences in baseline characteristics.

To derive outcome comparison between the study groups adjusting for differences in baseline characteristics, a multivariable analysis was performed for each of the outcomes of interest, including the exposure technique as the covariate of interest in the model and forcing the entrance of all the baseline factors that were significantly different between the study groups at \( P < .05 \) level.

Adjusted outcome comparisons for hospital length of stay and ICU length of stay were derived from general linear model logistic regression with the continuous outcome as the dependent variable and the exposure technique and all the baseline factors that were significantly different on univariate analysis as the independent variables. Adjusted mean differences with 95% confidence interval (CI) and adjusted \( P \) values were derived from the equation.

IBM SPSS Statistics (version 20; IBM Corp., Armonk, NY) was used for all the statistical analysis. A \( P \) value < .05 was considered statistically significant.

**RESULTS**

The VQI database from January 2003 to July 2014 had 3530 patients undergoing open AAA repairs who met the study inclusion criteria. RP technique for exposure of the aorta was used in 26% (919) of the patients and TP in 74% (2611).

The data set included data from 16 VQI regions, 148 centers, and 509 surgeons. The distribution of cases across the centers was skewed, with a median of nine cases performed per center (interquartile range, 2.3-25). RP exposure technique was used in all participating regions, but a wide variation in exposure technique utilization per region was identified, with a 2% to 59% rate of RP exposure use. One of the regions provided 55% of all cases in the database.

The rate of RP use for that region was 25%, which was lower than the 28% rate of RP for the remainder of the regions; however, this difference was not statistically significant (\( P = .75 \)).

Comparison of the two study groups demonstrated several significant differences, which are detailed in Table 1. A significantly higher proportion of patients with a high risk of cardiac complications according to the VSG-CRI was found in the RP group (26% vs 22%; \( P = .038 \)). Patients in the RP group were also significantly more likely to undergo placement of a suprarenal aortic clamp (61% vs 30%; \( P < .001 \)) and required increased blood transfusion of 3 units or more of packed red blood cells (22% vs 12%; \( P < .001 \)). Patients in the TP group were significantly more likely to have iliac artery aneurysmal involvement (31% vs 18%; \( P < .001 \)). For the patients with an associated iliac artery aneurysm, the size of the aneurysm was not significantly different between the study groups (32.6 mm vs 33.3 mm; \( P = .203 \)). Overall, 18% of the study population required both a suprarenal clamp and a bifurcated graft, a combination that suggests additional technical challenge. The proportion of those complex cases was significantly higher in the RP group compared with the TP group (23% vs 17%; \( P < .0001 \)).

The overall in-hospital mortality for the study population was 2.7%, with no crude or adjusted mortality difference between the two exposure techniques. Table II describes crude outcome comparison between the study groups. Regarding the respiratory outcomes, patients in the RP group had a significantly higher incidence of ventilatory support requirement longer than 24 hours compared with the TP group (10% vs 5%; odds ratio [OR], 2.09; 95% CI, 1.58-2.75; \( P < .001 \)); however this difference lost statistical significance after adjustment for between-group differences.
Table I. Comparison of baseline characteristics

<table>
<thead>
<tr>
<th></th>
<th>Total (N = 3530)</th>
<th>RP (n = 919; 26%)</th>
<th>TP (n = 2611; 74%)</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sex, female</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Race, white</td>
<td>27.2 (960)</td>
<td>30.9 (284)</td>
<td>25.9 (676)</td>
<td>.003</td>
</tr>
<tr>
<td>Age &gt;65 years</td>
<td>93.8 (3311)</td>
<td>94.3 (867)</td>
<td>93.6 (2444)</td>
<td>.447</td>
</tr>
<tr>
<td>Transferred from another hospital or rehabilitation facility</td>
<td>69.9 (2466)</td>
<td>73.6 (676)</td>
<td>68.6 (1790)</td>
<td>.004</td>
</tr>
<tr>
<td>Smoking history</td>
<td>3.3 (115)</td>
<td>3.5 (32)</td>
<td>3.2 (83)</td>
<td>.649</td>
</tr>
<tr>
<td>Hypertension</td>
<td>91.8 (3238)</td>
<td>91.5 (841)</td>
<td>91.9 (2397)</td>
<td>.681</td>
</tr>
<tr>
<td>Diabetes</td>
<td>83.7 (2952)</td>
<td>85.3 (784)</td>
<td>83.1 (2168)</td>
<td>.124</td>
</tr>
<tr>
<td>Diabetes on insulin</td>
<td>14.9 (524)</td>
<td>14.1 (129)</td>
<td>15.2 (395)</td>
<td>.425</td>
</tr>
<tr>
<td>Antibiotic cephalosporin</td>
<td>2.2 (77)</td>
<td>2.5 (23)</td>
<td>2.1 (54)</td>
<td>.437</td>
</tr>
<tr>
<td>Antibiotics longer than 24 hours</td>
<td>6.8 (239)</td>
<td>7.6 (70)</td>
<td>6.5 (169)</td>
<td>.235</td>
</tr>
<tr>
<td>BMI ≥35</td>
<td>33.2 (1170)</td>
<td>34.4 (316)</td>
<td>32.8 (884)</td>
<td>.357</td>
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<tr>
<td>Elevated creatinine (&gt;1.78 mg/dL)</td>
<td>0.7 (24)</td>
<td>0.9 (8)</td>
<td>0.6 (16)</td>
<td>.415</td>
</tr>
<tr>
<td>Nonobturator</td>
<td>5.9 (207)</td>
<td>5.6 (51)</td>
<td>6.1 (156)</td>
<td>.640</td>
</tr>
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<td>ASA class II or higher</td>
<td>92.4 (3311)</td>
<td>91.3 (661)</td>
<td>92.9 (1739)</td>
<td>.167</td>
</tr>
<tr>
<td>Hemoglobin &lt;10 mg/dL</td>
<td>3.6 (98)</td>
<td>3.9 (29)</td>
<td>3.5 (69)</td>
<td>.627</td>
</tr>
<tr>
<td>Prior bypass</td>
<td>3.4 (120)</td>
<td>2.6 (24)</td>
<td>3.7 (96)</td>
<td>.124</td>
</tr>
<tr>
<td>Prior CEA or CAS</td>
<td>6.7 (237)</td>
<td>6.6 (61)</td>
<td>6.7 (176)</td>
<td>.908</td>
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<td>Prior aneurysm repair</td>
<td>1.0 (37)</td>
<td>0.8 (7)</td>
<td>1.2 (30)</td>
<td>.320</td>
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<tr>
<td>Prior PVI</td>
<td>4.7 (166)</td>
<td>4.1 (38)</td>
<td>4.9 (128)</td>
<td>.341</td>
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<td>Preoperative aspirin</td>
<td>69.7 (2461)</td>
<td>69.5 (1824)</td>
<td>69.9 (1834)</td>
<td>.788</td>
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<tr>
<td>P2Y12 antagonists</td>
<td>7.5 (263)</td>
<td>9.1 (84)</td>
<td>6.9 (179)</td>
<td>.024</td>
</tr>
<tr>
<td>Preoperative antiplatelet agent (any)</td>
<td>71.3 (2515)</td>
<td>71.2 (654)</td>
<td>71.4 (1846)</td>
<td>.911</td>
</tr>
<tr>
<td>Intraoperative cold renal perfusion</td>
<td>70.1 (2473)</td>
<td>71.5 (657)</td>
<td>69.6 (1816)</td>
<td>.283</td>
</tr>
<tr>
<td>Beta blocker</td>
<td>73.0 (2576)</td>
<td>71.1 (653)</td>
<td>73.7 (1923)</td>
<td>.119</td>
</tr>
<tr>
<td>High VSG-CRI</td>
<td>23.1 (801)</td>
<td>25.6 (231)</td>
<td>22.2 (570)</td>
<td>.038</td>
</tr>
<tr>
<td>Family history of AAA</td>
<td>13.5 (461)</td>
<td>13.8 (124)</td>
<td>13.4 (337)</td>
<td>.725</td>
</tr>
<tr>
<td>AAA size, mm</td>
<td>58.6 ± 15.9</td>
<td>59.3 ± 14.0</td>
<td>58.4 ± 16.2</td>
<td>.128</td>
</tr>
<tr>
<td>Graft diameter, mm</td>
<td>18.4 ± 2.8</td>
<td>18.7 ± 2.9</td>
<td>18.3 ± 2.9</td>
<td>.005</td>
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<td>Iliac aneurysm</td>
<td>27.8 (977)</td>
<td>18.0 (165)</td>
<td>31.2 (812)</td>
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<td>Epidual anesthesia</td>
<td>55.7 (1966)</td>
<td>58.2 (535)</td>
<td>54.8 (1431)</td>
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</tr>
<tr>
<td>Tube graft</td>
<td>48.5 (1705)</td>
<td>63.3 (577)</td>
<td>43.4 (1128)</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>Supraaortic clamp</td>
<td>38.2 (1336)</td>
<td>60.9 (554)</td>
<td>30.2 (782)</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>Heparin</td>
<td>98.8 (3480)</td>
<td>99.1 (910)</td>
<td>98.7 (2570)</td>
<td>.263</td>
</tr>
<tr>
<td>Intraoperative cold renal perfusion</td>
<td>6.5 (227)</td>
<td>16.3 (148)</td>
<td>3.0 (79)</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>Mannitol</td>
<td>53.4 (1879)</td>
<td>60.8 (556)</td>
<td>50.8 (1323)</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>Chlorhexidine-based preparation</td>
<td>78.6 (1856)</td>
<td>77.3 (519)</td>
<td>79.1 (1337)</td>
<td>.359</td>
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<tr>
<td>Concomitant thromboembolocytome</td>
<td>5.6 (152)</td>
<td>5.4 (41)</td>
<td>5.7 (111)</td>
<td>.754</td>
</tr>
<tr>
<td>Renal bypass</td>
<td>8.3 (225)</td>
<td>16.2 (123)</td>
<td>5.3 (102)</td>
<td>&lt;.001</td>
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<tr>
<td>Infringuinal bypass</td>
<td>2.8 (76)</td>
<td>2.9 (22)</td>
<td>2.8 (54)</td>
<td>.861</td>
</tr>
<tr>
<td>Other intra-abdominal procedure</td>
<td>11.5 (311)</td>
<td>9.8 (74)</td>
<td>12.2 (237)</td>
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</tr>
<tr>
<td>Antibiotics not within 1 hour</td>
<td>1.5 (38)</td>
<td>1.7 (12)</td>
<td>1.4 (26)</td>
<td>.607</td>
</tr>
<tr>
<td>Antibiotics longer than 24 hours</td>
<td>5.6 (144)</td>
<td>7.1 (51)</td>
<td>5.0 (93)</td>
<td>.039</td>
</tr>
<tr>
<td>Antibiotic cephalosporin</td>
<td>89.3 (2315)</td>
<td>89.1 (644)</td>
<td>89.5 (1671)</td>
<td>.778</td>
</tr>
<tr>
<td>Visceral ischemia, minutes</td>
<td>13.6 ± 23.7</td>
<td>21.6 ± 24.2</td>
<td>10.9 ± 22.9</td>
<td>&lt;.001</td>
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<tr>
<td>Net EBL, L</td>
<td>1.52 ± 1.29</td>
<td>1.66 ± 1.31</td>
<td>1.51 ± 1.44</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>Net EBL, L</td>
<td>0.82 ± 0.91</td>
<td>0.88 ± 0.89</td>
<td>0.82 ± 0.99</td>
<td>.002</td>
</tr>
<tr>
<td>Crystalloids, L</td>
<td>4.55 ± 2.19</td>
<td>4.41 ± 2.47</td>
<td>4.68 ± 2.44</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>Transfusion ≥3 units PRBCs</td>
<td>1.5 ± 0.97</td>
<td>1.64 ± 3.05</td>
<td>0.95 ± 2.42</td>
<td>&lt;.007</td>
</tr>
</tbody>
</table>

**Note:** with multivariate analysis (adjusted OR, 1.15; 95% CI, 0.74-1.79; P = .531). No difference was identified in the incidence of pneumonia or respiratory failure.

For the renal outcomes, although the incidence of renal dysfunction was significantly higher for the RP group on univariate analysis (17% vs 13%; OR, 1.29; 95% CI,
DISCUSSION

This VQI study demonstrated that the use of an RP exposure technique for patients undergoing elective AAA repair was associated with lower incidence of cardiac events and renal dysfunction and a shorter ICU admission.
The TP approach remains the exposure modality of choice for the majority of surgeons. In this VQI study including data from 16 distinct regions with 509 contributing surgeons, three quarters of the aneurysm repairs were performed through a TP approach. Reasons for the lower rate of adoption of the RP technique include a lack of clear benefit and a perceived higher technical difficulty of this exposure compared with the TP approach, which seems to be more familiar to most vascular surgeons. Although it provides excellent exposure to the abdominal aorta, the RP exposure has a steeper learning curve, making it less acceptable to surgeons already comfortable with the TP technique. Proponents of the RP technique highlight the benefits of not violating the peritoneal cavity, avoiding a hostile abdomen,9,10,14 and providing easier exposure in obese patients.9,10

Multiple observational studies and randomized trials have been performed to date comparing these two aortic exposure techniques with heterogeneous and conflicting results. Earlier observational findings of a substantial decrease in pulmonary complications1,2 were not corroborated by later randomized trials.8,9 Available evidence comparing these two aortic exposure techniques has recently been summarized in a systematic review and meta-analysis.8 That meta-analysis identified that RP was associated with a significantly lower incidence of postoperative ileus, which was an outcome not captured by the VQI and therefore not evaluated in the present study. Contrary to the findings from the meta-analysis, which did not demonstrate a difference in the incidence of myocardial infarction or renal failure, both the incidence of cardiac complications and renal dysfunction in the present study were found to be significantly higher for patients in the TP technique group. Twine et al also found a decreased risk of postoperative pneumonia associated with the RP technique, a finding not replicated in this study. Similar to our findings, although patients in the RP group had a significantly lower ICU length of stay, the choice of exposure technique had no impact on mortality.8

The majority of the studies investigating these two exposure techniques precede the wide adoption of endovascular strategies for the treatment of AAAs. After the endovascular revolution, patients currently undergoing open aortic surgery are expected to be significantly different from those included in the original studies as open surgery has increasingly been reserved for unfavorable necks and challenging aortic anatomy. In fact, in the original series describing the anterolateral RP approach to the abdominal aorta, this exposure technique was applied to 500 patients with low-risk infrarenal aortic and iliac artery disease. The RP approach, particularly if it is modified to include a higher incision extending over the ninth or tenth rib, has been increasingly used for anatomically challenging cases with low rates of morbidity and mortality,1,2 and it has been considered the technique of choice to expose the juxtarenal and suprarenal aorta.9,14 Although no objective data exist to support this statement that the RP technique provides better exposure to the proximal abdominal aorta, the finding of a twofold higher proportion of patients requiring a suprarenal clamp in the RP group suggests that surgeons across the VQI participating centers tend to use this technique as their exposure of choice for aortic aneurysm repairs requiring high clamp placement.

The reasons for the adjusted higher incidence of cardiac complications despite a lower proportion of high-risk VSG-CRI patients identified in the TP group are not clear and cannot be derived from these data. It has been previously demonstrated that the TP approach has a measurable negative physiologic repercussion resulting in increased myocardial oxygen consumption, theoretically secondary to bowel manipulation and mesenteric traction.15 Another possible explanation for this finding would be related to postoperative fluid shifts. Avoidance of peritoneal violation and intestinal manipulation with the RP approach would result in significantly less bowel edema and postoperative fluid shifts. As a consequence, less intense third-space fluid mobilization would ensue as the patient recovers postoperatively, with fewer hemodynamic disturbances and minimization of intravascular volume overload. The patients in the RP group also had a significantly higher incidence of associated iliac artery aneurysms, which is a factor that notably adds significant operative technical difficulty to the case and is potentially associated with an increased incidence of morbidity and perioperative complications. The presence of associated iliac aneurysms, however, was included in the logistic regression model in an attempt to minimize the independent impact of this factor on the adjusted outcome comparison between the two study groups.

As a result of how the VQI historically evolved from the original Vascular Study Group of New England that pioneered this quality initiative, an imbalanced case contribution to the database exists and could have implications on the findings of this study. Although one single region disproportionately contributed to the overall number of cases in the study, the weight of that one region was balanced between the two study groups, therefore minimizing the potential confounder of a high-volume region on the outcomes.

This population database study has a number of limitations, most of them inherent in this type of data set. The retrospective nonrandomized design carries a significant potential for selection bias. It is plausible that this bias may be intensified by the intrinsic characteristics of the VQI, which is structured as a self-reporting platform with multiple contributing centers, possibly with different understanding of the selected data points.

CONCLUSIONS

In the VQI, the TP approach was used in 75% of aortic aneurysm repairs, with the RP technique being more common in patients with increased cardiac risk and patients who required a suprarenal aortic clamp. Despite increased risk profile of patients, the RP technique was associated with a lower incidence of risk-adjusted cardiac and renal...
complications compared with the TP technique. These findings suggest that in the endovascular age, the technique of RP aortic exposure should be mastered by all vascular surgeons and used to particular advantage in patients requiring complex juxtarenal and pararenal aortic reconstructions.

AUTHOR CONTRIBUTIONS
Conception and design: PT, KW, AA, SZ, MS, FW
Analysis and interpretation: PT, KW, FW
Data collection: PT, KW
Writing the article: PT, FW
Critical revision of the article: PT, KW, AA, SZ, MS, FW
Final approval of the article: PT, KW, AA, SZ, MS, FW
Statistical analysis: PT
Obtained funding: Not applicable
Overall responsibility: PT

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Two-Year Outcomes after Conventional or Endovascular Repair of Abdominal Aortic Aneurysms

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*The members of the DREAM Trial Group are listed in the Appendix.


ABSTRACT

BACKGROUND

Two randomized trials have shown better outcomes with elective endovascular repair of abdominal aortic aneurysms than with conventional open repair in the first month after the procedure. We investigated whether this advantage is sustained beyond the perioperative period.

METHODS

We conducted a multicenter, randomized trial comparing open repair with endovascular repair in 351 patients who had received a diagnosis of abdominal aortic aneurysm of at least 5 cm in diameter and who were considered suitable candidates for both techniques. Survival after randomization was calculated with the use of Kaplan–Meier analysis and compared with the use of the log-rank test on an intention-to-treat basis.

RESULTS

Two years after randomization, the cumulative survival rates were 89.6 percent for open repair and 89.7 percent for endovascular repair (difference, −0.1 percentage point; 95 percent confidence interval, −6.8 to 6.7 percentage points). The cumulative rates of aneurysm-related death were 5.7 percent for open repair and 2.1 percent for endovascular repair (difference, 3.7 percentage points; 95 percent confidence interval, −0.5 to 7.9 percentage points). This advantage of endovascular repair over open repair was entirely accounted for by events occurring in the perioperative period, with no significant difference in subsequent aneurysm-related mortality. The rate of survival free of moderate or severe complications was also similar in the two groups at two years (at 65.9 percent for open repair and 65.6 percent for endovascular repair; difference, 0.3 percentage point; 95 percent confidence interval, −10.0 to 10.6 percentage points).

CONCLUSIONS

The perioperative survival advantage with endovascular repair as compared with open repair is not sustained after the first postoperative year.
Two randomized trials have demonstrated better outcomes with elective endovascular repair of abdominal aortic aneurysms than with conventional open repair in the first month after the procedure.\textsuperscript{1,2} The reported in-hospital mortality rates in these two trials were 4.6 percent and 6.0 percent for open repair and 1.6 percent and 1.2 percent for endovascular repair, respectively. Although the relevance of a reduction in perioperative risk should not be underestimated from the patient’s perspective, the improvement in early survival with the use of a less invasive technique is not surprising.\textsuperscript{3} Consequently, both reports stressed the need for longer-term data before a decision could be reached about which therapy is better in patients who are suitable candidates for either procedure.

Findings in uncontrolled long-term studies of endovascular aneurysm repair have suggested that the early advantage of endovascular over open repair may not persist over time.\textsuperscript{4,5} Endovascular repair appeared to be associated with higher rates of reintervention and complications as well as a continued risk of aneurysm rupture. The Dutch Randomized Endovascular Aneurysm Management (DREAM) trial was conducted to assess the rates of death from any cause and complications in a multicenter, randomized trial comparing elective open and endovascular aneurysm repair.

METHODS

STUDY DESIGN AND PATIENTS

The design and methods of the trial have been described in detail elsewhere.\textsuperscript{2,6} In brief, patients referred to surgery clinics at 26 centers in the Netherlands and 4 centers in Belgium who had received a diagnosis of an abdominal aortic aneurysm of at least 5 cm in diameter and who were considered suitable candidates for both techniques were randomly assigned to undergo open or endovascular repair after giving written informed consent. Randomization was carried out centrally with the use of a computer-generated permuted-block sequence and stratified according to study center in blocks of four patients.

The study was performed according to the principles of the Declaration of Helsinki. The institutional review boards of all participating hospitals approved the protocol. The corresponding author assumed full responsibility for the conduct of the trial, had full access to all the data, and controlled the decision to publish. The study was publicly funded, and the sponsor had no role in the study design.

DATA COLLECTION AND FOLLOW-UP

All data were submitted to the trial-coordination center (Julius Center for Health Sciences and Primary Care, University Medical Center, Utrecht, the Netherlands). Follow-up visits were scheduled 30 days and 6, 12, 18, and 24 months after the procedure. Before hospital discharge and at each follow-up visit, all patients underwent a physical examination, which included calculation of the ankle–brachial blood-pressure index; abdominal helical computed tomographic angiography; and abdominal color duplex ultrasonography. In addition, patients in the endovascular group underwent plain abdominal radiography before hospital discharge and 12 and 24 months postoperatively.

Data acquisition was stopped on March 1, 2005, for this report. For all analyses, data on patients were censored after their last follow-up visit. For the crude survival analysis, however, reports on vital status obtained at any time before the cutoff date were also incorporated.

END POINTS

The primary end point of the trial was a composite of operative mortality and moderate or severe complications, as discussed in the initial report on the results of the trial.\textsuperscript{2} Mortality and complications at two years were predetermined secondary end points in the original trial design. The outcome events that we analyzed were deaths from all causes, aneurysm-related deaths, complications, and reinterventions.

The cause and exact date of death were determined by assessment of death certificates and by contacting the physicians involved (surgeons and general practitioners) and patients’ relatives if necessary. Aneurysm-related death was defined as death resulting from aneurysm rupture, graft infection, or thrombosis; any death occurring within 30 days after the original procedure or a reintervention; or any death occurring more than 30 days after the original procedure or a reintervention but during the same admission.

Complications were classified and graded according to the reporting standards of the Ad Hoc Committee for Standardized Reporting Practices in Vascular Surgery of the Society for Vascular Surgery/International Society for Cardiovascular Surgery.\textsuperscript{7,8} Three severity grades (mild, moderate, and
adjudication committee, consisting of five vascular surgeons, assessed the type and severity of each complication and reintervention in a blinded fashion and independently from each other. Disagreements were resolved in a plenary consensus meeting.

**Statistical Analysis**

All data were analyzed according to the intention-to-treat principle. Kaplan–Meier analysis was used to analyze survival and other end points, and differences between groups were compared with the use of the log-rank test. Cox proportional-hazards regression was used to estimate hazard ratios for the analysis of reintervention rates. Means (±SD) were used to describe continuous variables. Differences between groups were compared with the use of the Mann–Whitney U test for continuous variables and Fisher’s exact test for proportions. All reported P values are two-sided and are not adjusted for multiple testing.

**Results**

**Characteristics of the Patients and Treatment Assignments**

Between November 2000 and December 2003, 178 patients were randomly assigned to undergo open repair and 173 to undergo endovascular repair. Six patients did not undergo aneurysm repair after randomization: four declined treatment (three assigned to open repair and one to endovascular repair), one died from a ruptured abdominal aortic aneurysm before undergoing open repair, and one died from pneumonia before undergoing endovascular repair. There were six crossovers: five patients who were randomly assigned to undergo open repair underwent endovascular repair, and one patient assigned to endovascular repair underwent open repair. Overall, the operation was started according to the randomized assignment in 96.6 percent of patients (339 of 351).

The baseline characteristics of the patients are given in Table 1. Demographic characteristics, the prevalence of coexisting conditions, cardiovascular-risk profiles, the distribution of American Society of Anesthesiologists risk classes, and medication use were similar in the two groups.

The median interval between randomization and the procedure was 39 days in both the open-repair group (range, 4 to 260) and the endovascular-repair group (range, 1 to 183; P=0.76); 92.6 percent

---

**Table 1. Baseline Characteristics of the Patients.**

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>Open Repair (N=178)</th>
<th>Endovascular Repair (N=173)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age — yr</td>
<td>69.6±6.8</td>
<td>70.7±6.6</td>
</tr>
<tr>
<td>Male sex — no. (%)</td>
<td>161 (90.4)</td>
<td>161 (93.1)</td>
</tr>
<tr>
<td>Mild, moderate, or severe SVS/ISCVS risk-factor score — %†</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Diabetes mellitus</td>
<td>9.6</td>
<td>10.4</td>
</tr>
<tr>
<td>Tobacco use</td>
<td>55.1</td>
<td>64.2</td>
</tr>
<tr>
<td>Hypertension</td>
<td>54.5</td>
<td>58.4</td>
</tr>
<tr>
<td>Hyperlipidemia</td>
<td>52.6</td>
<td>47.0</td>
</tr>
<tr>
<td>Carotid artery disease</td>
<td>15.2</td>
<td>14.5</td>
</tr>
<tr>
<td>Cardiac disease</td>
<td>46.6</td>
<td>41.0</td>
</tr>
<tr>
<td>Renal disease</td>
<td>8.4</td>
<td>7.5</td>
</tr>
<tr>
<td>Pulmonary disease</td>
<td>18.5</td>
<td>27.7</td>
</tr>
<tr>
<td>Total SVS/ISCVS risk-factor score†</td>
<td>4.5±2.3</td>
<td>4.4±2.5</td>
</tr>
<tr>
<td>FEV₁ — liters/sec</td>
<td>2.6±0.7</td>
<td>2.5±0.7</td>
</tr>
<tr>
<td>Body-mass index</td>
<td>26.6±4.1</td>
<td>26±3.4</td>
</tr>
<tr>
<td>ASA class — no. (%)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>I (healthy status)</td>
<td>44 (24.7)</td>
<td>37 (21.4)</td>
</tr>
<tr>
<td>II (mild systemic disease)</td>
<td>110 (61.8)</td>
<td>122 (70.5)</td>
</tr>
<tr>
<td>III (severe systemic disease)</td>
<td>24 (13.5)</td>
<td>14 (8.1)</td>
</tr>
<tr>
<td>Medication use — no. (%)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Beta-blockers</td>
<td>92 (51.7)</td>
<td>76 (43.9)</td>
</tr>
<tr>
<td>Statins‡</td>
<td>72 (41.9)</td>
<td>63 (37.3)</td>
</tr>
<tr>
<td>Antiplatelet agents</td>
<td>72 (40.4)</td>
<td>70 (40.5)</td>
</tr>
<tr>
<td>Angiotensin-converting–enzyme inhibitors</td>
<td>50 (28.1)</td>
<td>58 (33.5)</td>
</tr>
<tr>
<td>Calcium-channel blockers</td>
<td>32 (18.0)</td>
<td>30 (17.3)</td>
</tr>
<tr>
<td>Anticoagulants</td>
<td>27 (15.2)</td>
<td>20 (11.6)</td>
</tr>
</tbody>
</table>

* Plus–minus values are means ±SD. There were no significant differences between the groups. FEV₁ denotes forced expiratory volume in one second, and ASA American Society of Anesthesiologists. The body-mass index is the weight in kilograms divided by the square of the height in meters. Because of rounding, not all percentages total 100.

† The Society for Vascular Surgery/International Society for Cardiovascular Surgery (SVS/ISCVS) risk-factor score is calculated for eight domains, and scores for each domain can range from 0 (no risk factors) to 3 (severe risk factors).

‡ No information on the use of statins was available for six patients in the open-repair group and four patients in the endovascular-repair group.
of patients (325 of 351) underwent aneurysm repair within 3 months after randomization. The mean duration of follow-up was 21 months in the open-repair group (range, 0 to 39) and 22 months in the endovascular-repair group (range, 1 to 42). A total of 6 patients were lost to follow-up during the first year (follow-up 98.3 percent complete) and 19 during the first two years (follow-up 94.6 percent complete).

**Mortality**

Two years after randomization, the cumulative survival rates were 89.6 percent for open repair and 89.7 percent for endovascular repair, for a difference of −0.1 percentage point (95 percent confidence interval, −6.8 to 6.7 percentage points; P=0.86) (Fig. 1). The small but apparent survival advantage in the first year after endovascular repair did not reach statistical significance (P=0.15) and appeared to be based entirely on a decreased rate of in-hospital (perioperative) mortality.

There was one preoperative death and eight in-hospital deaths in the open-repair group and one preoperative and two in-hospital deaths in the endovascular-repair group (Table 2). Taking into account the patients who declined treatment (three in the open-repair group and one in the endovascular-repair group), there were 166 discharges after open repair and 169 discharges after endovascular repair. The causes of death are listed in Table 2. After discharge, there were more deaths from cardiovascular causes in the endovascular-repair group than in the open-repair group (six vs. three), although this difference was not significant (P=0.50).

There was an unexplained cluster of deaths in the endovascular-repair group approximately one year after randomization (Fig. 1). None of these deaths were considered to be aneurysm-related as defined in the Methods section; two of the deaths were due to heart failure, one to acute cardiac arrest, one to stroke, and one to aspiration pneumonia in a patient with metastatic carcinoma of the bladder.

**Aneurysm-Related Mortality**

The cumulative rates of aneurysm-related death two years after randomization were 5.7 percent in the open-repair group and 2.1 percent in the endovascular-repair group, for a difference of 3.7 percentage points (95 percent confidence interval, −10.0 to 10.6 percentage points; P=0.88) (Fig. 1). The difference in aneurysm-related mortality at two years was based entirely on the difference in in-hospital (perioperative) mortality. After discharge, only one additional aneurysm-related death occurred in each group (Table 2).

**Complications**

Two years after randomization, the rates of survival free of severe events were 80.6 percent for open repair and 83.1 percent for endovascular repair, for a difference of −2.5 percentage points (95 percent confidence interval, −10.9 to 5.9 percentage points; P=0.39) (Fig. 2). As with the data on aneurysm-related mortality, the difference in the rate of survival free from severe events at two years was based entirely on the difference in in-hospital events. The rates of survival free of moderate or severe events two years after randomization were 65.9 percent for open repair and 65.6 percent for endovascular repair, for a difference of 0.3 percentage point (95 percent confidence interval, −10.0 to 10.6 percentage points; P=0.88).

There were no documented postoperative aneurysm ruptures. However, in two patients who died after endovascular repair, the possibility of aneurysm rupture was considered but not proved (Table 2).

Kaplan–Meier estimates of the likelihood of freedom from reintervention are shown in Figure 3. In the first nine months after randomization, the rate of reintervention after endovascular repair was al-
most three times the rate after open repair (hazard ratio, 2.9; 95 percent confidence interval, 1.1 to 6.2; P=0.03). Thereafter, reintervention rates were roughly parallel (hazard ratio, 1.1; 95 percent confidence interval, 0.1 to 9.3; P=0.95).

**DISCUSSION**

We found that by the end of the first year after randomization, the previously reported perioperative survival advantage of endovascular aneurysm repair over open repair was no longer apparent. Although a lower rate of aneurysm-related death after endovascular repair did appear to be maintained during the first two years, in terms of overall survival, this was cancelled out by excess mortality from other causes, including cardiovascular causes, in the first two years after discharge.

One other randomized trial, the Endovascular Aneurysm Repair (EVAR-1) trial, has compared the results of endovascular aneurysm repair with those of open repair. Whereas the early results of the two trials were similar, the long-term results of EVAR-1 are not yet available and thus cannot be compared with our findings.

Our results are similar to those of two recently reported retrospective, controlled studies comparing endovascular and open repair. In both studies, the respective one-year survival rates after open and endovascular repair were approximately 92 and 95 percent, and the respective two-year survival rates were approximately 88 and 89 percent, all of which are very close to our findings. The rates of aneurysm-related death two years after open and endovascular repair were 4.2 and 0.9 percent, respectively, in the study by Cao et al., as compared...
with 5.7 and 2.1 percent, respectively, in our study. It is possible that the prospective nature of our study allowed for more complete detection of aneurysm-related deaths. The difference in reintervention rates between the groups in our study is also similar to that reported in both retrospective studies. In one study, the divergence of reintervention rates did not start until after two years of follow-up, whereas in our study, there was no significant difference in reintervention rates beyond nine months after randomization. This variation may depend on how aggressively certain complications are addressed.

Although our findings — and those in the other trials discussed above — suggest that endovascular aneurysm repair may provide an early survival advantage over conventional surgery, it appears that this advantage is lost by the end of the first year. It is unknown whether the durability of the endovascular graft will jeopardize long-term outcomes. Although nonrandomized, follow-up studies of patients who have undergone aneurysm repair have failed to show a long-term advantage of open over endovascular repair, concerns persist, since the rates of aneurysm-related death and reintervention after endovascular repair have been reported to continue to increase over time. The overall survival curves in our trial appeared to converge in the second year after randomization. Our 2-year data do not exclude the possibility that these curves will actually cross, resulting in a higher rate of death for endovascular repair than for open repair after 24 months.

There may be two possible explanations for the convergence of survival curves in our study. One is that patients who have survived the stress of open repair may be somewhat less likely to die in the first few months after surgery than patients who have undergone endovascular repair, since the latter group has not been subjected to a conventional surgical procedure. In other words, the survival advantage resulting from a less-invasive approach to aneurysm repair may largely be based on postponing death among higher-risk patients from the perioperative period to the subsequent months. Although patients in our trial had to be eligible to undergo conventional open aneurysm repair before they could undergo randomization, the health of patients with abdominal aortic aneurysms is often seriously compromised by other types of cardiovascular disease. In our study, 58 percent of the deaths (22 of 38) were due to either cardiovascular causes or causes related to aneurysm repair. This finding is in accordance with those of other follow-up studies of aneurysm repair. Another possible explanation for the convergence of survival curves is the failure of endovascular repair to prevent rupture of the aneurysm. However, endograft failure is unlikely to occur during the first two years after implantation, and such fail-
ure would be reflected by a convergence of the rates of aneurysm-related death — an effect that was not found in our analysis. Although a grouping of deaths was seen in the endovascular-repair group about one year after randomization, the causes of death were not related to the aneurysm. Furthermore, the apparent grouping of these deaths was seen in a Kaplan–Meier survival analysis that measured the time from randomization, rather than the time from the procedure, indicating that this grouping of deaths was not related to the course after intervention. Only one patient in the endovascular-repair group died of an aneurysm-related cause (an infected endograft) after hospital discharge. Whether the rate of graft failure will increase with further follow-up remains to be seen.

In patients undergoing endovascular repair, efforts should be made to maintain the survival advantage associated with avoiding conventional surgery. This effort may at least in part be a matter of strict risk-factor management. Beta-blockers, antiplatelet agents, and statins were each being used in less than 50 percent of our patients at baseline. Clearly, less-than-optimally effective medication was used in view of current guidelines on risk management for patients with manifestations of atherosclerosis.14–16 Of course, better perioperative and postoperative management of risk factors could also improve the results of open aneurysm repair.

In conclusion, the two-year results of the DREAM trial indicate that the perioperative survival advantage with endovascular repair as compared with open repair is limited to the first postoperative year.

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We are indebted to the Netherlands Society for Vascular Surgery for its support and to Nicole Boekema for her outstanding efforts in data management.

### References


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10 Years of Emergency Endovascular Aneurysm Repair for Ruptured Abdominal Aortoiliac Aneurysms: Lessons Learned

Dieter Mayer, MD,* Thomas Pfammatter, MD,† Zoran Rancic, PhD,* Lukas Hechelhammer, MD,† Markus Wilhelm, MD,* Frank J. Veith, MD,‡ and Mario Lachat, MD*

Objectives: To evaluate a single center’s 10-year experience with emergency endovascular aneurysm repair (eEVAR) in 102 patients with ruptured abdominal aortoiliac aneurysms (RAAA).

Methods: Data from 102 patients (mean age, 73 ± 9 years) with RAAA treated by eEVAR from January 1998 to April 2008 were retrospectively reviewed. From January 2000, all patients were treated according to an intention-to-treat protocol. The only exclusion criterion was unsuitable anatomy. 31/102 patients had moderate shock and 14/102 patients had severe shock with a systolic blood pressure <70 mm Hg or <50 mm Hg, respectively. 71/102 procedures were carried out under local anesthesia. Endograft types used were mainly bifurcated (92/102). Open abdomen treatment (OAT) because of abdominal compartment syndrome (ACS) was used when signs of organ failure occurred and/or bladder pressure rose >20 mm Hg.

Results: The 30-day mortality for eEVAR was 13% (13/102). Technical success (defined as successful deployment of the endograft, absence of extravasation in the postprocedural contrast enhanced CT scan and hemodynamic stabilization) was 99% (101/102). Nineteen unstable patients (19%) required transfemoral suprarenal aortic balloon occlusion. ACS was detected and treated by OAT in 20 patients (20%). 16 type I, 26 type II and 1 type III endoleaks were detected on postoperative CT examination. Two patients had a combined type I and II endoleak. 11 patients were retreated for immediate correction of 10 type I and 2 type II endoleaks. 6 type I and 1 type III low-flow endoleaks were observed and resolved spontaneously within 30 days. Major 30-day morbidity was 35%.

Conclusion: In this 102 patient contemporary series of eEVAR for RAAA, endografting proved to be safe with a 30-day mortality of 13%. Key components of this favorable outcome result were adequate preoperative diagnostic imaging, hypotensive hemostasis, selective transfemoral suprarenal aortic balloon occlusion, predominantly local anesthesia, detection and treatment of ACS, and attention to logistics. Widespread adoption of these treatment components is recommended.


Emergency eEVAR (eEVAR) for ruptured abdominal aortic aneurysms has attracted the interest of the vascular community since open repair (OR) still carries a high risk of death and serious morbidity even after 50 years of experience.1 However, with few exceptions, the eEVAR series published to date report less than 5 year experiences with small numbers of patients. The present report describes a 10-year experience with eEVAR at the University Hospital Zurich and emphasizes some key treatment points developed and used over this period.
gist, cardiovascular anesthetist and vascular surgeon are available. As an institution with a vast activity in elective EVAR procedures (approximately 700 abdominal and 200 thoracic EVAR procedures to date), a broad stock of bifurcated and aorto-uni-iliac endografts is available. eEVAR procedures may be carried out in a fully equipped emergency OR or in an angiography suite. CT scans are available within 5–15 minutes as the scanner is part of the shock room. An eEVAR education program is provided to all potential staff members.

**Patient Sample**

102 patients with documented ruptured aortoiliac aneurysms underwent eEVAR from January 1, 1998 to April 30, 2008. The mean age was 73 ± 9 (median: 74, min: 48, max: 90) years and the proportion of male to female 84/18. Of the 102 patients, 31 had moderate shock and 14 had severe shock with a systolic blood pressure <70 mm Hg or <50 mm Hg, respectively. Nineteen patients were unstable (ie, could not be stabilized with a blood pressure >50 mm Hg even with fluids and positive inotropes). Sixty-four patients presented with a contained retroperitoneal rupture and 38 patients with a free rupture including 5 patients with an aortoduodenal fistula with coexisting AAA.

All but 2/223 announced RAAA patients were admitted to our shock room. On both occasions the eEVAR team was already occupied with another endovascular emergency case. Overall, 221 patients with RAAA were admitted. Nine of the 221 patients were not operated on because of their unfavorable prognosis. The proportion of patients treated by eEVAR compared with OR was 48%. The annual relative proportion treated by eEVAR increased in the later years up to 58% due to widening of the indications for EVAR (Fig. 1).

**Preoperative Management**

At first presentation, all involved emergency staff members and other centers were instructed to stop all fluids (infusions and blood products) and positive inotropes (catecholamines) and to strive for a target blood pressure of 70–90 mm Hg whenever possible. We practiced hypotensive hemostasis with strict limitation of fluid administration and active pharmacologic lowering of blood pressure. In patients with severe hypertension, we started intravenous administration of phentolamine and esmolol as needed. If the patient had a systolic blood pressure less than 120 mm Hg (but greater than 90 mm Hg) and a heart rate over 100 per minute, esmolol was given alone until the target blood pressure was reached. Additionally, most patients received nitroglycerine. However, there were no generally applicable rules and the active lowering of blood pressure was carried out based on the patient’s status and the physicians’ judgement. If the patient was unstable, a transfemoral supraceliac occlusion balloon was inserted under fluoroscopic control in the emergency room (or thereafter elsewhere whenever necessary).2,3 After obtaining a rapid history, physical examination, baseline blood tests and ECG and after insertion of appropriate tubes and lines by anesthesiologists and vascular surgeons, a thoracoabdominal CT scan was obtained when not provided by the referring center. CT scans were analyzed and discussed online between the interventional radiologist and the vascular surgeon in charge. Generally, decisions about the feasibility of eEVAR could be made within 10–20 minutes of a patient’s arrival.

**Treatment**

Interventions were carried out either in a fully equipped (fluoroscopy including digital subtraction angiography [DSA]) emergency operating room or in a dedicated angiography suite when anatomic challenges or morbid obesity were present. The team was always composed of one fully trained interventional radiologist and one fully trained vascular surgeon competent in endovascular and open techniques.

EVAR was carried out in a standardized fashion according to our procedures for elective patients. Briefly, a unilateral femoral open exposure was made under local anesthesia as the preferred access for introduction of the main body of the endograft.4 Percutaneous access was obtained on the contralateral side for imaging procedures and introduction of the contralateral limb or plug. Seventy-one of 102 procedures (71%) were carried out under local anesthesia (LA) and analgesedation with remifentanil. Conversion to general anesthesia was required after sealing of the rupture in 17/102 patients for secondary procedures such as open abdomen treatment (OAT), femorofemoral bypass and/or iliac reconstruction. Overall, sealing of the rupture site was obtained under LA in 88% of the cases. Fourteen of the 102 patients (14%) were treated from the

**TABLE 1. Exclusion Criteria for eEVAR**

<table>
<thead>
<tr>
<th>Exclusion Criteria</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>Proximal neck length</td>
<td>&lt;5 mm</td>
</tr>
<tr>
<td>Proximal neck diameter</td>
<td>&gt;30 mm</td>
</tr>
<tr>
<td>Proximal neck angulation</td>
<td>&gt;90°</td>
</tr>
<tr>
<td>Distal neck diameter</td>
<td>&gt;20 mm</td>
</tr>
<tr>
<td>Bilaterally occluded/inaccessible iliac arteries</td>
<td></td>
</tr>
</tbody>
</table>

eEVAR, emergency endovascular aneurysm repair.

**FIGURE 1. Proportion of eEVAR versus OR for RAAA over 10 years. OR = open repair, eEVAR = emergency endovascular aneurysm repair.**
beginning with general anesthesia for various reasons (intubated on arrival, agitations, too much pain in the abdomen). The endografts were deployed under fluoroscopic control. Bifurcated endografts were preferred. Aortouniliac (AUI) devices were used when indicated (only one suitable iliac artery, limited time due to unstable patient, difficult anatomy). Transfemoral suprarenal balloon occlusion with a Reliant balloon catheter, 46 mm in diameter and requiring a sheath size of 12F or larger (Medtronic Cardiovascular, Santa Rosa, California) was performed at any time during the procedure if necessary. At the end of the procedure, a contralateral open femoral exposure was made to control the access vessel. Bladder pressure was monitored. Inguinal compression bandages were placed to minimize bleeding and lymphatic fistulas.

Various types of endografts with or without extensions were used: Vanguard bifurcated \((n = 9)\), Excluder bifurcated \((n = 53)\), Talent bifurcated \((n = 3)\), Zenith bifurcated \((n = 27)\) and Zenith AUI \((n = 9)\). One Zenith cuff was inserted for a type Ia endoleak. In 24/102 cases (24%) endograft components from different manufacturers were used as hybrid endografts.

OAT was performed because of abdominal compartment syndrome (ACS) at any time after sealing of the rupture when signs of organ failure occurred and/or bladder pressure rose above 20 mm Hg. Abdominal decompression was carried out by a standard midline incision (laparotomy) from the xiphoid process to the pubic bone under general anesthesia (in 17 patients conversion from local to general anesthesia was necessary). Depending on the clinical findings, the abdomen was left open and the opening controlled either with a plastic bag silo fixed to the skin with a running suture or with a zipper drape (Ethizip, Ethicon Inc, New Jersey). Indications for the plastic bag silo were massive bowel and omental swelling due to edema and/or reperfusion injury and suspicion of bowel ischemia. The bag provided transparency and was the base layer of an exceedingly compliant dressing which was then completed with an absorptive gauze dressing. When there was no or only moderate bowel swelling and no suspicion of intestinal ischemia, the zipper drape was used as a sealing and gliding layer to separate the abdominal contents from the abdominal wall and the vacuum assisted closure system (standard black poly-urethane foam, continuous suction of 50–100 mm Hg, V.A.C., KCI International, Amsterdam, The Netherlands) applied on top of the zipper drape. This dressing system provided an easy to handle, sterile dressing that allowed early and full mobilization of the patient.

Retroperitoneal hematomas were usually not evacuated to avoid further bleeding due to coagulopathy. Second look interventions took place every 1 to 2 days when treated with the plastic bag silo and every 3 to 5 days when treated with the vacuum assisted closure. As soon as the bowel swelling decreased and intestinal ischemia was ruled out, the plastic bag silo was exchanged for the zipper drape and vacuum assisted closure system and/or progressive abdominal wall closure was initiated by fascial approximation with single size 1 monofilament polyglyconate synthetic absorbable sutures.

Postoperative Management

All patients received a control contrast CT scan immediately after the intervention. Type I and III endoleaks were corrected by reinterventions immediately after detection.

After the CT scan, all patients were admitted to the ICU where bladder pressure was monitored every hour or continuously with a pressure transducer connected to the urinary catheter. Whenever bladder pressure exceeded 20 mm Hg or signs of organ failure occurred, OAT was initiated. Low-dose heparin (5–10,000 U per day, continuously IV) was given 2 hours after the intervention when bleeding was under control. Aspirin (100 mg) daily was administered the next day and continued indefinitely. Oral anticoagulation was initiated after 2–5 days for 3 months to prevent deep vein thrombosis. All patients were given statins indefinitely. The hematocrit was corrected to a value of at least 30%. The use of acetaminophen and metamizol was avoided as were other nephrotoxic substances.

Oral intake was started as soon as possible. Patients were discharged from the ICU when they had no signs of organ failure and when bowel function resumed. Patients were progressively mobilized and oral intake increased. Before discharge, a control CT contrast scan and plain abdominal x-ray was carried out as a baseline examination. Compression bandages were left for 5–7 days. Further clinical examinations, contrast CT scans and plain abdominal x-rays were obtained 3 and 12 months after the intervention and yearly thereafter.

RESULTS

Endovascular grafts were placed with technical success (defined as successful deployment of the endograft, absence of extravasation on the postprocedural contrast enhanced CT scan and hemodynamic stabilization) in 99% (101/102) of patients. Nineteen patients (19%) were sufficiently unstable to need transfemoral suprarenal aortic balloon occlusion. ACS was detected in 20 patients (20%). All 20 underwent laparotomy and OAT. Immediately thereafter, pulmonary and urinary function improved and circulatory stabilization occurred.

Six type Ia, 10 type Ib, 26 type II, and 1 type IIIa endoleaks were detected on postoperative CT examination (Table 2). One patient had a combined type Ia and type II endoleak, and 1 patient had a combined type Ib and II endoleak. Eleven patients were returned to the operating room for early sealing of 1 type Ia, 9 type Ib and 2 large type II endoleaks. Five type Ia, 1 type Ib, and 1 type

| TABLE 2. Primary Endoleaks (First CT Scan Immediately After eEVAR) |
|---------------------|-----|-----|-----|-----|
| Endoleak Type | Ia | Ib | II | IIIa |
| Number of EL | 6 | 10 | 26 | 1 |
| Re-intervention (EVAR) | 1 | 9 | 2 | 0 |
| Conservative treatment* | 5 | 1 | 24 | 1 |

*Insignificant low flow endoleaks.

| TABLE 3. Reasons for 30-Day Mortality |
|-----------------|-----------------|-----------------|
| Mortality | Time |
| Suicide | 14 d po |
| Liver failure (Cirrhosis) | 10 d po |
| Bleeding (Re-rupture) | 7 d po |
| Sudden death (VF) | 3 d po |
| Pneumonia | 29 d po |
| Bleeding (after OAT) | On-Table |
| MOF | 12 d po |
| MOF | 2 d po |
| Acute MI | On-Table |
| Acute MI | 25 d po |
| MI, myocardial infarction; MOF, multi-organ failure; |
| MC, multi-organ failure; MI, myocardial infarction; ICU, intensive care unit. | |

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IIa were regarded as minor low-flow endoleaks and were observed with spontaneous resolution within 30 days. Twenty-four type II endoleaks were treated conservatively.

The 30-day mortality was 13% (13/102). The causes of death are shown in Table 3. It is noteworthy that one patient suffered from decompression bleeding after OAT for ACS and this patient died during conversion to OR. Patients undergoing OAT had multiple postoperative interventions until full primary or secondary closure. Twenty-four type II endoleaks were treated conservatively. With spontaneous resolution within 30 days. Twenty-four type II endoleaks were treated conservatively.

### DISCUSSION
Since its first performance in 1994 and descriptions by Yusuf, Hopkinson et al. and Marin, Veith et al., more than 3,000 papers have been published dealing with emergency EVAR (eEVAR) for RAAA. Although the overall evidence is growing that eEVAR may improve the outcome of RAAA, some groups have not been able to confirm the advantage of eEVAR over OR. To date, the positive impact of eEVAR on the mortality of RAAA patients has been attributed to its minimally invasive nature. Recently however, multiple adjunctive diagnostic and therapeutic measures that could improve the outcome of all patients treated for RAAA have been identified. These include the adoption of a discrete protocol for RAAA patients treated with eEVAR, the use of hypotensive hemo-

### Preoperative Diagnostic Imaging
Preoperative CT angiography is generally regarded as the best diagnostic imaging and is considered the gold standard. However, some groups advocate that eEVAR is feasible in some cases with only intraoperative angiography. In our institution, CT angiography is rapidly available, and all the patients underwent a thoracoabdominal contrast CT scan unless it had been obtained by the referring institution. Interdisciplinary online discussion and measurements allow for an ideal preoperative strategic planning including the choice of the optimal endograft type and size.

### Hypotensive Hemostasis
The concept that passive or active lowering of blood pressure can improve the outcome of patients with aortic and other bleeding is not new. The main benefit of the lower blood pressure is that it decreases arterial bleeding as shown by Andresen in gastro-intestinal bleeding in the late 1940s and by Shafran et al. in experimental work in 1965. In 2002, Veith et al. concluded that hypotensive hemostasis or restricted fluid resuscitation will prove valuable in the RAAA setting and would become the standard of care for this entity leading to improved treatment outcomes.

Group, along with a few others, has gone a step further in that we carry out active pharmacologic lowering of blood pressure to a target value of 70–90 mm Hg unless there are signs of cerebral or cardiac malperfusion. However, there is uncertainty about which drug is best for lowering the blood pressure of RAAA patients. In eEVAR for RAAA, there is to date only one report on this topic, indicating that this option might not be widely used. Further studies should be undertaken to clarify this issue.

### Transfemoral Supraceliac Balloon Occlusion
Although most RAAA patients can be rendered relatively stable over the short term by limiting resuscitation, some patients will undergo complete circulatory collapse which requires treatment. Intraluminal aortic occlusion was first described in experiments on dogs by Edwards et al. in 1953, followed by the reports of Hughes and others on clinical applications for control of massive intraab-

### Local Anesthesia
It has been shown that EVAR can be performed under local anesthesia and that the latter is beneficial. Transfemoral balloon occlusion for RAAA patients was first described by Ng and Ochsner in 1977. Open transaxillary (remote) aortic balloon occlusion for RAAA patients was first described by Ng and Ochsner in 1977. Open transaxillary (remote) aortic balloon occlusion for RAAA patients was first described by Ng and Ochsner in 1977. In 1999, Okhi and Veith advocated a more liberal use of a percutaneously insertable balloon from either the brachial artery or the femoral artery.

At our institution, the percutaneous transfemoral technique similar to that described by Malina, Veith, Ivancev et al. is the preferred method for temporary aortic occlusion. Since 1998, it has been used in 19/102 eEVAR patients (19%) successfully. However, such balloon occlusion is not without risks and one of our patients with severe supraceliac atheromatosis was lost from multiple emboli into visceral arteries. For this reason we only use balloon occlusion when we consider it absolutely necessary.
as reflected by the wide variability in the proportion of patients having eEVAR under LA in contemporary comparative studies.

Abdominal Compartment Syndrome

ACS is a major cause of death after treatment of RAAA and increases short-term mortality up to 5 times compared with patients with normal intra-abdominal pressure.22–25 Monitoring for ACS by bladder pressure measurement is mandatory in every RAAA patient and OAT must be considered when bladder pressure rises over 20 mm Hg.23,42,43 At our institution, OAT was necessary in 20/102 RAAAs (20%) treated by eEVAR. This rate of ACS corresponds well with the published rates for OR22,24,25 (20–33%) and e-EVAR22 (20%). In some of the comparative studies that failed to detect any advantage of eEVAR over OR, only sporadic cases of ACS were reported.10–12 This might reflect the under recognition of an entity described in 1984 for RAAA patients44 and raises the question of whether eEVAR outcomes would have been favorably influenced if ACS was more actively diagnosed and treated. However, decompression laparotomy after successful eEVAR may also lead to mortality due to decompression bleeding, as in one of our patients. Although decompressive bleeding is rare, we now routinely perform a repair of the aortic rupture site with a (biologic) patch graft when the abdomen is opened for any reason.

Logistics

Human resources and the in stock availability of adequate endograft components seem to be major issues in the eEVAR treatment of RAAA. In 7 contemporary comparative publications, logistic problems were mentioned and often led to the exclusion of patients suitable for eEVAR.12,17,33,45–47 Such patients were excluded from eEVAR treatment because of: (i) Lack of availability of EVAR trained staff (surgeons; radiologists; specialized scrub nurses; specialized radiologic technicians); (ii) Unavailability of appropriate endograft components. Several reports mention the importance of having a protocol for eEVAR in RAAA patients and emphasize the importance of having trained staff 24 hours a day, 7 days a week and an adequate stock of endograft components.13,14

CONCLUSION

In this contemporary 10 year series of 102 eEVAR for RAAA, endovascular graft treatment was employed whenever it was anatomically feasible. This form of treatment was safe with a 30-day mortality of 13%. Adherence to an intention to treat protocol with certain multimodal features appeared to play a key role in obtaining these low mortality results. These features include adequate preoperative diagnostic imaging, hypotensive hemostasis, transfemoral supraceliac aortic balloon occlusion, local anesthesia supplemented by analgesia, recognition and treatment of abdominal compartment syndrome by OAT and recognition of the importance of logistic considerations. We believe that adoption of these adjunctive treatment features will help to improve the outcomes for eEVAR treatment of RAAA, and that this will eliminate much of the controversy surrounding this new approach to treatment of RAAA. Future studies of eEVAR in this setting, including possible randomized controlled comparisons with OR, should incorporate these treatment features into their protocols to achieve meaningful results and assure a fair evaluation of eEVAR.

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Risk of new aneurysms after surgery for popliteal artery aneurysm

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Background: The risk of developing a new aneurysm after surgery for popliteal artery aneurysm (PAA) is not well known. The aim was to study this risk in a cohort of patients.

Methods: A total of 571 patients who had primary operation for PAA (717 legs) between 1987 and 2002 were identified from the Swedish Vascular Registry (Swedvasc). Of these, 190 patients were re-examined by ultrasonography after a median of 7 (range 2.9–18.7) years.

Results: The number of patients with at least one aneurysm in addition to the PAA was 108 (56.8 per cent) at the index operation and 131 (68.0 per cent) at re-examination. The overall number of aneurysms increased by 41.8 per cent, from 244 to 346. Among the 82 patients who had an isolated PAA at the index operation, 23 developed a new aneurysm; these patients tended to be older (P = 0.004). Bilateral PAA at the index operation was associated with a later development of abdominal aortic aneurysm (P = 0.004). Age (P = 0.004) and hypertension (P = 0.012) at the time of the index operation were associated with multianeurysm disease at any time. Six (4.3 per cent) of 138 legs treated by venous bypass grafts had developed a graft aneurysm by the time of re-examination. No normal arterial segment developed an aneurysm that required surgery within 3 years.

Conclusion: The development of new aneurysms was common in patients with a PAA; lifelong surveillance may be warranted.

Introduction

Follow-up after treatment of a popliteal artery aneurysm (PAA) focuses on surveillance of the reconstruction, normally ultrasonography during the first postoperative year. Although routines differ between centres, patients have often completed follow-up by 1 year. Whether continued surveillance is warranted remains controversial.¹

PAA are often bilateral and associated with abdominal aortic aneurysm (AAA).¹–⁵ The prevalence of aneurysms in other anatomical positions, at different time points, among patients who have surgery for unilateral or bilateral PAA is not well known. The only previous study¹ reported on 50 patients identified between 1958 and 1985 and followed for a mean of 5 years; ultrasonographic imaging was available only in the later years. This information is crucial in order to determine whether a surveillance programme is worthwhile and should include investigation of the operated PAA, because dilatation and aneurysm formation in venous grafts and even of the excluded aneurysm have been reported.⁶,⁷ The aim of this study was to investigate the long-term risk of developing new aneurysms in the contralateral leg, in other anatomical positions and in the venous graft of patients who had surgery for PAA. The risk factors for developing new aneurysms during follow-up were also investigated.

Methods

A total of 571 patients who had a primary operation for PAA on 717 legs between 1987 and 2002 were identified from the Swedish Vascular Registry (Swedvasc). The study design, including extensive validation of registry data, has been described in detail previously.²,⁶,⁸ Core surgery is registered with great validity in the Swedvasc³–⁵¹ and this is also relevant for PAA procedures.
The number of bilateral and extrapopliteal aneurysms at the time of the primary PAA procedure (index operation) was based on information from patients’ records at the local hospital. It was not always possible to distinguish whether an aneurysm was known previously, or if it was diagnosed in connection with the index operation. The characterization of any aneurysmal dilatation at the time of the index operation, based on data from case records, was clear for all contralateral popliteal arteries and abdominal aortas, although not for all femoral and iliac arteries. Most patients had been investigated by ultrasonography.

In Sweden every citizen has a unique personal identity code, which is used as the identification code in the Swedvasc. In January 2005 data were cross-checked against the national population registry, resulting in accurate survival data: 337 patients were alive at that time, and were asked to participate in a telephone interview and a re-examination, of whom 240 agreed.

The patients were asked about smoking habit, medication, family history of aneurysms, history of amputation and of aneurysm repair in the contralateral popliteal artery or at other anatomical locations, and any symptoms from the operated leg.

A total of 192 patients (242 legs) participated in the re-examination. Two patients (three legs) had a clinical examination only and were excluded from this study. Most patients who declined to participate in the interview or re-examination were either elderly or infirm. Several patients died between the telephone interview and the re-examination, and others decided to participate in the telephone interview only.

The re-examination was carried out at one of 38 local hospitals, during 2005–2006. The principal investigator, together with the same experienced ultrasound technologist from the vascular laboratory at Uppsala University Hospital, examined 163 patients by visiting the local hospital. Another 27 patients were examined by a local vascular surgeon and ultrasound technologist (25) or by computed tomography (two). The bypass was examined for flow, aneurysm formation and stenosis. The previously operated PAA was evaluated for size and blood flow in the aneurysm sac, and the patients were screened for aneurysms in other arterial segments: infrarenal aorta, common iliac arteries, common femoral arteries and contralateral popliteal artery. The ultrasonographic examination took approximately 20 min. Any previously unknown aneurysm found at re-examination was reported to the local surgeon for treatment or included in the local aneurysm surveillance programme. If the patient had a history of aneurysm repair at any location, the date of the operation was noted.

Table 1 Definitions of aneurysm in the different arterial segments

<table>
<thead>
<tr>
<th>Diameter of vascular segment (mm)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Infrarenal aorta</td>
</tr>
<tr>
<td>Common iliac artery</td>
</tr>
<tr>
<td>Common femoral artery</td>
</tr>
<tr>
<td>Popliteal artery</td>
</tr>
</tbody>
</table>

The definition of aneurysms used at re-examination is given in Table 1. Hypertension was defined as self-reported, medically treated, hypertension, and smoking as ever smoked at the time of the index operation. Multianeurysm disease was defined as any additional aneurysm (aortic, iliac, femoral or contralateral popliteal aneurysm) at any time (before or after index operation until re-examination). A new AAA or contralateral PAA was defined as one treated before re-examination or detected at re-examination, but not operated on or known about at the index operation.

The study was approved by all nine Regional Ethics Committees in Sweden. According to the administrative rules of the Swedvasc, each patient is asked for informed consent before registration. Each patient gave written informed consent before re-examination.

Statistical analysis

Independent samples t test was used for comparison of normally distributed data and Fisher’s exact test for comparison of two proportions. To estimate the odds ratio (OR) for factors associated with multianeurysm disease (after adjustment for sex, age, hypertension, smoking, first-degree relative with aneurysm and duration of follow-up), the variables were entered into a logistic regression model. Separate models were analysed for new AAA (or no AAA) and new contralateral PAA (or no contralateral PAA) as the dependent variables, where only subjects at risk were included. Thus, those with an AAA or PAA who had surgery before, or known about at, the index operation were excluded from the respective analysis. Bilateral PAA was included in the model when AAA was analysed, and AAA when contralateral PAA was analysed.

Results

Seven (3.7 per cent) of the 190 patients who participated in the telephone interview and the re-examination were women. The median (range) age of the group at index operation was 64 (18–84) years for men and 56 (22–79) years for women; age at re-examination was 71 (29–91) and 63 (30–85) years respectively. The median interval from index operation to re-examination was 7 (range 2.9–18.7) years. Information on hypertension was obtained for 188...
New aneurysms after popliteal artery aneurysm repair

The distribution of extrapopliteal aneurysms and PAA in the contralateral leg at index operation and re-examination is shown in Table 2. The number of patients with multiple aneurysms (at least one aneurysm in addition to the index PAA) at the index operation was 108 (56.9 per cent), which increased to 131 (68.9 per cent) after a median follow-up of 7 years. Between the index operation and re-examination the total number of aneurysms increased from 244 to 346 (by 41.8 per cent); the 102 new aneurysms developed in 74 patients.

The location of the new aneurysms that developed during the interval between the index operation and re-examination is shown in Table 3. Of 117 non-index aneurysms that were evaluated on re-examination, 61 (52.1 per cent) required operation. Of 72 new aneurysms, not previously detected but identified during re-examination, 32 (44 per cent) needed repair.

Of 82 patients who had an isolated PAA at the index operation, 23 developed new aneurysms during follow-up. They tended to be older at the index operation (mean age 64.0 versus 59.7 years; \( P = 0.004 \)). None of the 23 patients developed or had surgery for a new aneurysm within 3 years. Nine patients, however, had the non-index aneurysm detected during the fourth or fifth year of follow-up. Six of these patients had an aneurysm detected at re-examination; five met the criteria for surgery, three of whom had an AAA. Aneurysms in the other three patients were detected and operated on before re-examination (two PAA and one femoral artery aneurysm).

Of 108 patients with known non-index aneurysms at the primary operation, eight were re-examined within 3 years.

---

**Table 2** Prevalence of multiple aneurysms at three time points

<table>
<thead>
<tr>
<th></th>
<th>Treated before index operation</th>
<th>All aneurysms at index operation</th>
<th>All aneurysms at re-examination</th>
</tr>
</thead>
<tbody>
<tr>
<td>Total no. of patients</td>
<td>190</td>
<td>190</td>
<td>190</td>
</tr>
<tr>
<td>Total no. of aneurysms</td>
<td>76</td>
<td>244</td>
<td>346</td>
</tr>
<tr>
<td>No. of additional aneurysms</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>None</td>
<td>159 (83.7)</td>
<td>82 (43.2)</td>
<td>59 (31.1)</td>
</tr>
<tr>
<td>Extra aneurysms</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>One</td>
<td>9 (4.7)</td>
<td>47 (24.7)</td>
<td>41 (21.6)</td>
</tr>
<tr>
<td>Two</td>
<td>6 (3.2)</td>
<td>21 (11.1)</td>
<td>26 (13.7)</td>
</tr>
<tr>
<td>Three</td>
<td>12 (6.3)</td>
<td>19 (10.0)</td>
<td>25 (13.2)</td>
</tr>
<tr>
<td>Four</td>
<td>2 (1.1)</td>
<td>13 (6.8)</td>
<td>24 (12.6)</td>
</tr>
<tr>
<td>Five</td>
<td>1 (0.5)</td>
<td>2 (1.1)</td>
<td>8 (4.2)</td>
</tr>
<tr>
<td>Six</td>
<td>1 (0.5)</td>
<td>6 (3.2)</td>
<td>7 (3.7)</td>
</tr>
<tr>
<td>No. of affected regions</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Only index PAA</td>
<td>159 (83.7)</td>
<td>82 (43.2)</td>
<td>59 (31.1)</td>
</tr>
<tr>
<td>Extra regions</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>One</td>
<td>31 (16.3)</td>
<td>106 (56.8)</td>
<td>131 (68.9)</td>
</tr>
<tr>
<td>Two</td>
<td>9 (4.7)</td>
<td>47 (24.7)</td>
<td>41 (21.6)</td>
</tr>
<tr>
<td>Three</td>
<td>6 (3.2)</td>
<td>21 (11.1)</td>
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<td>Five</td>
<td>1 (0.5)</td>
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<td>8 (4.2)</td>
</tr>
<tr>
<td>Six</td>
<td>1 (0.5)</td>
<td>6 (3.2)</td>
<td>7 (3.7)</td>
</tr>
</tbody>
</table>

Values in parentheses are percentages. Four possible regions were evaluated: aortic, iliac, femoral and contralateral popliteal regions. PAA, popliteal artery aneurysm.

**Table 3** Distribution and size of aneurysms at re-examination

<table>
<thead>
<tr>
<th>Diameter (mm)</th>
<th>Total no.</th>
<th>Diagnosed at re-examination</th>
</tr>
</thead>
<tbody>
<tr>
<td>Aorta</td>
<td>30–39</td>
<td>16</td>
</tr>
<tr>
<td></td>
<td>40–49</td>
<td>10</td>
</tr>
<tr>
<td></td>
<td>≥ 50</td>
<td>6</td>
</tr>
<tr>
<td>Popliteal*</td>
<td>15–19</td>
<td>81</td>
</tr>
<tr>
<td></td>
<td>≥ 20</td>
<td>29</td>
</tr>
<tr>
<td>Iliac‡</td>
<td>&lt; 25</td>
<td>16</td>
</tr>
<tr>
<td></td>
<td>25–29</td>
<td>8</td>
</tr>
<tr>
<td></td>
<td>≥ 30</td>
<td>3</td>
</tr>
<tr>
<td>Femoral</td>
<td>&lt; 20</td>
<td>6</td>
</tr>
<tr>
<td></td>
<td>≥ 20</td>
<td>15</td>
</tr>
</tbody>
</table>

*Contralateral popliteal artery. †Six of these small popliteal artery aneurysms had mural thrombus, requiring surgery. ‡Three patients had bilateral iliac artery aneurysms.
Factors associated with the development of AAA after popliteal artery aneurysm surgery

Thirty-four AAA developed in 139 patients (24.5 per cent) with no AAA or history of AAA repair at the index operation. In a univariable analysis bilateral PAA at the index operation was significantly associated with the development of AAA ($P = 0.004$), whereas sex, age, hypertension, smoking, family history of aneurysm and duration of follow-up were not. Bilateral PAA retained the association with development of AAA in a logistic regression model after adjustment for all other factors (OR 2.96, 95 per cent confidence interval 1.26 to 6.98; $P = 0.013$).

Factors associated with the development of popliteal artery aneurysm in the contralateral leg

Of 110 patients with no PAA or history of PAA repair in the contralateral leg at index operation, 28 (25.5 per cent) developed a contralateral PAA. No significant associations were observed in univariable analysis, whereas both age (OR 1.1 per year; $P = 0.036$) and duration of follow-up (OR 1.2 per year; $P = 0.030$) were associated with the development of PAA in a logistic regression model after adjustment for all other factors.

Factors associated with multianeurysm disease

Of all 190 patients, 131 had a history of or developed additional aneurysm(s) at any site during follow-up. Univariable analysis revealed that age ($P = 0.004$) and hypertension ($P = 0.012$) at index operation were significantly associated with multianeurysm disease, whereas sex, smoking, family history of aneurysm and follow-up time were not. Age (OR 1.1 per year; $P = 0.003$) and hypertension (OR 2.1; $P = 0.041$) retained an association with multianeurysm disease in a logistic regression model after adjustment for all other factors. In addition, duration of follow-up was significantly associated with multianeurysm disease (OR 1.1 per year; $P = 0.036$).

Aneurysm formation in venous grafts

A vein graft was used in 140 (58.6 per cent) of 239 PAA repairs among 190 patients. Six of the 239 legs were amputated before re-examination, two of which had a vein graft. Thus, 138 legs with a vein graft PAA repair were re-examined. In six ($4-3$ per cent) there was segmental aneurysm formation of the vein graft. The time between index operation and re-examination in these patients ranged from 5 to 15 years. Five of the legs had a reoperation through a medial approach and one through a posterior approach. Five reconstructions were with reversed vein and one involved an in situ bypass.

Discussion

Most published reports on PAA have focused on surgical management and results; few studies reported long-term outcome. Patients with PAA are known to have a high prevalence of extrapopliteal aneurysms, but the risk of developing new aneurysms during follow-up after PAA repair is almost unknown. In this study, an additional 102 aneurysms developed in 74 patients, demonstrating the value of a surveillance programme. Early detection is fundamental to allow treatment before limb- or life-threatening complications occur.

Of 571 patients treated surgically for PAA and registered in Swedvasc, 190 (33.3 per cent) were re-examined and included in the study. Some 233 patients were no longer alive at the time of invitation, and so the true attendance rate was 56.2 per cent. In cohort studies it is essential to analyse and report subjects lost to follow-up. No information was available on the prevalence of aneurysms or the cause of death among non-attenders. The present study is not a true cohort study, although data obtained at index operation were registered prospectively. Non-attenders were older than attendees. As age was a risk factor for multianeurysm disease it is likely that non-attenders would have had an even higher prevalence of additional aneurysms, had they been examined.

Age, hypertension and duration of follow-up were independently associated with multianeurysm disease, in accordance with previous reports. Only 59 of 190 subjects did not develop any additional aneurysms. Whether isolated aneurysms and multiple aneurysms represent two separate cohorts is not clear. Subjects with isolated aneurysms were younger and had shorter follow-up, indicating that they may just have been studied at different stages of disease.

Bilateral PAA at the index operation was independently associated with the later development of AAA, in accordance with the report of Dawson and colleagues. The presence of an AAA at the index operation for PAA was, however, not associated with the later development of contralateral PAA, suggesting that bilateral PAA is a...
stronger marker for generalized aneurysm disease than the combination of a PAA and an AAA.

Although all patients with hypertension were receiving medical treatment, no information was available on the blood pressure, nor on the compliance with anti-hypertensive treatment. The possible preventive effect of antihypertensive therapy is therefore difficult to evaluate.

No patient developed a new aneurysm requiring surgery within 3 years of the index operation, but later during the fourth and fifth years several such aneurysms were detected. Based on this observation a practical suggestion would be to reinvestigate for new aneurysms every 3 years. Surveillance of known aneurysms was not the topic for this study, but has been investigated previously.\textsuperscript{5,12,16–19}

Dawson and colleagues\textsuperscript{1,11} suggested that patients older than 65 years, or with hypertension, are at high risk of developing a new aneurysm and should be in a lifelong surveillance programme after PAA repair. This recommendation can certainly be supported by the findings of the present study.

The development of new aneurysms is common among patients operated on for PAA. A complete examination of the aortoiliac and femoropopliteal arteries is warranted at the time of surgery. Consideration should be given that all patients, regardless of age or other risk factors, be kept under lifelong surveillance to detect and treat new aneurysms. This study suggests that previously normal arterial segments should be re-examined every 3 years.

**Acknowledgements**

Names of the responsible surgeons at the participating hospitals and members of the Swedvasc steering committee have been listed previously\textsuperscript{6,8}. This study was supported financially by the Swedish Research Council (grant K2007-64X-20406-01-3), Swedvasc, Erik, Karin and Gösta Selanders Foundation, and research funds of the County of Jönköping and of Uppsala University.

**References**


Mid-term outcomes of endovascular popliteal artery aneurysm repair

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Objective: This study documents mid-term outcomes of a series of endovascular popliteal aneurysm repairs compared with concurrent results of open surgical repair.

Methods: A retrospective chart review was done of all popliteal artery aneurysm repairs since January 1, 2000. Patency was defined as continued presence of palpable pulses or maintenance of postoperative ankle-brachial index ± 0.15. Statistical methods included χ², t test, Fisher’s exact test, and Kaplan-Meier plots with log-rank comparison.

Results: A total of 56 popliteal artery aneurysm repairs were performed. All endovascular popliteal artery aneurysm repairs (EVPAR, n = 15) were performed using Viabahn endoprostheses. Patients with open repair (OR, n = 41) underwent surgical bypass and aneurysm exclusion with great saphenous vein (n = 26), short saphenous vein (n = 3), or polytetrafluoroethylene (n = 12), through either a medial (n = 28) or posterior (n = 13) approach. All urgent cases received open repair. Technical success was 100% in both groups. Mean follow-up was 16.5 ± 3 months (range, 0.5 to 56 months). Aneurysm size, location, and outflow were similar between groups. Primary patency, secondary patency, and survival did not differ between groups. Endoleaks were observed in three (20%) of 15 endovascular cases, and type I and III endoleaks were treated with additional endografts.

Conclusion: To our knowledge, this represents the largest United States series of EVPAR to date. Early mid-term results of elective endovascular repair of popliteal artery aneurysms are encouraging. Further studies are warranted to define optimal indications for EVPAR and to generate long-term outcomes for this technique. (J Vasc Surg 2007;45:505-10.)

Popliteal artery aneurysm (PAA) is the most commonly encountered peripheral arterial aneurysm. Owing to high rates of thromboembolic complications, PAA confers significant risk of limb loss, and surgical exclusion has been advocated for aneurysms displaying mural thrombus or >2 cm in diameter. Marin et al first described the use of a covered stent as a means of PAA repair in 1994. Although early reports detailed the technical feasibility of endovascular popliteal aneurysm repair (EVPAR), outcomes with first-generation devices were poor. The subsequent introduction of expanded polytetrafluoroethylene (ePTFE)-lined nitinol stents, such as the Viabahn endoprosthesis (W. L. Gore & Assoc, Inc, Flagstaff, Ariz), has renewed interest in the potential application of these devices to EVPAR.

This report reviews our 6-year experience with both EVPAR and open repair (OR) of PAs at Washington University. Indications, technique, and early mid-term results for our EVPAR experience are compared with contemporaneous OR outcomes.

METHODS

A retrospective review was performed of all patients who underwent PAA repair at Washington University from January 2000 to March 2006. The study design and protocol was approved by the institutional Human Studies Committee. Hospital records, clinic notes, and vascular laboratory reports were used to compile a database of all PAA repairs. Procedures were grouped by method of repair (EVPAR vs OR). The two groups were then compared, with graft patency as the primary end point. Secondary end points included complication rates, frequency of endoleak, and overall survival.

Indications, complications, endoleak characterization, patency rates, and survival are reported in accordance with recommended standards. Symptomatic patients included those experiencing claudication, rest pain, embolic stigmata, or venous hypertension attributed to the PAA. Urgent procedures were those undertaken after the minimum necessary preoperative studies; in general, urgent operation was dictated by a perceived limb threat. Outflow was characterized as the number of tibial vessels intact from origin to ankle. Patency was defined as continued presence of palpable pulses or maintenance of a postoperative ankle-brachial index (ABI) with a change of <0.15.

Statistical analysis. Statistical analysis was performed using SPSS software (SPSS, Inc, Chicago, Ill) for Windows (Microsoft Corp, Redmond, Wash). Patient characteristics, indications, and complications were compared using Pearson χ² analysis. Mann-Whitney tests were used for nonparametric comparison of means. Patency and survival rates were compared using Kaplan-Meier plots and log-rank analysis. Statistical significance was accepted at P < .05.

Endovascular popliteal aneurysm repair technique. EVPAR was accomplished using Viabahn endoprostheses.
to exclude the aneurysm sac. Viabahn endografts are available in diameters of 5 to 13 mm, and lengths of 2.5 to 15 cm. The introducer sheath size required for graft placement ranges from 8F to 12F. Computed tomography (CT) angiography was the favored preoperative imaging modality.

Although rigid guidelines for EVPAR technique were not established at the outset of our experience, proximal and distal landing zones of nonaneurysmal artery of ≥2 cm length were sought, with the distal terminus of the endograft(s) not extending beyond the popliteal artery. Endografts were oversized 10% to 15% relative to the landing zones. When sequential endografts were required to achieve aneurysm exclusion, 2 to 3 cm of overlap between endografts was generally used. Overlapping endografts differed by no more than 2 mm in diameter, because greater differences in diameter may lead to longitudinal pleating of the larger graft, with loss of seal.

Arterial access was accomplished by a mini-cutdown to the common femoral or proximal superficial femoral artery, percutaneous antegrade approach, or percutaneous retrograde crossover approach. Because the sheath sizes required for repair preclude the use of most closure devices, the small cutdown permits placement of a transverse mattress suture at the intended antegrade arterial puncture site. Intraoperative imaging of landing zones with a 0.018-inch guidewire-compatible intravascular ultrasound probe (Volcano, Rancho Cordova, Calif) was frequently used to complement intraoperative angiography. Figs 1, 2, 3, and 4 show intraoperative angiograms from a representative EVPAR procedure.

**RESULTS**

A total of 43 patients underwent PAA repair in 56 limbs. Of these, 30 patients underwent OR in 41 limbs, and 13 patients underwent EVPAR in 15 limbs. Patient characteristics are summarized in Table I. The patients treated for PAA were primarily men in the seventh and eighth decades of life. Patient groups differed only in respect to age. The EVPAR group was a mean 7 years older than the OR group (75 ± 1.6 vs 68 ± 2.4, *P* < .05). Rates of comorbidities were high and similar between groups (Table I). A contralateral PAA was present in 70% of patients.

A significantly higher percentage of OR cases (54%) were performed for symptomatic patients vs EVPAR (15%...
P/H11021.05), and the five urgent cases received OR (12% vs 0% EVPAR, P = NS). Anatomic variables related to aneurysm size and tibial outflow vessels (Table II) did not differ significantly between groups. Of note, approximately one third of patients in each group had single vessel runoff at the time of surgery.

All EVPAR cases were performed using the Viabahn endoprosthesis. Open cases (n = 41) underwent surgical bypass and aneurysm exclusion with great saphenous vein (n = 26), short saphenous vein (n = 3), or PTFE (n = 12) through either a medial (n = 28) or posterior (n = 13) approach. Outflow thrombectomy or thrombolysis was performed in eight (15%) cases and did not differ between groups (7% EVPAR vs 17% OR, P = NS). In two EVPAR procedures (13%), dilation of stenosis adjacent to the PAA was performed before endograft delivery. The dilated area was subsequently covered by the deployed endograft. Technical success was 100% in both groups.

Although no EVPAR patients were anticoagulated, 22% of the OR group was discharged on warfarin therapy (P = .05). Indications for warfarin therapy included long-term treatment of intracranial atherosclerosis or atrial fibrillation (44%) and a new diagnosis of hypercoagulable state (22%). Use of warfarin therapy did not appear to influence patency rates. Nearly all EVPAR patients (87%) were treated with clopidogrel, compared with 11% of the OR group (P = .05). Mean length of stay was significantly shorter in the EVPAR group (0.9 ± 0.2 days vs 4.9 ± 5.6 days). Comparative treatment data are summarized in Table III, and additional data on EVPAR are provided in Table IV.

Major complications occurred in 7% of patients in both groups. One EVPAR patient experienced femoral puncture site bleeding that required suture repair; this patient had received percutaneous antegrade arterial access with 9F sheath placement. Wound abscesses developed in three OR patients that required intervention, and one of these infections resulted in sepsis and death.

Mean follow-up was 14 ± 3 months for EVPAR and 17 ± 3 months for OR (total mean, 16.5 ± 3 months; range, 0.5 to 56 months). At 24 months, primary patency...
(83% ± 15% vs 88% ± 6%, P = NS), secondary patency (100% vs 92% ± 5%, P = NS), and survival (90% ± 9% vs 90% ± 6%, P = NS) did not differ between the EVPAR and OR groups, respectively (Figs 5, 6, and 7). Comparison of only electively performed repairs (Fig 8) also demonstrated no difference in secondary patency at 24 months between OR (92 ± 8%) and EVAR (100%).

Endoleaks have been detected in three (20%) of 15 EVPAR patients. A combined type I and type III endoleak owing to endograft migration was found in one patient. This was resolved by deployment of additional endografts. Type II endoleaks were found in two additional patients and were followed up conservatively, with shrinkage of one PAA and no change in the other.

**DISCUSSION**

Acute ischemia is the first presenting symptom in 20% to 50% of patients with PAA. Continued observation of asymptomatic PAA is associated with complication rates of 15% to 25% at 1 year and 60% to 75% at 5 years.5-6 To prevent the limb loss and morbidity engendered by thromboembolic complications, early repair of asymptomatic aneurysms exceeding >2 cm or containing mural thrombus is recommended.7 This has traditionally been accomplished with elective open surgical repair of PAA and has yielded 5-year secondary patency rates of 60% to 80%.8-13 Factors affecting patency and limb salvage include the number of

<table>
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<th>Variable</th>
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<tr>
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<td>Endograft size (mm)</td>
<td>8 (6-11)</td>
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<td>IVUS imaging</td>
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*Data are presented as n (%) for categoric variables and median (range) for continuous variables.

**Table IV. Intraoperative endovascular popliteal aneurysm repair variables**

**Fig 5.** Kaplan-Meier analysis of primary patency of open repair (OR) compared with endovascular popliteal aneurysm repair (EVPAR). Broken lines indicate SEM >10%.

**Fig 6.** Kaplan-Meier analysis of secondary patency of open repair (OR) compared with endovascular popliteal aneurysm repair (EVPAR).

**Fig 7.** Kaplan-Meier analysis of patient survival after undergoing open repair (OR) compared with endovascular popliteal aneurysm repair (EVPAR).

**Fig 8.** Secondary patency of endovascular repairs (EVPAR) compared with only the elective open repairs (OR) showing no significant difference between groups.
patent tibial outflow vessels, urgency of repair, and conduit choice.

Although open surgical repair effectively mitigates the risk of thromboembolic complication, investigators have sought to adapt endovascular technology to PAA repair. Howell et al. repaired 13 aneurysms using the Wallgraft endoprosthesis (Boston Scientific, Natick, Mass), but thrombosis was detected in 31% at 12 months’ follow-up. More recently, Tielliu et al. reported a series of 57 cases in which EVPAR was performed using Viabahn stent grafts. Technical success was 100%. Primary and secondary patency rates at 2 years were 77% and 87%. Acute ischemia was the indication for PAA repair in five (8.8%) of 57, and only one third of patients received postprocedural antiplatelet or anticoagulation therapy. The only variable found to be associated with success was treatment with clopidogrel.

Antonello et al. published a prospective randomized trial comparing 15 OR and 15 EVPAR in patients with PAA ≥2 cm, adequate proximal and distal landing zones, and acceptable outflow. These patients were significantly younger than in the current series, with a mean age of 63, and all were suitable candidates for open repair. With primary and secondary patency rates of 87% and 100% at 24 months, the results in this randomized group were remarkably similar to those in the current report.

CONCLUSION

This retrospective review demonstrates encouraging mid-term results of EVPAR in a small group of selected patients. Reasonable patency rates were achieved, and no instances of limb loss occurred. However, several weaknesses of the study necessitate caution at this juncture:

1. Study design and duration. As noted, the relatively low numbers of patients and early mid-term follow-up do not permit us to forecast long-term outcomes.

2. Absence of standardized inclusion and exclusion criteria. The recommendation for EVPAR was ultimately made at the surgeon’s discretion, although patient variables (other than advanced age) favoring the use of EVPAR included absent or inadequate saphenous vein conduit (33%), simultaneous or recent other major surgery (27%), and asymptomatic contralateral lower extremity occlusive disease (20%). All urgent cases and most symptomatic cases underwent open repair, likely reflecting reluctance on the part of our surgeons to attempt this new technique on the subset of patients at greatest risk for limb loss. Therefore, although the patency outcomes in Figs 5 to 8 are of interest, the heterogeneous nature of patient groups studied mandates caution in their interpretation.

Anatomic contraindications to EVPAR have not been well defined, but landing zones diameters >12 mm and <4 mm would not be expected to offer adequate seal with the available Viabahn endoprostheses. Likewise, wide variation in the diameters of the proximal and distal landing zones may preclude an appropriate telescoping of grafts, particularly when the length of aneurysm to be covered is short. Finally, within our group, predicted terminus of repair within a tibial artery, or covering the orifice of a patent tibial artery, is felt to negate consideration for EVPAR.

3. Variables related to procedure and endograft. Variables such as the length of the landing zone and overlap between components were not standardized; indeed, the appropriate lengths in each instance have yet to be rigorously established. Such variables can affect endograft performance: in the solitary EVPAR that developed type I and III endoleaks, a review of radiographic images suggests that the initial distal landing zone and overlap between endograft components may have been as little as 1 cm in length. Long-term durability of the Viabahn endograft in popliteal aneurysms, including the rate and clinical significance of nitinol component fractures, remains unknown.

The current series should not be misrepresented as an endorsement of the unrestricted use of EVPAR. Further studies will be needed to define the optimal role and candidates for this technique.

AUTHOR CONTRIBUTIONS

Conception and design: MA, PG, ET
Analysis and interpretation: MA, PG, GS
Data collection: MA, PG, OM, RV
Writing the article: MA, PG, RV
Critical revision of the article: MA, PG, OM, RV, BG, LS, EC, GS
Final approval of the article: MA, PG, OM, RV, BG, LS
Statistical analysis: MA, OM, PG
Obtained funding: Not applicable
Overall responsibility: PG

REFERENCES


Acute Limb Ischemia Due to Popliteal Artery Aneurysm: A Continuing Surgical Challenge

William P. Robinson, III, MD, and Michael Belkin, MD

Up to 50% of all popliteal artery aneurysms (PAA) present with acute limb ischemia (ALI). ALI due to PAA is a difficult surgical problem, with a 20% to 60% incidence of limb loss and up to 12% mortality reported in the literature in the last three decades. Imminent limb threat requires emergency infrainguinal reconstruction, preferably with autogenous conduit. ALI due to PAA is limb-threatening, often due to obliteration of the tibial vessels in addition to thrombosis of the PAA itself. Arteriography is needed to define inflow vessel and outflow vessel anatomy followed by thrombectomy of the run-off vasculature to establish an appropriate target for bypass. Patients without evidence of neurologic deficit are best served by formal arteriography. Intraarterial thrombolysis is used to establish an outflow vessel for bypass if no runoff vessels are visible. In general, emergency operations are associated with inferior patency and limb salvage compared to elective procedures. Endovascular exclusion of PAA with covered stent graft is used increasingly in the elective setting and has been reported in patients presenting with limb ischemia. The following discussion outlines our algorithm in managing ALI from PAA and reviews management decisions and results of treatment.

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Overview of Popliteal Artery Aneurysms

Popliteal artery aneurysms (PAA) have a prevalence of less than 1.0%. Nevertheless, they are the most common peripheral arterial aneurysm. It is accepted that a popliteal artery greater than 15 mm is, by definition, aneurysmal, although aneurysms greater than 10 cm in size have been reported. They occur almost exclusively in men (96%) and are discovered most commonly in the 6th and 7th decades of life. Patients with PAAs frequently have other arterial aneurysms, ie, 50% to 64% will have a contralateral PAA, 40% to 62% will have an abdominal aortic aneurysm (AAA), 40% and 40% will have a femoral aneurysm. Conversely, patients with a known AAA have been reported to have an incidence of PAA of 1% to 14%. PAAs are generally degenerative in etiology, although familial, mycotic, syphilitic, and traumatic popliteal aneurysms have been described.

The natural history of PAAs is variable. Approximately 55% to 66% of PAAs are symptomatic, with lower extremity ischemia from either acute or chronic thrombosis and/or distal embolization accounting for 85% of symptomatic cases. In addition to acute limb threat, these patients commonly present with claudication, rest pain, ulceration, or "blue toe syndrome." A small number of symptomatic patients present with compressive symptoms (eg, pain, deep venous thrombosis) due to aneurysm expansion and a small minority present with aneurysm rupture. Asymptomatic PAAs cause complications in 15% to 25% at 1 year and 60% to 75% at 5 years, if left untreated. Although consensus has not been reached, most authors recommend repair of asymptomatic aneurysms when they reach 2 cm in diameter or possess significant mural thrombus, given the significant rate of limb loss and mortality and inferior results generally reported with emergency repair.

The most dreaded complication of PAA, acute limb ischemia, is reported to occur in 17% to 46% of cases. One large national registry (Swedish Vascular Registry) confirmed that of 743 patients who underwent repair during a 15-year period, 235 presented with acute ischemia. Acute limb ischemia (ALI) due to PAA is an ominous problem for even an experienced vascular surgeon; this subset of patients with ALI will be the focus of this discussion.
Pathophysiology of ALI due to PAA

Thromboembolism from PAA can be a progressive process in which the popliteal artery and crural arteries are obliterated over time. ALI may be brought on by acute thrombosis of the popliteal artery. Ninety percent of patients have abnormalities in their tibial arteries, with 22% to 38% of patients having only single vessel runoff and 17% having no tibial vessels in continuity with the pedal arch. Bouhoutos and Martin reported that 34% of patients who present with aneurysm thrombosis have occlusion of their tibial vessels. Marty et al reported that patients who presented with grade Ila ischemia due to PAA had arteriographic occlusion of their PAA and absence of runoff in 12 of 13 patients (including 10 with no visualization of tibial vessels). Lack of a visible runoff vessel leaves the vascular surgeon with no target for revascularization and puts the patient at particularly high risk for limb loss.

Clinical Presentation of ALI due to PAA

Patients with ALI present with abrupt onset of foot coolness, foot or leg pain, and/or numbness. Careful questioning may elicit a history of recent claudication associated with compromise of popliteal and crural vessels that has progressed to limb threat, with occlusion of the popliteal artery, and/or remaining runoff. As in ALI of other etiologies, physical exam reveals a cold limb with diminished or absent distal pulses. Deficiencies in sensation and motor strength may be present depending upon the duration and severity of ischemia. Most patients present with grade Ila or Iib ischemia. A diagnosis of PAA is suggested by the presence of a prominent popliteal pulse, although a pulse often will not be palpable due to thrombosis. A prominent contralateral popliteal pulse or aortic pulsation should also raise the suspicion for PAA as the etiology of ischemia.

Diagnosis of Popliteal Artery in Setting of ALI

ALI in the clinical setting mentioned here should arouse suspicion of PAA. In this scenario, duplex ultrasound can be used to examine both the ipsilateral and contralateral popliteal arteries (Fig 1). Duplex ultrasound is more sensitive than physical examination in diagnosing PAA. Furthermore, it provides information on the presence and velocity of flow, presence of mural thrombus, and patency of outflow arteries. Computed tomographic arteriography and magnetic resonance arteriography are useful adjuncts because they provide information on outflow and inflow as well as provide imaging of the artery in the popliteal space (Fig 2). To our knowledge, specific data regarding the utility of any of these modalities in the setting of ALI have not been published. Their usefulness would be dependent on their immediate availability in a particular center. The availability of duplex scanners in emergency rooms and their increasingly facile use by emergency physicians, house staff and fellows, and vascular surgeons is helpful for rapid diagnosis. Use of these modalities should not impede prompt revascularization in cases of limb threat.

Diagnosis of PAA in the setting of ALI is helpful because thrombosed PAA is not amenable to standard balloon catheter thromboembolectomy. Furthermore, a diagnosis of PAA will lead to a workup for concomitant aneurysms, help the surgeon develop a revascularization strategy, and provide prognostic information to the patient. However, the diagnosis may not be known at the time treatment is initiated and is...
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not essential in the setting of ALI. On-table arteriography may show ectasia of the superficial femoral artery (SFA) and popliteal artery suggestive of a PAA, but will likely demonstrate only popliteal occlusion with or without compromised infrageniculate flow (Fig 3).

**Natural History of ALI Due to PAA**

Acute thrombosis of a PAA without development of a collateral network makes this a particularly dangerous entity, demanding prompt revascularization in the physiologically stable patient. Management without attempts at revascularization results in above-knee amputation in up to 43% of patients.\(^1\,^2\,^3\,^4\,^5\,^10\,^15\,^22\,^24\)

While a heparin bolus (80 U/kg) followed by continuous infusion (18 U/kg/h) should be initiated in ALI in the absence of contraindications, anticoagulation alone would not be expected to be limb-saving. However, anecdotal evidence suggests preventing propagation of clot may impact the ability to preserve the knee joint should limb loss ensue.

**Surgical Approach to ALI Due to PAA**

**Ischemia with Neurologic Deficit**

As in any patient with ALI, the degree of ischemic insult determines the course of management (Fig 4). Patients with evidence of sensory loss or motor weakness, Rutherford class IIb and III ischemia, are in need of urgent revascularization and must proceed immediately to the operating room. These patients cannot tolerate additional ischemic time required by arteriography and thrombolysis. If PAA is either known or suspected based on preoperative studies, the focus should be on identifying an inflow vessel and a distal target for bypass with runoff to the foot. We prefer to begin with lower extremity arteriography via a percutaneous femoral approach. This often demonstrates abrupt occlusion of the popliteal artery with varying degrees of obstruction of the infrageniculate vessels (Fig 5A). In contradistinction to acute embolism, the distal superficial femoral and popliteal arteries are generally ectatic with luminal thrombus. If a patent runoff vessel is identified with flow to the ankle, we then proceed to bypass.

Inflow is generally provided by the distal SFA via a medial approach. The SFA can often be utilized as inflow unless there is evidence of severe ectasia or coexistent atherosclerotic disease throughout the vessel, which requires use of the proximal SFA or common femoral artery as inflow. Patency of SFA-origin bypass equals that of common femoral origin,\(^25\) and a shorter segment of saphenous vein can be harvested from the distal thigh, often through extensions of the same medial incisions.

In most cases, a below-knee medial approach is then utilized to expose the distal popliteal artery, tibioperoneal trunk, or proximal posterior tibial artery for the distal spatulated end-to-side anastomosis. This exposure also provides access to the origins of the tibial and peroneal vessels should thromboembolectomy and/or intraoperative thrombolysis be required. Aneurysm exclusion with proximal and distal ligation above and below the knee can easily be accomplished with this medial exposure.

Autogenous vein, including arm vein if necessary, should be used preferentially because it has superior patency in comparison to prosthetic conduit.\(^13\,^15\,^26\,^27\) A comprehensive review reported 5-year patency of synthetic bypasses ranging from 29% to 74%, while that of autologous vein was 77% to 100%.\(^28\)

A distinctive feature of ALI due to PAA is the severely compromised infrageniculate flow. If intraoperative arteriography fails to demonstrate a patent infrageniculate vessel with runoff to the ankle, exploration and balloon catheter thrombectomy should be performed in an attempt to establish a target for bypass. Thrombectomy via a femoral or above-knee popliteal approach provides little chance of success given the
thrombus burden in the PAA and runoff vessels. Trifurcation embolectomy via a medial incision is the first alternative (Fig 5B and C). Thrombectomy via arteriotomy in the posterior tibial or distal anterior tibial arteriotomy at the ankle may also be required to clear outflow.29,30 Balloon thromboembolectomy must be performed carefully in these small-caliber vessels. Injections of intraarterial recombinant tissue plasminogen activator) may be a useful adjunct in this scenario (see report on intraoperative, intraarterial thrombolysis later in this issue). If flow can be established in a runoff vessel, we then proceed to bypass as mentioned here. Surgeons should use either duplex ultrasonography and/or completion arteriography to evaluate the bypass graft for technical error and presence of outflow to the foot. If a target cannot be established, the operation is terminated and anticoagulation with heparin continued; observation is undertaken to evaluate proper demarcation to determine an appropriate amputation level. Amputation is then performed at a second operation as dictated by clinical and physiologic findings.

In some instances, a patient with ALI in which the diagnosis of PAA is not known or suspected may be taken to the operating room for femoral exploration and thromboembolectomy. Failure to pass a Fogarty balloon past the popliteal artery should arouse suspicion for PAA. At this point, arteriography should be performed. As discussed, an enlarged or ecstatic SFA with occlusion of the popliteal artery strongly supports diagnosis of PAA. The necessity of four-compartment lower extremity fasciotomy should be determined based upon clinical examination and the length and degree of ischemia as in ALI of other etiologies.

**Ischemia without Neurologic Deficit**

If the patient has acute ischemia but no neurologic deficit (grade I, IIa ischemia), we anticoagulate with heparin and proceed to formal arteriography via the contralateral common femoral artery. If arteriography demonstrates an inflow source and suitable outflow, our preference is expeditious infrainguinal reconstruction with autogenous vein.

If the popliteal artery is thrombosed and there is no visible run-off vessel, intraarterial thrombolysis is initiated to restore patency to at least one tibial vessel (Fig 3). The technique of thrombolysis will be described.
Technique of Thrombolysis

A 4Fr multihole catheter is positioned within the proximal thrombus of the PAA via an up-and-over maneuver from contralateral femoral access. Recombinant tissue plasminogen activator is infused with a bolus of 5 to 10 mg, followed by an infusion of 0.5 to 1.0 mg per hour. The catheter is advanced distally as lysis allows. Patients are admitted to the intensive care unit for monitoring of the ischemic limb, access site, evidence of hemorrhage, and neurologic status. Continuous anticoagulation with heparin for a goal partial thromboplastin time of 60 seconds is maintained. Patients are returned to the catheterization lab every 6 to 12 hours for repeat arteriography. When a flow channel through the PAA is achieved, the catheter is advanced over a guide wire into the distal popliteal artery for perfusion of recombinant tissue plasminogen activator into the tibial arteries. We terminate lysis when a single tibial artery in continuity with the pedal arch is demonstrated or there has been no progress in recanalization. The patient is then taken for expeditious infragenual reconstruction as described in this article. Thrombolysis should be terminated if hemodynamic instability, change in mental status, or bleeding requiring transfusion occurs.

If the patient shows progression in limb ischemia or failure of lytic therapy to recanalize outflow, we then proceed to trifurcation and/or ankle-level tibial thrombectomy, followed by attempts at bypass grafting as described here. Failure to establish a bypass target by either lytic or operative means may necessitate amputation at a time dictated by the patient’s clinical condition.

Results of Surgical Therapy

Results of operative repair of PAA in the setting of ALI vary greatly. Graft patency in the setting of ALI is difficult to decipher accurately because these patients are few in number and often included as part of a larger report of patients with chronic symptoms or subacute ischemia. Five-year and 10-year patency rates are in the range of 39% to 68%, and 60%, respectively. The incidence of limb loss in ALI has been reported as high as 20% to 60%, with mortality rates reported between 5% and 12%. More recently, groups have reported good results with repair of PAA in the setting of ALI. Mahmood et al report a 1-year and 5-year secondary patency of 94% and...
80% in 13 patients with ALI who underwent immediate surgical repair. Limb salvage was 94% at 1 year and 80% at 5 years. Their approach was one of trifurcation and/or ankle-level thromboembolectomy in conjunction with autogenous bypass grafting and exclusion via a medial approach. They concluded that acute ischemia and bypass to crural vessels were not predictive of graft failure. Aulivola et al.26 report comparable results of popliteal aneurysm repair in the 14 emergent and 37 elective limbs. Initial arteriography demonstrated an average of 1.1 patent runoff vessels in ALI patients and 2.2 runoff vessels in elective repairs. The emergent group had a primary and secondary patency of 85% and 100% at both 1 and 5 years, which was similar to a primary and secondary patency of 85.6% and 95.7% at 5 years in the elective group. Limb salvage at 5 years was 93% in the emergent group and 100% in the elective group. It should be noted, however, that all three patients presenting with absence of pedal signals underwent thrombolyis to uncover a bypass target with success in two of three patients. This report supports the approach of immediate bypass grafting if there is at least one-vessel distal runoff and the reservation of thrombolysis for limbs without a target.

Elective repair can be accomplished via either a medial approach as described or a posterior approach with aneurysmorrhaphy or resection and interposition grafting. The posterior approach is suboptimal in the setting of ALI because it precludes arteriography and great saphenous vein harvest. It should be noted, however, that continued expansion and progressive compressive symptoms or rupture have been reported post-procedure due to failure to ligate feeding vessels with the medial approach.34,35 Therefore, postoperative ultrasound surveillance of the excluded aneurysm is warranted.

Results of Elective PAA Repair
Overall results are superior in elective repair of asymptomatic patients compared to results in patients with acute or chronic symptoms.2,5,10,18,32,33,36,37 Five-year graft patency rates in excess of 85% have been reported by multiple groups in elective patients.18,26,27,32,36,38,39 At our institution, a 5-year primary patency of 92% ± 4% for vein grafts performed for PAA has been superior to that in patients with occlusive disease, a finding potentially related to beneficial graft remodeling in these patients.39 Limb-salvage rates in excess of 90% are standard in asymptomatic patients.1,10,13,15,20,27,32,37,38 Perioperative mortality is reported between 0 and 2%.20,32,39

Results of Thrombolytic Therapy for ALI due to PAA
Thrombolytic therapy for thrombosed PAs was introduced in 1984.40 Embolization and “packing-in” of thrombus in the runoff vessels is believed to account for the 20% to 40% major amputation rate for ALI due to PAA that was reported in some studies.10,16,18 The rationale for thrombolysis before operation, therefore, is primarily to restore run-off for bypass grafting. Proponents also argue that there is a benefit to gradual reperfusion, which minimizes reperfusion injury and the ability to uncover underlying arterial lesions before bypass. Reports on thrombolytic therapy all include fewer than 14 patients, with the exception of a series of 100 patients from the Swedish Vascular Registry. Results of published series are summarized in Table 1.

The value of preoperative lysis lies in the ability to clear thrombus from the runoff vessel(s). One- or two-vessel runoff can be established in 77% to 100% of patients, including those in which all runoff vessels are occluded. In 1994, Carpenter et al.10 reported complete clearing of all runoff vessels in six of seven patients (and clearing of two vessels in the remaining patient) with ALI and thrombosis of PAA. These patients had 100% early graft patency and limb salvage, which was superior to comparable patients treated with emergency operation. Other studies have confirmed similar success (Table 1). Overall, early graft patency of 68% to 100% and limb-salvage rates of 73% to 100% compare favorably to the results of emergent open revascularization in most series, but are similar to the results reported by Mahmood and Aulivola.2,26

Conclusions from these studies must be drawn with caution. These series suffer from small numbers of patients and inevitable selection bias. Quite appropriately, patients selected for thrombolytic therapy in these series presented almost exclusively with Rutherford class I or IIa ischemia. Successful lysis generally requires 24 to 48 hours with repeated trips to the catheterization lab for catheter positioning. Patients with neurologic deficit should proceed immediately to operation as they cannot tolerate the additional ischemic time required by preoperative lysis.

Complications and Failure of Lytic Therapy
Thrombolysis is associated with major risks, including bleeding, intracranial hemorrhage, and stroke. The rate of bleeding complications is reported between 0 and 25% (Table 1). Furthermore, there is a risk of distant thromboembolism reported in up to 13% of patients,41 an occurrence that could accelerate ischemia in previously stable patients. This is generally treated with continuing or increasing the rate of infusion.42 Furthermore, thrombolysis may fail in up to 33% of patients.20,43 This is likely the result of organized, “packed-in” clot in the runoff arteries from chronic embolization. Thrombolitics are known to be less effective in lysing older, organized thrombus.24 These patients often go on to amputation as attempts at operative thrombectomy and bypass typically fail. One group had reported that the primary benefit of thrombolysis is to identify patients who should undergo primary amputation.20 While this approach seems applicable to patients who are at high medical risk for multiple operations, we feel that operative thrombectomy and bypass should be attempted in most patients before committing to amputation. Furthermore, with development of hybrid operating rooms with high-quality fixed imaging and a full complement of endovascular devices, open and endovascular techniques can be combined more effectively.
Endovascular Popliteal Aneurysm Repair

Endovascular repair of PAAs with covered stent grafts in the elective setting has produced good short-term results. Two-year primary and secondary patency rates of 77% to 87% and 87% to 100%, respectively, have been reported.44-46 Results at 5 years are limited, but the primary and secondary patency rates of 70% and 76% that have been reported appear inferior to that of open repair.47 To our knowledge, only one existing report has included patients with ALI (five patients with Rutherford class Ila ischemia).46 At this stage, endovascular repair in the setting of ALI cannot be recommended. It would only be possible after thorough lysis of popliteal artery occlusion to allow passage and deployment of the stent graft through the aneurysm. However, further investigation of this therapy in patients who would require below-knee bypass with prosthetic conduit due to lack of autogenous vein or those medically unfit for operation is warranted.

Conclusions

In summary, ALI from PAA remains a challenging surgical disease, which demands excellent clinical judgment and aggressive and meticulous surgical management. The last two decades have seen considerable growth in the tools available to vascular surgeons to diagnose and treat this condition. Operative and thrombolytic therapy should be seen as complementary techniques best managed by a vascular surgeon experienced with the natural history and surgical management of ALI and PAA. Good bypass graft patency and limb salvage can be achieved with proper patient selection for primary operative and thrombolytic therapy. The challenge of ALI due to PAA underscores the importance of repair in the elective setting when possible.

References

Visceral Artery Aneurysms: Review of Current Management Options

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ABSTRACT

Visceral artery aneurysms are relatively rare clinical entities, although their detection is rising due to an increased use of cross-sectional imaging. Rupture is the most devastating complication, and is associated with a high morbidity and mortality. For this reason, elective repair is preferable in the appropriately chosen patient. In general, splenic artery aneurysms measuring 2 cm or larger and those found in women of childbearing age and in persons undergoing liver transplantation should be treated. Hepatic artery aneurysms 2 cm or larger and those that are multiple or nonatherosclerotic in nature should be repaired in the appropriate patient due to a higher risk of rupture. Endovascular coil embolization has excellent success rates and is the first-line treatment for anatomically suitable splenic artery aneurysms and intrahepatic hepatic artery aneurysms. However, reperfusion is an important complication of endovascular management. Aneurysms involving the celiac, superior mesenteric, pancreaticoduodenal, gastroduodenal, and inferior mesenteric arteries, as well as visceral artery pseudoaneurysms, are unpredictable and should be repaired in the appropriate medical patient. These aneurysms are often amenable to ligation due to the presence of collateral circulation. Endovascular management is particularly useful in the treatment of pseudoaneurysms where comorbidities and previous surgery make open surgical repair less desirable. Mt Sinai J Med 77:296–303, 2010. © 2010 Mount Sinai School of Medicine

Key Words: aneurysm, endovascular coil embolization, splanchnic circulation, visceral artery.

Visceral artery aneurysms (VAA) are uncommon entities that affect the splenic, hepatic, celiac, and superior mesenteric arteries. Increased imaging of the abdomen with ultrasound, computed tomography (CT), and magnetic resonance imaging (MRI) to evaluate nonvascular pathology has led to a concomitant increase in their diagnosis in asymptomatic patients. Elective repair is recommended in certain situations to prevent the high morbidity and mortality that is associated with aneurysm rupture.

Conventional repair of VAA has included excision, bypass, ligation, and end-organ resection. Endovascular techniques have gained popularity and have become the first-line treatment for certain types of aneurysms, such as those that affect the mid- to proximal splenic artery. Finally, laparoscopic techniques have been used with excellent results, and have allowed for the treatment of patients who would otherwise be unfit for open surgery due to comorbidities. This article will systematically review the natural history (when known), management
recommendations, and current treatment options for aneurysms involving branches of the celiac and superior mesenteric arteries, with particular emphasis on endovascular therapies. Management of visceral artery pseudoaneurysms also will be discussed.

**SPLENIC ARTERY ANEURYSMS**

Splenic artery aneurysms (SAA) account for about 50% to 75% of all VAA,\(^1\)\(^–\)\(^6\) are estimated to have a prevalence of <0.1%, and may be associated with other intra-abdominal aneurysms involving visceral (3%) and renal (14%) arteries.\(^4\) They have a female to male predominance of 3–4:1\(^5\)\(^–\)\(^7\) and are associated with multiparity.\(^5\) In a review of 217 patients with SAA, mean age was 61 years, 79% were women, 95% of the aneurysms were solitary, and the majority were asymptomatic.\(^3\) However, rupture was the presenting symptom in close to 5% of patients, with a mean diameter of 3.5 cm (3.2 cm for males, 2.3 cm for females).\(^3\) In contrast, the mean diameter of the nonruptured SAAs was 2.2 cm. Interestingly, calcification, which is often thought to offer evidence of chronicity and stability, was seen in 90% of the ruptured aneurysms;\(^3\) and should therefore not be used to guide management decisions.

True SAAs are associated with multiparity as well as portal hypertension.\(^4\)\(^,\)\(^5\)\(^,\)\(^7\) Hormonal changes and increased portal congestion associated with pregnancy are thought to lead to loss of structural integrity within the splenic artery, leading to aneurysm formation.\(^1\) SAA has a prevalence as high as 10% in patients with portal hypertension,\(^4\) and aneurysm rupture after liver transplantation is not an uncommon outcome. In a series of 30 patients who underwent repair of SAA at a busy liver transplant center, 20 patients had a previous orthotopic liver transplant (OLT) and 12 patients had a history of portal hypertension. Rupture was an indication for repair in almost half the patients in this study, and occurred within 2 weeks of liver transplantation in 7 patients.\(^7\) Other etiologies of SAA include systemic hypertension, as well as less-common conditions such as medial fibrolydysplasia and \(\alpha-1\) antitrypsin deficiency.\(^4\) Although calcification is commonly seen within them, atherosclerosis does not appear to be a major factor in the development of SAA.\(^4\)

The natural history of untreated SAA is poorly defined, but may be best described in the Mayo Clinic experience by Abbas et al. in which the decision was made to observe 168 patients at the initial presentation of SAA.\(^5\) Sixty-five percent of these patients had aneurysm size documented on presentation, and 47% had size information available at follow-up. Mean aneurism size at presentation in the nonoperative group was 2.1 cm, with approximately one-third of those patients having aneurysms between 2 and 3 cm. Of the 79 patients in whom follow-up measurements were obtained, 11 (14%) had evidence of aneurysm growth; of these, 3 underwent elective repair. None of the patients who were observed ruptured their aneurysms during a mean follow-up period of 75 months (range, 1–371 months).\(^3\) To summarize natural history information as well as indications of repair, SAA can have an appreciable growth rate, and increased aneurysm size is associated with rupture. Current available data would indicate that repair should be offered for symptomatic aneurysms, aneurysms \(\geq\) 2 cm aneurysms found in women of childbearing age (because rupture in pregnant women is associated with high maternal and fetal mortality),\(^8\) and in patients with SAA who are undergoing OLT. In the latter case, repair of the aneurysm at the time of transplant has been reported.\(^7\)

Treatment options for SAA include open surgical repair, endovascular management with either coils or covered stents, and, more recently, laparoscopic excision. The location of the aneurysm as well as its presentation dictates the type of open procedure that is performed. Resection with end-end repair can be performed in many cases, owing to the predominantly proximal location of SAA and to the redundancy and tortuosity of the artery.\(^5\) This allows for splenic preservation, which has important implications for the immune system. However, splenectomy may become necessary in aneurysms involving the splenic hilum,\(^5\) and is performed more commonly in the setting of aneurysm rupture.\(^5\)\(^,\)\(^5\)\(^,\)\(^7\)

Endovascular management of SAA has gained popularity over the last decade, owing to its acceptable technical success rate and low morbidity.\(^1\)\(^,\)\(^2\)\(^,\)\(^6\)\(^,\)\(^10\)\(^,\)\(^11\) Percutaneous embolization with either coils or glue (N-butyl cyanoacrylate) is particularly appropriate for saccular aneurysms, fusiform aneurysms with good collateral flow to the end organ, and aneurysms of vessels in which other sources of arterial flow to the end organ are present.\(^5\)\(^,\)\(^10\) Thus, SAA are often ideal candidates for coil embolization (Figure 1). Triaxial systems that utilize microcatheters allow for super-selective catheterization of branches to optimize aneurysm exclusion.\(^2\)\(^,\)\(^10\) Procedures can be performed under local anesthesia with sedation and are generally ambulatory in the elective setting.\(^1\) Percutaneous access is most commonly achieved through cannulation of the femoral artery.\(^2\)\(^,\)\(^10\) When there is no evidence of rupture, systemic heparin is administered to prevent thrombosis of the sheath and access vessels. Rates of successful exclusion of SAA range...
Fig 1. Angiogram demonstrating splenic artery aneurysm (A) before and (B) after successful coil embolization.

from 90% to 100%. Placement of stent grafts to maintain perfusion in the main artery has also been described with success in the splenic artery (Figure 2); however, tortuosity of the artery can complicate stent graft placement and deployment.

Complications of SAA coil embolization include splenic infarct (Figure 3) and reperfusion of the aneurysm, which can occur in 5% to 20% of patients. Thus, yearly follow-up with CT or MRI is necessary to evaluate for leak and subsequent growth. In our previously reported experience at Mount Sinai Medical Center, 3 out of 15 patients treated with endovascular coil embolization developed evidence of reperfusion. However, all were able to be treated by repeat embolization through endoluminal access, avoiding the need for laparotomy. Splenic infarcts have been seen more commonly in patients with portal hypertension and hilar location of aneurysms, but usually are successfully treated with pain control. In reported series, splenic infarcts occurred in 25% to 40% of the patients treated, yet no patient developed sequlae requiring further management.

Laparoscopic management of SAA was first reported in 1993, and though it is not a commonly performed procedure, it does offer an alternative to endovascular and open surgical management. Although the literature describes only small series, outcomes appear to be acceptable, and the procedure offers certain advantages over other treatment options. Patients are spared the need for yearly CT follow-up to evaluate for aneurysm reperfusion, which is required for endovascular management and may be problematic in young patients. Furthermore, splenectomy can be performed at the time of the procedure, which obviously prevents the syndrome associated with postembolization infarction, pain, and potential for infection. In 1 small series, splenectomy was performed when laparoscopic-assisted Doppler sonography demonstrated markedly diminished flow to the spleen after aneurysm exclusion. This occurred most commonly in hilar aneurysms.
Although, theoretically, end-to-end arterial anastomoses can be performed laparoscopically to maintain arterial perfusion, in the aforementioned small series a robotically assisted anastomosis was performed in the 1 case in which reconstruction was performed, adding to the complexity and cost of the procedure.16

HEPATIC ARTERY ANEURYSMS

Hepatic artery aneurysms (HAA) are the second most common true VAA (Figure 4). However, increased performance of percutaneous biliary procedures, nonoperative management of trauma, and liver transplantations has led to an increase in hepatic artery pseudoaneurysms,1,2,9,10,17,18 making them the most commonly reported VAA in the period from 1985 to 1995.17,19 In contrast to SAA, HAA are more commonly identified in men (male/female [M/F] ratio 3:2),4,20 although the mean age of presentation is similar (sixth to seventh decade).3,4,20 In a series of 36 true HAA reported by the Mayo Clinic, hypertension was the most commonly associated comorbidity, occurring in 72% of patients.20 Nonhepatic VAA were identified with HAA in 31% of patients and were identified in the splenic artery in 20% of patients.20 The majority of aneurysms were solitary and atherosclerotic in nature, and aneurysm rupture was seen only in patients with multiple aneurysms or with aneurysms of nonatherosclerotic etiology (eg, fibromuscular dysplasia, polyarteritis nodosa, or mycotic).20 Most aneurysms were located in the extrahepatic circulation, and fewer were seen in both the extrahepatic and intrahepatic arterial system. Pure intrahepatic aneurysms were an uncommon finding.20

Most HAA are identified as incidental findings on cross-sectional imaging or abdominal ultrasound.4 However, abdominal or back pain can be present in rapidly expanding aneurysms.4 Importantly, rupture into the biliary tree is more common than free, intraperitoneal rupture, and can present with jaundice, biliary colic, and gastrointestinal hemorrhage as is described in Quincke’s triad.4,19 In the Mayo Clinic experience, 22 patients (61%) were managed nonoperatively for aneurysms <2.0 cm (n = 14) and >3.0 cm (n = 4).20 Two of these patients presented ruptured, and 1 died. The other had contained hematoma, was observed, and was alive at 3 months follow-up. In 3 patients managed nonoperatively, aneurysm growth was noted, but repair was precluded due to either poor medical condition or aneurysm size <2.5 cm.20 None of the observed patients, including those with aneurysm diameter >3 cm, proceeded to rupture.20

Based on the Mayo Clinic experience, which represents one of the largest series of true HAA in the literature, indications for treatment include symptomatic aneurysms, nonatherosclerotic aneurysms, multiple aneurysms, and aneurysms >2 cm in good-risk patients with a life expectancy of at least 2 years. In their series, patients with significant comorbidities who presented with aneurysms >3 cm were observed, and none proceeded to rupture.20 Alternatives to open surgical management, including endovascular treatment, may allow for treatment of large aneurysms in the medically compromised patient.18

Traditional surgical management of HAA depends on the location of the aneurysm, presence of collateral flow, and medical state of the patient. Procedures can include ligation, ligation with bypass using autologous vein or prosthetic graft, hepatectomy, and, in rare instances, OLT.1,19,20 In either the open or endovascular strategy, ligation or coil embolization of the hepatic artery should be performed only if the portal vein is patent.18 Aneurysms involving the proper hepatic artery require vascular reconstruction as a component of repair, whereas

Fig 4. Angiogram demonstrating hepatic artery aneurysm.
common HAA may be managed without reconstruction if collateral flow through the gastroduodenal artery is adequate.\textsuperscript{4,5,17,18} Complications of open surgical repair include graft thrombosis, bile leak, and intra-abdominal abscess or sepsis.\textsuperscript{1,20} Open surgical repair for rupture is associated with a high morbidity and mortality (100% and 33%, respectively\textsuperscript{20}); thus, elective repair, when appropriate, is preferred.

Literature describing the use of endovascular techniques to manage HAA is limited to small series. Percutaneous embolization is considered first-line treatment for intrahepatic aneurysms owing to the complicated nature of open repair.\textsuperscript{21} Percutaneous approaches appear to have particular use in the treatment of hepatic artery pseudoaneurysms, in which previous abdominal surgery and medical comorbidity are prominent features.\textsuperscript{1,11,10,17} In our series describing the experience at Mount Sinai, an endovascular approach predominated in the treatment of hepatic artery pseudoaneurysms, whereas true aneurysms involving the extrahepatic circulation were more likely to be treated by surgical resection (n = 10:1, pseudo:true treated endoluminally versus 4:7, pseudo:true treated by open surgery; P = 0.002).\textsuperscript{1} In another series from the Cleveland Clinic, technical success at coil-embolizing 10 HAA was 80%, and failures were attributed to an inability to cannulate the aneurysm neck.\textsuperscript{10} Although follow-up imaging was not available for all patients, no reperfusion was seen in patients in whom imaging was available.\textsuperscript{10} In another small series of 12 hepatic artery embolizations, no evidence of hepatic ischemia occurred after aneurysm ablation with either coils or glue.\textsuperscript{2}

Stent graft repair may be desirable in patients with baseline hepatic insufficiency, or in aneurysms involving the proper hepatic artery in order to maintain antegrade perfusion. However, anatomic suitability for stent graft repair must be determined preoperatively to evaluate the adequacy of the seal zones, the potential for kinking of the artery, and the accessibility of proximal vessels for delivery sheaths. Reported complications of endovascular therapy include access-related problems such as groin hematomas, pseudoaneurysms, and arterial thrombosis, which can occur during any percutaneous intervention. Procedure-specific complications can also include arterial dissection\textsuperscript{21} and aneurysm rupture.

\section*{SUPERIOR MESENTERIC ARTERY ANEURYSMS}

Superior mesenteric artery (SMA) aneurysms are the third most common type of VAA, and are a rare entity. Most commonly they involve the proximal 5 cm of the SMA.\textsuperscript{22} Extension of aneurysm-related thrombus or dissection beyond the collateral circulation of the celiac and inferior mesenteric arteries may result in ischemic symptoms.\textsuperscript{23} Historically, the etiology of SMA aneurysms was infectious.\textsuperscript{22} However, in a series of 21 patients, etiologies varied from atherosclerosis and cystic medial dysplasia to collagen vascular disorders and polyarteritis nodosa.\textsuperscript{24} Medial dysplasia may be a secondary finding.\textsuperscript{25} Only 1 patient had evidence of infection. In that same series, 8 patients presented ruptured, and nearly half of the patients with rupture subsequently died.\textsuperscript{24} In contrast to SAA aneurysms, patients with calcified SMA aneurysms did not have rupture either at presentation or follow-up.\textsuperscript{24} There was no accurate description of aneurysm size in patients who ruptured, but the mean size in patients who did not rupture was 2.2 cm. Furthermore, male patients were more prone to rupture. Of the 13 patients who did not rupture, 10 were asymptomatic.\textsuperscript{24} Patients were repaired unless comorbidities precluded operative intervention. Although elective repair did not require bowel resection, repair for rupture was associated with small-bowel resection in 3 out of the 8 patients for bowel ischemia.\textsuperscript{24} Thus, elective repair may be beneficial in terms of bowel preservation. Based on limited literature, indications for repair other than rupture include size \( \geq 2 \text{ cm} \) in a good-risk patient, growth, and symptoms.

Ligation of the vessels that enter and exit the aneurysm is one of the more common surgical approaches to treat SMA aneurysms.\textsuperscript{23,24} Collaterals from the celiac and inferior mesenteric arteries are usually sufficient to prevent ischemia, although test occlusion with an intraoperative assessment of bowel viability can be performed.\textsuperscript{25} Other open surgical approaches include endoaneurysmorrhaphy and arterial reconstruction with bypass after ligation using either vein or prosthetic graft. Vein is the preferred conduit in the setting of an infected aneurysm. Operative exposure is transmesenteric in more distal aneurysms, whereas those in the proximal SMA may require exposure through medial visceral rotation.\textsuperscript{23}
Reports of endovascular management of SMA aneurysms describe the use of coil embolization and stent grafts.\textsuperscript{6,24} Percutaneous management is particularly useful in patients with a hostile abdomen and medical comorbidities. In our previously published experience, 3 patients with SMA pseudoaneurysms were successfully managed by endovascular means without procedure-related complications. Two of these patients had hostile abdomens, and 1 had medical comorbidity.\textsuperscript{10} In general, literature on these techniques is limited to very small series or case reports, thus long-term outcomes are difficult to assess.

**CELIAC ARTERY ANEURYSMS**

Celiac artery aneurysms (CAA) are quite rare, accounting for 4\% of VAA.\textsuperscript{22} The etiology of these aneurysms is usually atherosclerosis, and infectious causes are rare. Most are asymptomatic, but symptoms can mimic pancreatitis due to the location of the arteries.\textsuperscript{25} Of note, association with other aneurysms is common, occurring in 66\% of patients in 1 series.\textsuperscript{25} Concomitant aneurysms included aortic, renal, popliteal, and femoral in that report.\textsuperscript{25} In 1 series, CAA were the most common VAA encountered (9 of 23 patients), most likely because SAA were seen and treated by interventional radiologists.\textsuperscript{26}

Because these aneurysms are so rare, reports of their natural history are limited. Again, the Mayo Clinic experience represents the largest compilation of patients with CAA (n = 18), some of whom were treated and some of whom were observed.\textsuperscript{25} The patients who were observed were considered too high-risk for repair, and were thus followed with serial imaging.\textsuperscript{25} Mean aneurysmal size was 2.1 cm (range, 1.5–3.5 cm) in this group of 8 patients who were observed. One patient with a 2.5-cm aneurysm who declined repair ruptured 5 years after presentation, whereas the others did not demonstrate any sign of growth over a 91-month mean follow-up period (range: 1–371 months).\textsuperscript{25} Thus, observation in medically compromised patients is a reasonable option.

Traditional open repair can be performed through a transabdominal route, and may require medial visceral rotation of left colon, spleen, and pancreas to gain access to the proximal celiac artery.\textsuperscript{25} A thoracoabdominal approach may be required in some instances when the aneurysm is large.\textsuperscript{25} Ligation can be performed in the absence of liver pathology.\textsuperscript{25} Aneurysmectomy with arterial reconstruction using vein or prosthetic graft is another approach, using the supraceliac aorta as an inflow source.\textsuperscript{25} In the Mayo Clinic experience, vein bypasses occluded within 6 months follow-up. Kinking of the graft and thrombosis in the setting of competitive flow were proposed explanations for this finding.\textsuperscript{25} Thus, prosthetic grafts may offer some advantage in this setting.

Endovascular management of CAA has been described and may be appropriate in high-risk patients without liver dysfunction and without disease of the collateral circulation, including the SMA and gastroduodenal arteries.\textsuperscript{2,11,27} Endovascular treatment of CAA may also be performed during endoluminal repair of other VAA,\textsuperscript{1} or before treatment of aortic aneurysms.\textsuperscript{27}

Endovascular management of celiac artery aneurysms (CAA) has been described and may be appropriate in high-risk patients without liver dysfunction and without disease of the collateral circulation, including the SMA and gastroduodenal arteries.

The nature of the collateral circulation theoretically allows for embolization of the inflow, outflow, and branches of the aneurysm without arterial reconstruction. Though reports of this technique are limited in regard to CAA repair, celiac artery coverage and occlusion is sometimes utilized in endovascular repair of thoracic artery aneurysms (TEVAR). The safety of celiac artery coverage in that setting is a matter of current investigation. Most authors recommend preoperative angiography to evaluate collateral circulation,\textsuperscript{28,29} and some have advocated selective arteriography through the SMA with concomitant celiac artery test occlusion to evaluate the adequacy of mesenteric collaterals.\textsuperscript{28,30} However, in 1 study, test occlusion of the celiac and mesenteric...
angiography did not prevent foregut ischemia in the setting of celiac artery coverage. Furthermore, in the same study, coverage of the celiac artery during TEVAR led to foregut ischemic complications in 25% of patients who did not have preoperative celiac artery revascularization. Complications included an ischemic gastric ulcer, gangrenous cholecystitis, liver abscess, and exacerbation of cirrhosis. Whether the ischemic complications were due to inadequate flow or procedural embolization was an unresolved issue in that report. Complications specifically related to endovascular aneurysm exclusion do not appear to commonly involve organ ischemia, although reports are limited. Late coil migration leading to fatal gastrointestinal hemorrhage has been described.

GASTRODUODENAL, PANCREATICODUODENAL, AND INFERIOR MESENTERIC ARTERY ANEURYSMS

True aneurysms involving the gastroduodenal, pancreaticoduodenal, and inferior mesenteric arteries are rare. However, their behavior is unpredictable. In 1 series of VAA in 34 patients, all 6 patients with pancreaticoduodenal artery aneurysms presented ruptured, and 2 died. Thus, aneurysms in these locations should be treated. Management with ligation or coil embolization is appropriate, as reconstruction is not only technically difficult, but is likely not required due to adequate collateralization. However, adequate collateral flow should be documented with preoperative imaging if permitted. Imaging with CT and magnetic resonance angiography demonstrate excellent resolution for preoperative planning. However, angiography may allow for better evaluation of real-time flow dynamics.

VISCERAL ARTERY PSEUDOANEURYSMS

Visceral artery pseudoaneurysms (VAPA) occur in the setting of infection, chronic inflammation, and trauma. The literature suggests that VAPA are more prone to symptoms or rupture. In 1 series, 7 out of 25 patients who presented with rupture had VAPA secondary to chronic pancreatitis. Endovascular treatment of VAPA has certain advantages in anatomically difficult locations, and in the postoperative abdomen. In our series from Mount Sinai, ruptured VAPAs were significantly more likely to be treated by endovascular techniques. Hemodynamic instability did not preclude endovascular repair, which has also been seen in other studies. Indeed, endovascular approaches may have some advantage in the hemodynamically unstable patient with VAPA in difficult locations—angiography not only confirms the pathology, but, in addition, endovascular access may allow for rapid control with intraluminal balloon occlusion.

CONCLUSION

In summary, although VAA and VAPA are relatively rare, the mortality associated with rupture is significant. Thus, a rigorous plan should be adopted when they are recognized in the elective setting. In general, SAA measuring ≥2 cm and those found in women of childbearing age and in persons undergoing OLT should be treated. Hepatic artery aneurysms >2 cm and that are multiple or nonatherosclerotic in nature should be fixed in the appropriate patient due to a higher risk of rupture. Endovascular coil embolization has become the treatment of choice for anatomically suitable SAA and intrahepatic HAA. Success rates range from 85% to 100%, but reperfusion is a reported complication. Aneurysms involving the celiac and superior mesenteric arteries are much less common, but should be repaired in the appropriate medical patient. Ligation without reconstruction is feasible in the management of these aneurysms, provided that collateral circulation, often determined on preoperative imaging, is adequate. Otherwise, bypass with vein (in the setting of infection) and prosthetic graft is recommended. Finally, aneurysms involving the pancreaticoduodenal, gastroduodenal, and inferior mesenteric arteries, as well as VAPA, should be repaired, as rupture risks are high and behavior is unpredictable. Endovascular coil embolization is particularly useful in these settings, in which open operative exposure may be difficult and arterial reconstruction is not necessarily required.

DISCLOSURES

Potential conflict of interest: Nothing to report.

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