A 66-Year-Old Man With an Abdominal Aortic Aneurysm
Review of Screening and Treatment

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DR BURNS: Mr F is a 66-year-old man with an abdominal aortic aneurysm (AAA). He has a medical history of coronary artery disease, coronary artery bypass graft surgery in 1995, hyperlipidemia, hypertension, tobacco use, and gastroesophageal reflux disease. He has Medicare insurance with coinsurance.

In November 2004, Mr F saw his primary care physician for a routine physical. At that time, he smoked 1 pack of cigarettes per day, his blood pressure was controlled (110/70 mm Hg) with lisinopril, and his cholesterol was high (low-density lipoprotein cholesterol [LDL-C], 168 mg/dL [4.35 mmol/L]) despite atorvastatin. He was counseled to quit smoking and his atorvastatin dosage was increased. He was seen a few months later for persistent gastroesophageal reflux symptoms that disrupted his sleep and were unresponsive to proton pump inhibitors. When upper endoscopy did not reveal a cause, he underwent chest computed tomography (CT) because of a concern about external compression of the esophagus. The CT scan revealed ectasia of the descending thoracic aorta and aneurysmal dilation of the abdominal aorta. A subsequent CT angiogram revealed heavy atheroma and mild aneurismal dilation of the descending thoracic aorta (3.8 cm at its maximum) and the infrarenal AAA (4.3 cm at its maximum). It also showed extensive thrombus at the origin of the right renal artery; the right kidney appeared diminutive.

At Mr F's next visit with his physician, his cholesterol remained above goal (LDL-C, 83 mg/dL [2.15 mmol/L]) and his atorvastatin dosage was increased to 80 mg/d. He began taking aspirin and metoprolol. Although counseled again on the importance of smoking cessation, he continued to smoke 1 pack per day. His creatinine was slightly elevated at 1.3 mg/dL (114.9 µmol/L). When a CT angiogram was repeated 6 months later, the thoracic and infrarenal aortic aneurysms were unchanged in size at 3.5 cm and 4.4 cm, respectively.

A little more than 1 year later, Mr F had a repeat abdominal ultrasound and the infrarenal AAA was somewhat larger at 5.0 × 3.8 cm (FIGURE 1A). Over the next year, the aneurysm remained stable as measured by abdominal ultrasound. At

Ruptured abdominal aortic aneurysm (AAA) is a common cause of death. Abdominal aortic aneurysms tend to be asymptomatic until the time of rupture, which has a mortality rate of greater than 80%. Therefore, elective repair prior to rupture is preferred if life expectancy is reasonable and the risk of rupture outweighs the risk of repair. Mr F, a 66-year-old man with a 5.2-cm AAA, illustrates the issues surrounding monitoring and treating AAA. Risk factors for AAA include older age, male sex, smoking history, and a family history of AAA. Screening for AAA with ultrasound has been shown to prevent rupture, prevent AAA-related death, and be cost-effective. Risk factors for rupture include larger diameter, female sex, and smoking history. Endovascular repair has lower operative mortality and complications and has replaced standard open surgery in more than half of patients. However, long-term survival is similar after endovascular and open surgical repair. Those at risk of AAA who would benefit from repair should undergo screening.

this time, he was seen by a vascular surgeon who recommended continued cardiovascular risk factor reduction.

Early in 2008, Mr F underwent surgery for an ulnar nerve entrapment. At that time, an anesthesiologist recommended cardiology clearance because Mr F reported chest pain with exertion. An exercise MIBI revealed a moderated fixed, inferobasilar, lateral, and apical defect, and his ejection fraction was reduced to 31%. Cardiac catheterization was performed, the right coronary artery was stented, and balloon angioplasty of a small distal right coronary vessel was executed. Mr F continued his same medication with the

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addition of clopidogrel for 3 months. He was again encouraged to stop smoking.

At a subsequent visit with his primary care physician, Mr F's ulnar nerve entrapment symptoms persisted. He was still smoking 1 pack per day. His blood pressure (118/60 mm Hg) and cholesterol (LDL-C, 63 mg/dL [1.63 mmol/L]) were well controlled and his creatinine was stable (1.2 mg/dL [106.1 µmol/L]). A repeat abdominal ultrasound and subsequent CT angiogram revealed that his AAA had grown to 5.2 cm (Figure 1B). He continued taking atorvastatin, 80 mg/d; clopidogrel, 75 mg/d; lisinopril, 10 mg/d; metoprolol, 50 mg/d; omeprazole, 20 mg/d; aspirin, 325 mg/d; and niacin-inositol, 100 mg/400 mg twice daily. He was also taking hydrocodone, acetaminophen, and tramadol for wrist and shoulder pain. Surgery for his wrist was postponed until the clopidogrel was stopped.

MR F: HIS VIEW

I was first diagnosed with my aneurysm about 4 or 5 years ago. My doctors found it accidentally when they were looking for problems with my esophagus. After the tests, my doctor called me and said, "I need to see you . . . now." So I went in and she told me about the aneurysm and I went, "Oh," feeling as if there wasn't much else to say. I backed up, relaxed a little bit, and tried to grasp what was going on.

She referred me to a cardiovascular specialist who follows me. We've been following the aneurysm now for about 4 years. I see my cardiovascular surgeon about every 6 months and so far it hasn't gotten big enough for me to worry about.

I know the aneurysm can break or rupture and have dire consequences. It took about a year to let that information settle. But when it did, I realized that we have winter too, and I don't like it either.

I came to terms with my aneurysm on an intellectual basis. There's nothing I or anybody else can do. At some point I'm going to die, so all I can do is enjoy what I've got. I can't spend my life worrying about an inevitability. That's a waste of my time and a misdirection of my efforts.

As a preventive measure, my doctor has recommended I maintain my blood pressure. So far this is going very well. I have hypertension and I've had a quadruple bypass. In addition, my cholesterol is down, both from medication and my diet. I generally eat mostly chicken, fish, vegetables, and fruit. However, I do smoke. I should quit, but I haven't been able to yet.

## AT THE CROSSROADS: QUESTIONS FOR DR SCHERMERHORN

What are the risk factors for AAA and who should be screened? When should a patient with an AAA be referred for surgical evaluation? What are the surgical treatment options and what are the risks of each? What do you recommend for this patient? What does the future hold?

DR SCHERMERHORN: Mr F is a 66-year-old man with a profile typical of patients with AAA, a male smoker with hypertension and coronary artery disease with an asymptomatic AAA detected incidentally by an imaging study performed for workup of other potential abdominal pathology. His AAA is growing slowly over time and he is struggling with attempts at smoking cessation.

Epidemiology and Risk Factors

Abdominal aortic aneurysm is the 13th leading cause of death in the United States with up to 9000 annual deaths from ruptured AAA.[1] However, this is likely a gross underestimate of the actual number since approximately 300 000 persons

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Figure 1. Mr F's Abdominal Ultrasound and Images From His Computed Tomographic Angiogram

A Abdominal ultrasound

B Computed tomographic angiography (coronal plane)
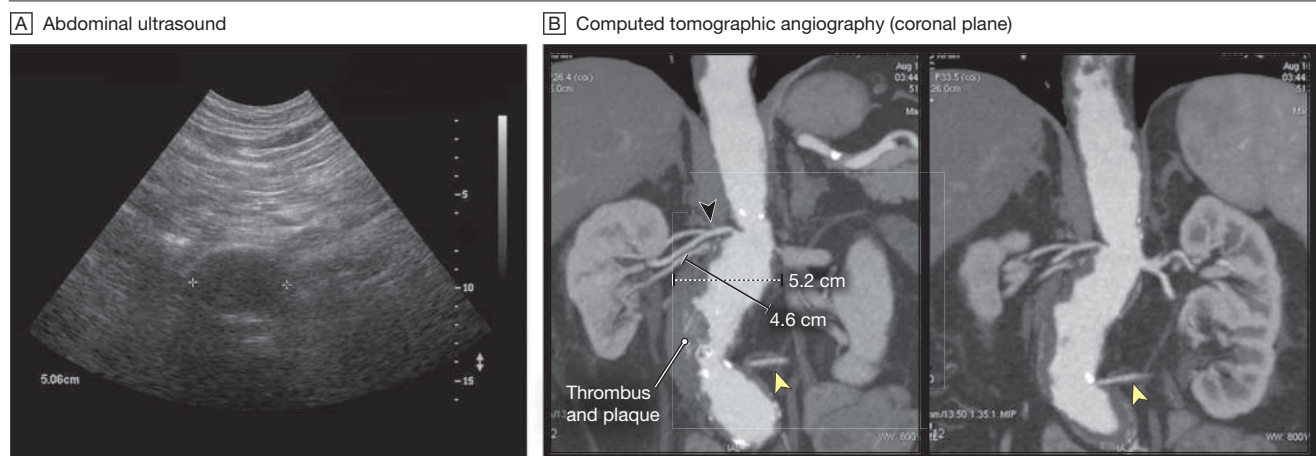
A, The ultrasound shows the diameter of the infrarenal abdominal aortic aneurysm. B, Left slice shows the aneurysm and thrombus involving the infrarenal aorta. The aneurysm extends up to and possibly involves the main renal artery origins. The left slice illustrates the importance of appropriate plane when assessing diameter in the presence of angulation or tortuosity of the aorta. The horizontal line shows Mr F's aorta measurement as 5.2 cm, whereas the diagonal line shows the more accurate measurement, perpendicular to the center line of the aorta, as 4.6 cm. In both slices, a large accessory left renal artery arising from lower aorta involved in the aneurysm can be seen (yellow arrowheads). Note also the presence of 2 right renal arteries (black arrowhead). See interactive Figure of the computed tomographic angiogram at http://www.jama.com.

die annually without medical care and 4% to 5% of sudden deaths are caused by ruptured AAA.[2-4]

Abdominal aortic aneurysm is defined as a 50% increase in diameter compared with the normal adjacent aorta or, for practical purposes, an aortic diameter greater than 3.0 cm.[5] The prevalence of AAA is 3% to 10% in those older than 50 years and varies based on the prevalence of risk factors in the population.[6-12] Men are 5 times as likely as women to have an AAA with a prevalence of 3% to 10% compared with only 1% to 2% for women. The most important modifiable risk factor is smoking; smokers are approximately 5 times as likely as nonsmokers to develop AAA. Current smokers are more likely than former smokers, who, in turn, are more likely than never smokers, to have AAA.[12,13] Years of smoking are more important than the absolute number of cigarettes smoked.[13] Abdominal aortic aneurysm is rarely seen younger than 50 years and its prevalence increases with age. Women tend to develop AAA approximately 10 years later than men.[14] Other risk factors are hypertension and white race.[8] Diabetes has been found to be protective, with half the risk of developing AAA.[8] Mr F's profile fits these criteria as a man older than 50 years with hypertension who has smoked for many years and continues to smoke.

Natural History

The natural history of AAA is slow growth with eventual rupture if the aneurysm reaches a large enough diameter before the patient dies of other causes. Unfortunately, AAAs tend to remain asymptomatic until the time of rupture. The AAA sac is often lined with thrombus, which can lead to distal embolization as the presenting symptom in 2% to 5% of patients who eventually undergo repair. Emboli may cause "blue toe" syndrome, with palpable pedal pulses and cyanosis of the toes.[15] Those AAAs that do become symptomatic typically do so at the time of rupture or acute expansion with presumed impending rupture.[16,17] The most typical symptoms are severe abdominal or back pain. Pain may radiate to the flank or groin. Hypotension or loss of consciousness may be the only signs or symptoms of AAA rupture in many patients. In Sweden, with a high autopsy rate, it was discovered that 30% to 50% of patients with ruptured AAA die before reaching the hospital.[18] An additional 30% to 40% die after reaching the hospital without an operation. Operative mortality after rupture is 40% to 50%, making overall mortality with rupture 80% to 90%.

Screening for AAA

Since AAAs cause few symptoms, they are typically detected as an incidental finding on imaging studies performed for other purposes. The calcified wall of the AAA sac may be seen on plain abdominal radiograph but more typically AAAs are discovered on ultrasound or CT scan and, occasionally, magnetic resonance imaging. Nearly two-thirds of all patients with AAAs undergoing repair have their AAA detected in this manner, as was the case with Mr F.[19] Physical examination is unfortunately inaccurate for detection of AAA. The ability to detect AAA on abdominal palpation is dependent on the AAA diameter, the abdominal girth, and the thoroughness of the examination. In a focused physical examination with the intent to detect AAA, the diagnosis was suspected in 29% of AAAs sized 3.0 to 3.9 cm, 50% of those 4.0 to 4.9 cm, and 75% of those at least 5.0 cm.[20]

Ideally, AAAs that are destined to rupture would be identified by screening since identification of AAA prior to rupture allows elective repair with a mortality rate of approximately 5% compared with an operative mortality of 40% to 50% after rupture.[21] Ultrasound is the preferred mode because it is able to accurately identify AAA with sensitivity and specificity that approach 100%.[22,23] Visualization may be limited in 2% to 3% of patients because of overlying bowel gas or truncal obesity. In addition, ultrasound is inexpensive, causes minimal pain, and has minimal associated risk.

Four randomized trials have examined the effectiveness of ultrasound screening for AAA and were summarized in a Cochrane review and meta-analysis.[24-31] In total, there were 127 891 men and 9342 women in multiple countries ranging in age from 65 to 80 years. Between 63% and 80% of those invited underwent screening. With follow-up ranging from 4 to 15 years, there was a 50% reduction in AAA rupture, a 50% decrease in aneurysm-related mortality, and a 6% reduction in overall mortality, although the presence of a significant reduction in all-cause mortality has been disputed.[32] The Multicenter Aneurysm Screening Study (which evaluated men only) found an incremental cost-effectiveness ratio of $19 500 per life-year gained, confirming the cost-effectiveness of screening for men age 65 to 74 years such as Mr F.[33] The US Preventive Services Task Force analysis also found population-based ultrasound screening for AAA to be cost-effective.[34] There is little benefit from additional screening studies if the initial examination result is negative.[35]

Screening Recommendations

The US Preventive Services Task Force has recommended a one-time screening for men age 65 to 75 years who have ever smoked.[36] The Society for Vascular Surgery recommends a one-time screening for men at age 65 years, men with a family history of AAA at age 55 years, and women who ever smoked or have a family history of AAA at age 65 years.[37] The Centers for Medicare & Medicaid Services funds a screening ultrasound for men who ever smoked, but only as part of their "welcome to Medicare" visit (within a 12-month window). Women tend to develop AAA at a later age than men, have a lower prevalence of AAA than men, and have a higher operative risk.[14,38-41] However, they tend to live longer than men and have a higher rupture risk at any given diameter.[41-43] Therefore, screening in women may be indicated, particularly in smokers or those with a family history of AAA.[37,41,44,45] Pending legislation[46] would unlink the screening benefit from the Medicare welcome visit and extend the benefit to men aged 65 to 75 years who ever smoked and to men and women with a family history of AAA.

Aneurysm disease is often multifocal and may be found concomitantly in iliac, popliteal, and femoral arteries, as well as the thoracic aorta.[47,48]

Small aortic aneurysms (3-5.5 cm) have little risk of rupture and can be safely observed with ultrasound surveillance. Abdominal aortic aneurysms 3 to 4 cm tend to grow slowly (<10% per year) and are typically imaged on an annual basis.[10,21,49] Abdominal aortic aneurysms larger than 4 cm tend to grow on average by 10% per year, although there is considerable variability.[10,21,50,51] Abdominal aortic aneurysms in this diameter range are typically scanned with ultrasound every 6 months until a decision to intervene is made.[52,53] Mr F had annual ultrasound surveillance until the diameter exceeded 4.5 cm. After this point, ultrasounds at 6-month intervals revealed slow expansion of his AAA over time.

Surgical Evaluation and Treatment Options

Decision making for AAA patients such as Mr F includes determining the appropriate timing for repair and the appropriate type of repair (endovascular or open surgical). β-Blockade, doxycycline, and statins have all been proposed to slow the expansion of AAAs, but none have been demonstrated to be effective.[54,55] Timing for intervention has been clarified by 2 large randomized trials: the UK Small Aneurysm Trial and the Aneurysm Detection and Management Trial, both of which showed no benefit of early surgery for AAAs sized 4.0 to 5.5 cm vs strict ultrasound surveillance and intervention at 5.5 cm.[8,52,53,56,57] The indications for repair at diameters less than 5.5 cm in these trials were rapid growth (>1 cm/y), development of abdominal pain, or tenderness to palpation. With this "watchful waiting" policy, more than 70% of patients eventually undergo repair.[8,52,53,56,57] Of note, there was a rupture risk of 0.7% and 1% per year for AAAs observed in the 2 trials.[8,52,53,56,57] Rupture risk was significantly higher for those with larger initial AAA diameter (odds ratio [OR], 2.5 per 1 cm).[43] Additionally, rupture risk during surveillance was 4.5 times greater for women than men, prompting many to use a lower threshold for intervention in women compared with men.[41,43] While these trials have given enormous help in management of small AAAs, it would be prudent to avoid a "one size fits all" approach to AAA. One should consider the rupture risk, operative risk, and life expectancy for each individual patient to determine when repair should be considered. As discussed below, each of these factors are important for Mr F in considering his management options.

Abdominal aortic aneurysm diameter is an important predictor of rupture risk. In a separate analysis from the UK trial including the 1090 randomized patients as well as 1167 patients not randomized because of diameter smaller than 4.0 cm (n=507), diameter larger than 5.5 cm (n=100), randomization refusal (n=122), and those unfit for surgery (n=340), the annual risk of rupture was estimated to be 0.3% for AAAs smaller than 4 cm, 1.5% for AAAs 4 to 4.9 cm, and 6.5% for those 5 to 5.9 cm.[43] This likely underestimates the risk for women, who made up only 17% of the UK trial. However, the fact that not all AAAs rupture at a given diameter suggests that other factors are involved. The UK Small Aneurysm Trial demonstrated that predictors of rupture are female sex (hazard ratio [HR], 3.00; 95% confidence interval [CI], 1.99-4.53), hypertension (per 1 mm Hg, HR, 1.02; 95% CI, 1.00-1.03), and chronic obstructive pulmonary disease (per 1 L forced expiratory volume in first second of expiration, HR, 0.62; 95% CI, 0.45-0.86). Tobacco use had a nonsignificantly elevated risk (HR, 2.11; 95% CI, 0.95-4.67). Mr F continues to smoke, likely putting him at increased risk of rupture. His hypertension is well controlled.

Type of Repair

For patients considering repair, there are 2 options: open repair or endovascular AAA repair (EVAR) (**FIGURE 2**). Open repair of AAA was popularized by Creech[58] in 1966. The aorta is exposed through a laparotomy and clamped above the aneurysm; the iliac arteries are clamped below. The AAA sac is opened and a prosthetic graft is sutured in place directly. The sac is closed over the graft. EVAR was introduced by Parodi et al[59] in 1991. US Food and Drug Administration approval was granted in 1999. Since then, use of this technique has expanded such that nearly 60% of AAA repairs in the United States are now EVAR.[60] Compared with open surgery, EVAR has lower operative mortality (1.2% vs 4.8%), lower morbidity, shorter length of hospital stay (3 vs 9 days), and greater likelihood of discharge to home (95% vs 82% for discharge to home vs to rehabilitation or nursing home).[61] These data were confirmed in the recently published Open Versus Endovascular Repair (OVER) randomized trial.[62] The EVAR procedure involves transfemoral insertion of a stent graft made of fabric and self-expanding metal stents that attach to a segment of healthy aorta below the renal arteries and again to the healthy iliac arteries below, thereby excluding the aneurysm. To be a candidate for EVAR, there must be an adequate segment of healthy aorta below the renal arteries as well as adequate access vessels (iliac arteries). This is typically assessed by preoperative CT angiography with 3-dimensional reconstructions.[63] Because of these anatomic constraints, not all patients are candidates for EVAR. Mr F is not a candidate for EVAR because of lack of adequate infrarenal neck; he would therefore require open repair.

Because stent grafts are not sutured in place as with open repair, the long-term durability of EVAR is not equal to open repair. EVAR patients typically undergo surveillance at 1 and 6 months and then annually, usually with CT angiography, to ensure adequacy of the repair.[64] A small proportion (2%-5% per year) need to undergo a revision procedure to maintain the integrity of the repair and prevent rupture.[61,65,66] These reinterventions are typically minor and are performed either percutaneously or with a small groin incision to expose the femoral vessels. Occasionally, however, it is necessary to convert an EVAR to a more conventional surgical repair (1%-3% at 4 years).[61,65,66] While patients treated with EVAR are more likely to undergo AAA-related reinterventions (9.0% vs 1.7% at 4 years), those undergoing open repair are more likely to require abdominal incision–related readmissions (14.2% vs 8.1% at 4 years) and reinterventions (9.7% vs 4.1% at 4 years), such as abdominal wall hernia repair and lysis of adhesions for small bowel obstruction.[47]

EVAR may allow treatment of older, sicker patients who are at risk of rupture and who would not have been considered for repair when open surgery was the only choice.[60] Elective AAA repairs have increased in the elderly population, primarily because of expansion of EVAR. At the same time, elective repairs have actually decreased in younger patients, likely because of data from small aneurysm trials demonstrating the safety of watchful waiting until diameter reaches 5.5 cm. Mortality rates have decreased with elective repair because of increasing use of EVAR in both older and younger age groups. After introduction of EVAR, AAA ruptures in the United States have decreased in both younger (aged 65-74 years; 9.3/100 000 in 1996 vs 4.0/100 000 in 2006) and older (aged ≥75 years; 23.4/100 000 in 1996 vs 13.6/100 000 in 2006) age groups, with a greater decline in rupture rate in older patients.[67] This suggests that the overall incidence of AAA may be decreasing in the United States, perhaps because of a decline in smoking. Additionally, introduction of EVAR, with expansion of elective repair in older patients, has likely led to rupture prevention while simultaneously reducing deaths due to elective surgery despite an increase in procedure volume.

Operative Risk

Operative risk is dependent on comorbid conditions as well as type of repair. EVAR has significantly lower mortality than open repair (1.2% vs 4.8%).[61,68-70] Other predictors include older age (76-80 years: OR, 1.9; 95% CI, 1.4-2.5; >80 years: OR, 3.1; 95% CI, 2.4-4.2; both compared with 67-70 years), renal failure (OR, 2.6; 95% CI, 1.5-4.6) or insufficiency (OR, 2.0; 95% CI, 1.6-2.6), congestive heart failure (OR, 1.7; 95% CI, 1.5-2.1), and female sex (OR, 1.5; 95% CI, 1.3-1.8).[40] Coronary ischemia on resting electrocardiogram has also been shown to predict mortality.[71,72] Symptomatic coronary disease mandates preoperative cardiac evaluation and treatment with optimization of medications and potentially coronary revascularization.[73] Noninvasive testing in asymptomatic individuals who are inactive is reasonable to improve risk stratification and potentially improve the outcome of surgery.[73] Additional preoperative assessment should include evaluation of pulmonary function in those with a history or symptoms of chronic lung disease.[5] Standard blood testing may detect significant renal disease or coagulation disorders.[5] Mr F has a reduced ejection fraction and coronary artery disease that has been treated with stenting. He also has mild renal insufficiency. As discussed herein, the factors that increase his operative risk must be considered when deciding whether to intervene.

Watchful Waiting vs Surgical Repair

Elective AAA repair is a prophylactic procedure with the potential benefit of preventing rupture at a later date. Each patient has a different perspective on the value of immediate time vs future time, as well as separate needs regarding the ability to take time away from work or family activities for recovery. These preferences must be taken into account in combination with individual rupture risk, operative risk, and life expectancy to determine the appropriate threshold for intervention.

Figure 2. Open and Endovascular Repair of Abdominal Aortic Aneurysm
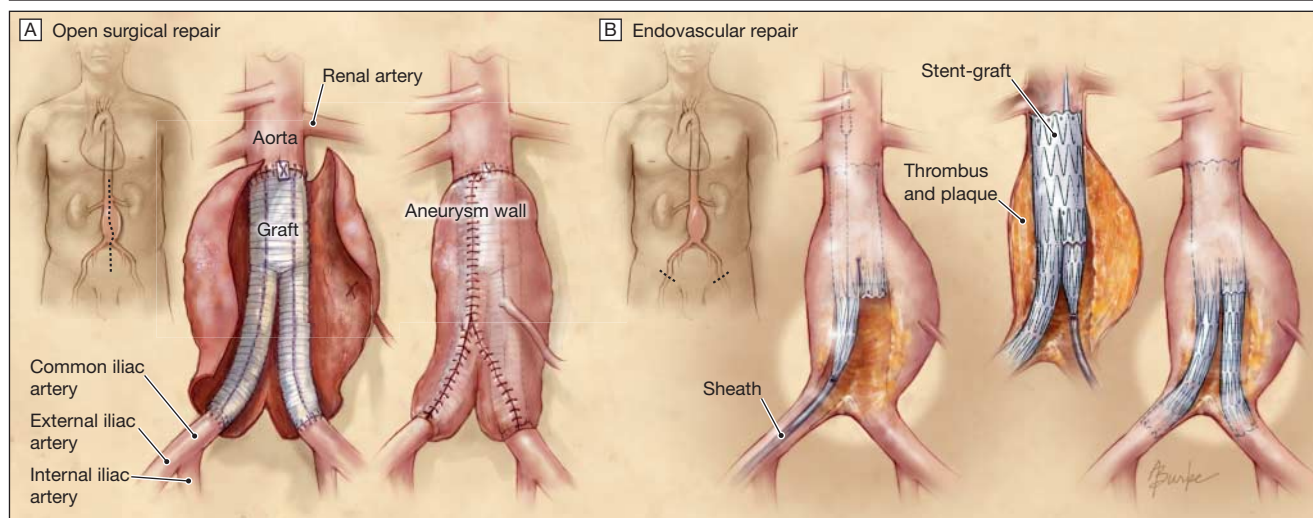
A, With open repair, the abdomen is opened anteriorly (as shown) or from a lateral retroperitoneal approach. The aorta is clamped, preferably below the renal arteries, and the common iliac arteries are both clamped. The aneurysm sac is opened longitudinally; backbleeding lumbar arteries and the inferior mesenteric artery are typically suture-ligated. A prosthetic graft is then sutured in place proximally and distally. A bifurcated graft (shown) is used in more than half of cases with the distal anastomoses to the common iliac or, rarely, the common femoral arteries, as opposed to a straight tube graft sewn to the aortic bifurcation. The aneurysm sac is then closed over the graft to provide separation of the graft from the intestines. B, With endovascular repair, stiff wires are introduced through the common femoral arteries over which a fabric covered stent (stent-graft) is introduced. The proximal graft is positioned just below the renal arteries. The stent-graft is initially constrained in a low-profile state until deployment. A modular device is depicted in which a separate component for the left iliac limb is inserted through and overlaps with a docking limb on the main device. Ultimately, there is a seal zone in the normal infrarenal aorta and bilateral iliac arteries, thereby excluding the abdominal aortic aneurysm. See animation of surgical procedures at http://www.jama.com.

After an AAA has been diagnosed and a course of watchful waiting has been chosen, there are several management issues to be addressed. The primary modifiable predictor of expansion and rupture is smoking.[43,74] Mr F continues to smoke despite being informed about the increased risk of rupture; if he has not tried a smoking cessation program, he should be advised to do so.[75] Hypertension should be controlled.[43,76] Mr F's hypertension has been well controlled with metoprolol and lisinopril. Many patients are fearful of vigorous activity once an AAA has been diagnosed, but there are no data to suggest that this is necessary[64]; in fact, it is wise to avoid a sedentary lifestyle and stay (or become) fit in the event that surgery is required.[64]

When repair is being considered, further imaging of the aorta is indicated based on ultrasound diameter and the general health of the patient.[5] Computed tomography angiography with thin "slices" has virtually eliminated the need for arteriography. Computed tomography allows for more accurate diameter measurement compared with ultrasound, particularly when combined with 3-dimensional reconstructions and measurements perpendicular to the center line of the aneurysm. Computed tomography angiography provides detailed information about the extent of aneurysm involvement in relation to the visceral, renal, and iliac arteries as well as occlusive disease that might alter the surgical plan; it also can define the most appropriate location for suprarenal clamp placement when necessary, based on the relationship of the visceral arteries and the presence of aortic wall calcification and luminal plaque. Computed tomography angiography is the test of choice to identify suspected inflammatory aneurysm and may reveal unsuspected abdominal pathology such as malignancy or gallbladder disease. It also detects anatomic venous variants such as retroaortic left renal vein, duplicate cava, or renal abnormalities such as pelvic or horseshoe kidney.[5]

Follow-up Care

Ultimately, survival to at least 7 years is similar after EVAR vs open repair.[61,65,66,77] The most likely explanation for the eventual overlapping of the survival curves is that a portion of the least fit patients (eg, those with older age, heart failure, renal failure) die perioperatively with open surgery but survive EVAR. Less fit patients surviving EVAR die sooner in the postoperative period because of their other underlying coexisting disease, and eventually the 2 groups are similar in general "fitness."[61,65]

Life expectancy is reduced in those with AAA compared with the general population because of associated comorbidity.[78-80] Despite this, estimated life expectancy for a typical 75-year-old after AAA repair is 8 years and for a typical 80-year-old is 6 years. Average life expectancy for a 65-year-old after AAA repair is 11 years, but Mr F may not achieve this life span given his additional risk factors.[5]

Future Directions

Genetic testing may assist at some point in diagnosing AAA,[81] but a genetic abnormality has not yet been adequately defined. Branched endografts may be available in the future to accommodate aneurysms that involve the origins of the renal and visceral vessels, but this is unlikely to become available soon.[82] Promising early work demonstrates that peak wall stress in AAAs (measured by analysis of CT angiography data) may predict rupture risk better than diameter.[83-86] In the analysis by Fillinger et al,[84] predictors of rupture were wall stress (HR, 25) and female sex (HR, 3); after controlling for these, diameter did not predict rupture. This observation will require prospective confirmation in a large cohort before widespread clinical use. At this time, the greatest potential for prevention of AAA-related deaths is through smoking prevention and cessation. Aside from this, the greatest effect would be through expansion of screening efforts with elective repair in those with reasonable life expectancy whose rupture risk outweighs operative risk.

RECOMMENDATIONS FOR MR F

There are several points regarding Mr F's case that bear mentioning. First, it is important to obtain accurate diameter measurements to better estimate the risk of rupture. Ultrasound and standard axial CT imaging may overestimate the true aortic diameter, particularly if there is angulation or tortuosity of the aorta. Mr F's aorta was measured at 5.2 cm by ultrasound and standard CT, while a more accurate diameter (measured in an orthogonal plane perpendicular to the center line) is actually 4.6 cm (Figure 1B). Second, examination of his CT shows that the aneurysm extends to the renal arteries (Figure 1B). Because of this, he would not be a candidate for EVAR. Additionally, open repair would require clamp placement above the renal arteries. He has a fairly large accessory left renal artery arising from the lower aorta involved in the aneurysm that should be reimplanted onto the graft at the time of AAA repair. He may require reimplantation of the upper left renal artery as well. Because of the high clamp and the need for renal revascularization, the operative mortality risk would be increased by a relative 40%[87] and there would also be increased risk of renal failure. Both of these factors should be considered in his decision.

Given Mr F's low risk of rupture at only 4.6 cm, I would favor watchful waiting at this time. Even if his aortic diameter were 5.2 cm, as suggested by ultrasound, several factors still favor medical management: the fact that he is not a candidate for EVAR, the complexity of the open repair necessary to fix this aneurysm, the relatively low risk of rupture, and his renal insufficiency and heart failure. However, if expansion continues, the rupture risk will eventually outweigh the risk of surgery, and repair may be considered. His preferences regarding imminent risk of surgery vs risk of rupture with ongoing surveillance must be considered. In the meantime, continued efforts should be made to assist him with smoking cessation, and his blood pressure should be controlled with medication as needed. Additionally, I would encourage him to engage in regular activity to lose weight and improve overall fitness so that if the AAA expands he will be a better candidate for surgery. Any siblings older than 55 years should be screened for AAA.[37]

QUESTIONS AND DISCUSSION

QUESTION: Are there any data about the differences in outcome between surgeons who do this a lot vs who don't do it very often?

DR SCHERMERHORN: Yes; Birkmeyer et al[88,89] first demonstrated this correlation with open repair, both at the hospital level and the surgeon level. My coinvestigators and I are now looking at endovascular repair combined with open repair to see if there is any effect on outcome with one procedure based on the volume of the other procedure or the total volume of both. This may be important as the overall volume of open repairs decreases.

QUESTION: What is the difference in cost between EVAR and open surgery?

DR SCHERMERHORN: Perioperative costs are similar.[90] The cost of the graft material is much higher for stent grafts. However, this is counterbalanced by a decrease in intensive care unit length of stay and total length of stay with EVAR.[91] The increase in expense for EVAR comes with follow-up CT scans and reinterventions to maintain graft integrity.[92,93] Previous analyses have only looked at the cost of reinterventions associated with the aorta and have ignored the cost of reinterventions related to the laparotomy and readmissions related to bowel obstruction.[48] There hopefully will be some data to evaluate this more thoroughly in the near future.

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