REVIEW

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Managing abdominal aortic aneurysms: Treat the aneurysm *and* the risk factors

ABSTRACT

Abdominal aortic aneurysms (AAAs) are not only a danger in themselves, they also signify underlying vascular disease that warrants intensive cardiovascular risk reduction, especially smoking cessation. Aneurysmal size and the patient's fitness for surgery are the main determinants of timing and method of elective repair. The choice of open surgery vs endovascular repair depends on the patient's condition, preference, and life expectancy, and the surgeon's experience.

KEY POINTS

Physical examination is fairly insensitive for detecting AAAs. Nevertheless, looking for AAAs is worthwhile during regular physical examinations. Contrary to a once-popular belief, gentle palpation of AAAs is safe and does not precipitate rupture.

The threshold for AAA repair is 5.5 cm in men or 5 cm in women. AAAs larger than 6 cm should be repaired if possible. Small aneurysms (4.0–5.0 or 5.5 cm) should be followed with serial ultrasound scans until they become symptomatic, enlarge rapidly, or become large enough to repair.

Endovascular repair is appropriate for older patients and others at higher operative risk, whereas open surgery may be preferred for younger patients because its results have proven more durable.

ABDOMINAL AORTIC ANEURYSMS (AAAs) are common and, if they rupture, lethal. They are also a marker for atherosclerotic cardiovascular disease.

Physicians treating patients with AAAs must decide when and how to treat these lesions. But they must also address the underlying vascular disease, with aggressive risk reduction strategies.

This article discusses the management of AAAs, including when and how it is appropriate to use the new technique of endovascular repair.

DEFINITIONS

Aneurysm is a Greek word meaning dilatation. The aorta below the renal arteries is the most common site for true arterial aneurysms.

A true *abdominal aortic aneurysm* is a circumscribed, permanent, segmental dilatation involving the three layers of vessel wall with a minimum increase in diameter of 50% compared with the expected normal (or the proximal) segment.[1]

Since the abdominal aorta tends to be about 2 cm in diameter, a true AAA measures 3.0 cm or more. However, the normal diameter varies depending on the patient's age, sex, height, weight, race, body surface area, and baseline blood pressure. Hence, using a diameter ratio may be better, particularly in smaller people such as women and those of short stature.

Aortic ectasia is a mild generalized dilatation (< 50% of the normal diameter or ≤ 2.9 cm)[1] that is due to age-related degenerative changes in the vessel wall.

Aneurysms are generally classified accord-

TABLE 1

Conditions associated with abdominal aortic aneurysms

Atherosclerosis (degenerative)
Promotes AAA formation

Cystic medial necrosis
Usually affects the ascending aorta and seen in Marfan syndrome

Vasculitis
Takayasu arteritis, giant cell arteritis, spondyloarthropathies, rheumatoid arthritis

Infectious diseases (rare)
Syphilitic arthropathy (rare ascending aorta), tuberculosis, mycotic aneurysms

Congenital (rare)
May be associated with cardiac anomalies

Trauma (rare)
Usually affects the ascending or descending aorta after closed deceleration injuries

ing to their shape. *Fusiform aneurysms*, the most common type seen in the infrarenal aorta, are diffuse and circumferential, while *saccular aneurysms* involve only a portion of the circumference, with a characteristic outpouching of the vessel wall.

Most AAAs are asymptomatic and expand silently

HOW ANEURYSMS DEVELOP

The aortic wall has a specific arrangement of structural proteins that give it strength and elasticity. With age or in response to other conditions (TABLE 1), the composition of the extracellular matrix protein in the media may change, with subsequent destruction of the elastic lamella, rendering the aorta less able to withstand the force of systolic pressure. Consequently, the wall dilates and may rupture.

The infrarenal aorta is more prone to develop aneurysms than other segments, for several reasons. Mechanically, it is the segment that must expand the most during systole and contract the most during diastole. Its wall is thinner and has fewer adventitial vasa vasora than the thoracic aorta. Physiologically, it is more prone to atherosclerosis, a proposed nidus for aneurysmal dilatation.

Atherosclerosis, inflammation, and MMPs

Many patients with AAAs also have atherosclerosis in the aorta and in other arteries, suggesting that aneurysmal disease may be a part of a larger spectrum of vascular disease and that atherosclerosis promotes AAA formation.

In atherosclerotic AAA, inflammatory cells infiltrate into the vessel wall and may secrete specific matrix metalloproteinases (MMPs). The different types of MMPs play diverse roles via complex interactions that eventually lead to degradation of the structural media proteins, and subsequently to aneurysmal dilatation (FIGURE 1).[2]

In addition, there are significantly fewer smooth muscle cells in human AAA tissues than in normal or atherosclerotic nonaneurysmal aortic tissue.[3] This decrease in smooth muscle cells is suspected to be secondary to apoptosis, suggesting a role for focal cell apoptosis in the pathogenesis of AAA.

A DISEASE OF THE ELDERLY

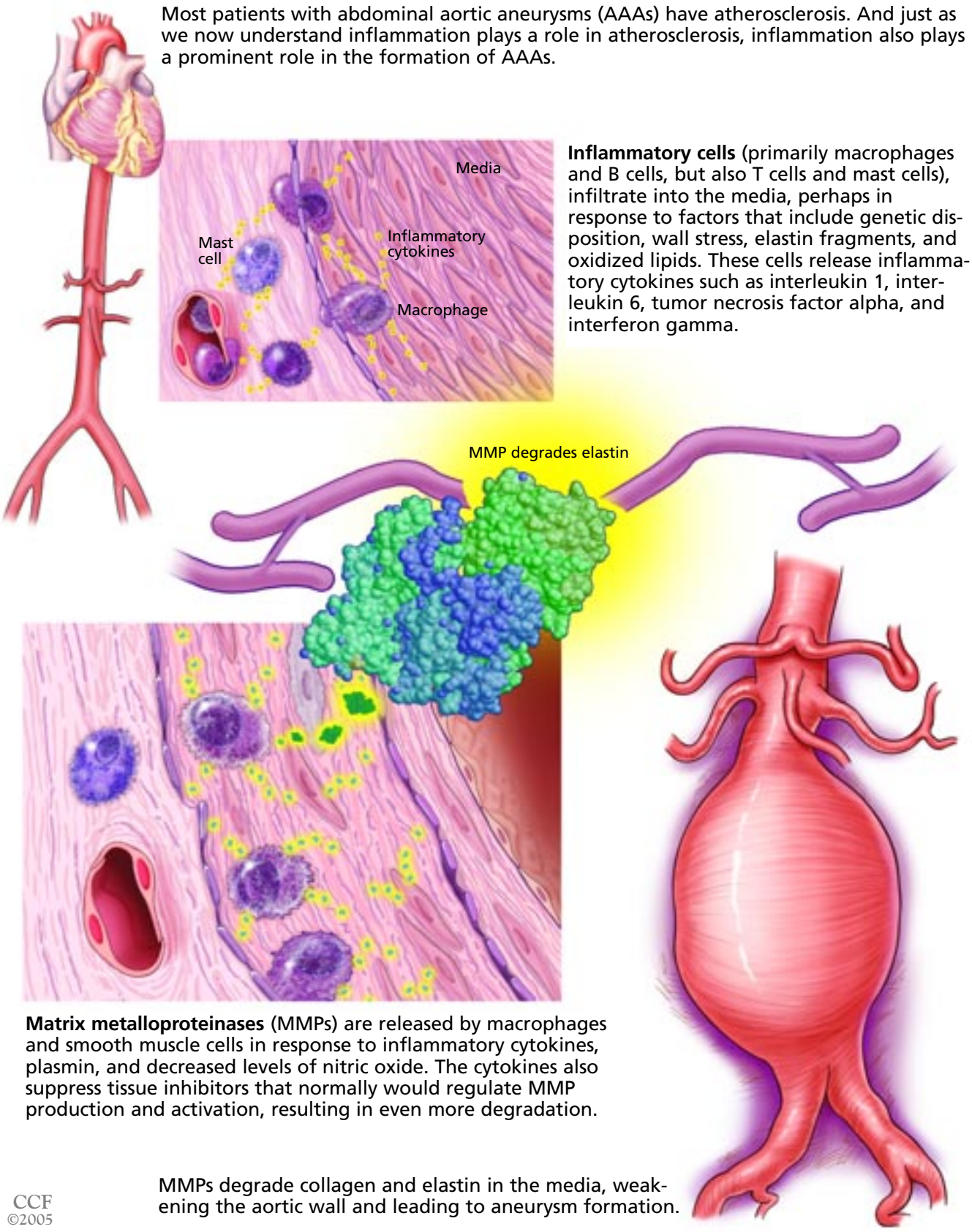
AAA is a disease of the elderly; it is the 10th leading cause of death in older men in the United States, and the individual's risk of AAA increases by 6% per decade of life.[4]

Incidence may be increasing

AAAs seem to have increased in incidence over the past 50 years. A population-based study from Rochester County, Minnesota, estimated that the incidence tripled between 1951 and 1980 (from 12.2 to 36.2 per 100,000 person-years).[5,6]

The complex role of inflammation in AAA formation

Most patients with abdominal aortic aneurysms (AAAs) have atherosclerosis. And just as we now understand inflammation plays a role in atherosclerosis, inflammation also plays a prominent role in the formation of AAAs.



Inflammatory cells (primarily macrophages and B cells, but also T cells and mast cells), infiltrate into the media, perhaps in response to factors that include genetic disposition, wall stress, elastin fragments, and oxidized lipids. These cells release inflammatory cytokines such as interleukin 1, interleukin 6, tumor necrosis factor alpha, and interferon gamma.

Matrix metalloproteinases (MMPs) are released by macrophages and smooth muscle cells in response to inflammatory cytokines, plasmin, and decreased levels of nitric oxide. The cytokines also suppress tissue inhibitors that normally would regulate MMP production and activation, resulting in even more degradation.

MMPs degrade collagen and elastin in the media, weakening the aortic wall and leading to aneurysm formation.

FIGURE 1

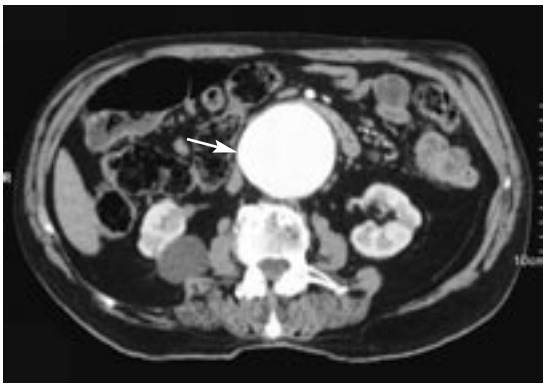


FIGURE 2. Computed tomographic scan of the abdomen showing a large abdominal aortic aneurysm (arrow).

It is tempting to attribute this trend to increased life expectancy and increased awareness of aneurysms and screening for them. However, the incidence of complicated, symptomatic, large AAAs also increased,[5,6] and necropsy studies also show an increased incidence, both of which suggest that awareness and screening do not explain the trend. On the other hand, the increased incidence may have been biased by the introduction of new surgical techniques and decreased operative mortality, similar to what happened when the reported incidence of proximal femur fractures increased when hip pinning was introduced in the 1930s.[7]

Smoking is the strongest modifiable risk factor for AAAs

More common in men than in women

In a large multinational study,[8] the prevalence of AAA was four times higher in men than in women (0.4% in nonsmoking women vs 1.6% in nonsmoking men; 1.5% in women who smoke vs 6.3% in men who smoke). The prevalence is two to five times higher in men and two to three times higher in women with cardiovascular risk factors or atherosclerotic cardiovascular diseases than in control groups without such risk factors.[9]

The disease tends to affect older Caucasian men more than their black counterparts.[8] It tends to cluster in families, often affecting young members who may not have the traditional acquired risk factors.

OFTEN DIAGNOSED INCIDENTALLY

Many AAAs are detected incidentally during cardiac catheterization, computed tomography (CT), ultrasonography, or magnetic resonance imaging (MRI) performed for unrelated reasons. Up to 50% of AAAs can be recognized on plain roentgenograms as a calcified aneurysmal wall.

Most AAAs are asymptomatic

Most AAAs are asymptomatic and expand silently. New spontaneous abdominal pain in a patient with a pulsatile epigastric mass or a known AAA may signal rupture into the retroperitoneum or leakage within the aneurysm wall, leading to rapid expansion or imminent rupture. This requires immediate evaluation with CT if possible or urgent exploratory laparotomy and aggressive management.

Peripheral embolization to the lower extremities, which is common with popliteal artery aneurysms, is rare with AAAs even though many of them contain a mural thrombus. Rarely, disseminated intravascular coagulopathy may develop, particularly with larger or unstable aneurysms.[10]

Physical examination is worthwhile

Physical examination may miss a substantial number of asymptomatic AAAs. Nevertheless, one should check the abdominal aorta during regular physical examinations because it is easy to do and may detect a life-threatening aneurysm.

The sensitivity of physical examination increases with the size of the aneurysm: 29% to 61% for AAAs 3.0 to 3.9 cm in diameter and 76% to 82% for those 5.0 cm or larger.[11,12] It is generally easier to detect a pulsatile mass in thin patients and those who do not have a tense abdomen. Fink et al[12] found that a focused abdominal examination is very sensitive for detecting AAAs large enough (≥ 5 cm) to warrant elective intervention in nonobese patients (abdominal girth < 100 cm [40 inches]).

Contrary to a once-popular belief, gentle palpation of AAAs is safe and does not precipitate rupture.[11]

Imaging studies

Ultrasonography has a sensitivity close to 100%, is well accepted by patients, and is the preferred method for detecting and following the progression of AAAs. It can show

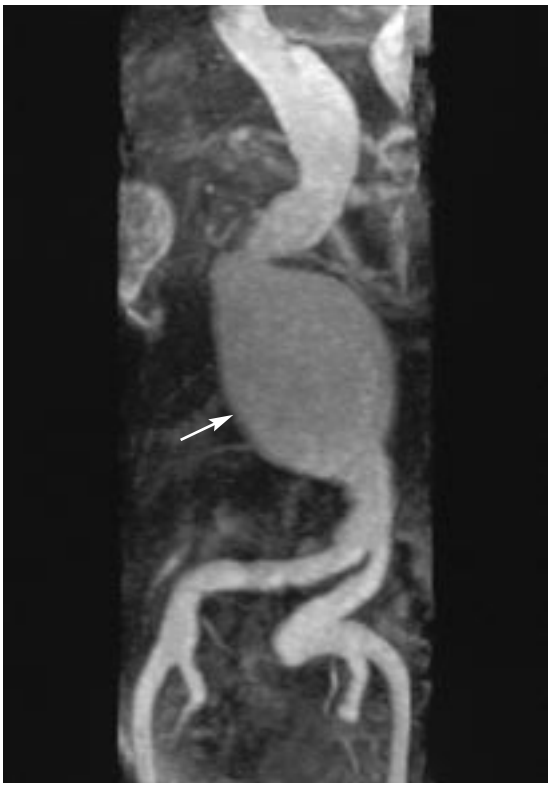


FIGURE 3. Magnetic resonance angiogram of the abdomen showing a large abdominal aortic aneurysm (arrow).

the dimensions of the abdominal aorta and other relevant findings such as mural thrombus and iliac artery aneurysms. To optimize image quality, patients should fast before the examination, which reduces bowel gas. Ultrasonography is operator-dependent and may not be as accurate in obese patients.[13]

Contrast CT provides detailed anatomic information and is valuable when planning AAA repair (FIGURE 2). The disadvantages include nephrotoxicity, cost, exposure to radiation, suboptimal visualization of the origins of aortic branch vessels (compared with conventional angiography), and, occasionally, inaccurate localizing of the aneurysmal neck.

Magnetic resonance angiography (MRA) does not require nephrotoxic contrast but is not more accurate than thin-slice CT (FIGURE 3). MRA is costly and not as readily available as contrast CT and ultrasonography.[14]

Contrast aortography is performed before surgery in patients suspected of having suprarenal or juxtarenal aneurysms, renovascular hypertension, ischemic nephropathy,

Thrombus can cause angiography to underestimate aneurysm size

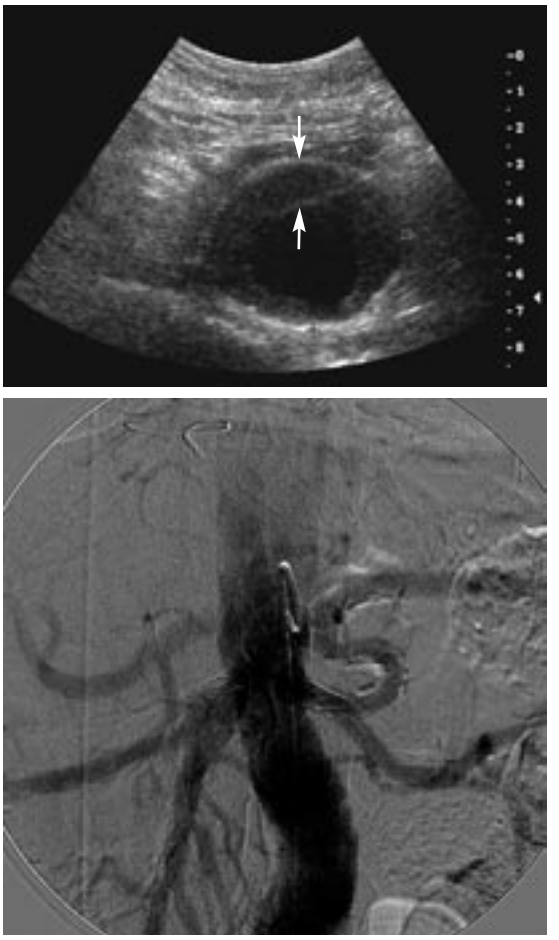


FIGURE 4. **Top**, Ultrasonographic image showing a transverse section in an abdominal aortic aneurysm with intramural thrombus (arrows). **Bottom**, an angiogram in the same patient underestimating the size of the aneurysm.

mesenteric ischemia, or associated iliofemoral arterial occlusive disease. Although it is routinely used to determine anatomy and morphology, it should not be used to assess the size of an AAA because the common presence of mural thrombus often leads to diameter underestimation (FIGURE 4). This holds true for all types of aneurysms.

Ultrasound scanning is the preferred method for detecting and following AAAs

RISK FACTORS

Risk factors for AAA formation

Smoking is the strongest independent acquired risk factor for AAA.[15] Wilmink et

TABLE 2

Variables associated with abdominal aortic aneurysm rupture in the UK Small Aneurysm Trial

VARIABLE	HAZARD RATIO	
	INITIAL ANALYSIS*	SECONDARY ANALYSIS*
Female sex	3.00	4.50
AAA diameter (per cm)	2.94	2.51
Current smoker	1.00	2.11
Mean blood pressure (per mm Hg)	1.02	1.04
Age (per year)	1.03	1.02
Forced expiratory volume in 1 second (per L)	0.62	—

*The initial analysis included 2,257 patients, some of whom refused surgery or were unfit for surgery. Of the aneurysms, some were smaller than 4.0 cm or larger than 5.5 cm in diameter; 103 ruptured.
The secondary analysis included a homogenous group of 1,090 fit patients with AAAs 4.0 to 5.5 cm in diameter, of which 25 ruptured. Follow-up was 7 years, and patients' ages ranged between 60 and 76 years at entry. All baseline variables were adjusted for one another by Cox regression analysis. People who never smoked and ex-smokers were combined and compared with current smokers.
All of the hazard ratios were statistically significant ($P < .05$) except for those for age in both the initial and secondary analyses.

DATA FROM BROWN LC, POWELL JT, THE UK SMALL ANEURYSM TRIAL PARTICIPANTS. RISK FACTORS FOR RUPTURE IN PATIENTS KEPT UNDER ULTRASOUND SURVEILLANCE. ANN SURG 1999; 230:289–297.

al[16] showed that current smokers were 7.6 times more likely to have an AAA than nonsmokers. The longer one smoked the greater the risk, but the number of cigarettes smoked per day did not seem to matter after adjustment for smoking duration.

Age and family history are the strongest nonreversible risk factors. The prevalence of AAAs among first-degree relatives of patients with AAAs ranges from 15% to 29%, compared with 2% among relatives of controls.[17–19]

Other important acquired risk factors include hypertension, chronic obstructive pulmonary disease, and atherosclerotic disease in other vascular beds, eg, coronary artery disease, cerebrovascular disease, and peripheral arterial disease.

Risk factors for rapid AAA expansion

Size. A very slow expansion rate (mean 0.65 mm/year) was reported in a recent prospective study[20] of 116 patients with ectatic aortas (2–2.9 cm) followed by annual ultrasonography. The expansion rate was independent of the baseline vessel size. Although they expanded slowly, a surprising one fifth of the aortas (22 of the 116) became aneurysmal (diameter ≥ 3 cm) within 3 years.

In small AAAs (3–5.4 cm), the expansion rate appears to be proportional to the initial diameter. Other factors linked to rapid expansion include systolic hypertension, a wide pulse pressure, and ongoing smoking.[16,21–24] Once a small AAA is detected, serial ultrasonography may help determine if it is growing rapidly (> 0.47 cm/year) or slowly (< 0.23 cm/year).[25]

C-reactive protein levels have been found to be elevated in larger aneurysms but do not appear to be associated with rapid expansion.[26]

Risk factors for AAA rupture

Important independent variables for AAA rupture were identified in the UK Small Aneurysm Trial (TABLE 2).[27]

Size at diagnosis is one of the best predictors of rupture.[27–29] The risk increases substantially when the diameter exceeds 6 cm in men and 5 cm in women.[27,30]

In a subgroup analysis of the Aneurysm Detection and Management (ADAM) Veterans Affairs Cooperative Study,[31,32] the incidence of rupture at 1 year was 9.4% if the initial diameter was 5.5 to 5.9 cm, 10.2% if 6.0 to 6.9 cm, and 32.5% if 7.0 cm or larger.

Small aneurysms can rupture, however. In autopsy studies, up to 24% of ruptured AAAs were smaller than 4.0 cm.[18,33] Small aneurysms are threefold more likely to rupture in women.[34] These observations led investigators to explore whether local factors play a role in aneurysm instability.

Localized outpouchings seem to increase the aneurysm's vulnerability for rupture.[35]

Intramural thrombosis. Small case series suggest that aneurysm growth[36] and rupture[37] correlate with growth of the aneurysm's mural thrombus. Furthermore, the part of the aneurysm wall that is covered with thrombus was thinner and more often showed signs of focal anoxia, inflammation, apoptosis of smooth muscle cells, and degraded extracellu-

lar matrix than wall segments not covered by the thrombus, signifying a likely pathogenic role for mural thrombosis in aneurysmal wall instability.[38,39]

Rapid expansion. A multivariate analysis suggested that rapid expansion (≥ 1 cm/year), usually an indication for elective repair of asymptomatic small AAAs, was inferior to initial aneurysm size in predicting rupture.

SCREENING

A recent consensus statement from the Society of Vascular Surgery, the American Association of Vascular Surgery, and the Society of Vascular Medicine and Biology [40] recommended that all men age 60 to 85 years be screened for AAAs, as should women age 60 to 85 years with cardiovascular risk factors, and men and women older than 50 years with a family history of AAA.

The US Preventive Services Task Force (USPSTF) now recommends one-time screening for AAAs by ultrasonography in men age 65 to 75 years who have ever smoked.[41] This recommendation is rated as grade B ("The USPSTF found at least fair evidence that [screening] improves important health outcomes and concludes that benefits outweigh harms"). However, some experts feel this guideline should be more inclusive.

The topic of screening will be covered in detail in an upcoming article in this journal.

MANAGEMENT OF AAA

Medical management involves early detection, surveillance, and risk factor modification. Drugs that may retard AAA growth are under study.

AAA as a marker of vascular disease

The diagnosis of AAA signifies a need for intensive cardiovascular risk reduction.[4,42–45]

In the Cardiovascular Health Study,[46] after adjustment for various demographic and noninvasive markers of atherosclerosis, the relative risk for all-cause mortality at 4.5 years was 1.32 in participants with AAA; the relative risk of incident cardiovascular disease was 1.57.

In the UK Small Aneurysm Trial,[47] the size of the AAA before surgery predicted non-aneurysm-related perioperative morbidity and mortality due to cardiovascular causes even after adjustment for other risk factors, supporting the notion that AAA diameter is an independent marker of cardiovascular disease risk and mortality.

Similarly, patients with atherosclerosis in other arteries are prone to develop AAAs. In 561 patients with proven symptomatic peripheral arterial disease or cerebral arterial disease, MacSweeney et al[48] reported AAA prevalences as high as 11.2% in men and 6.4% in women. In patients with hemodynamically significant carotid artery stenosis, AAAs were found in 11% to 20% and were more common than in the control groups.[34,49–51]

AAAs can also indicate aneurysms elsewhere in the body. In 313 consecutive patients with AAAs,[52] 36 (12%), all men, had coexisting femoral and popliteal aneurysms. We routinely screen for iliac aneurysms as part of the AAA ultrasound examination and often, in men, also look at the femoral and popliteal arteries. Coronary artery aneurysms have also been reported in patients with AAA, but this correlation has not yet been established.[53,54]

Finding an AAA is an opportunity to start risk factor modification

Smoking and high blood pressure are among the most important reversible risk factors for AAA formation, expansion, and rupture.[27] Smoking cessation and management of hypertension are the mainstays of medical therapy. Given the pathogenic role that hypertension plays in the development and progression of AAA, one may consider keeping the blood pressure lower than the usual goal of 140/90 mm Hg, although specific data are lacking on this point. The goal for patients with diabetes or kidney disease is lower than 130/80 mm Hg.

Treatment of hyperlipidemia, although not proven to affect aneurysm expansion or rupture, is recommended for primary and secondary cardiovascular event reduction. Since AAA is a coronary risk equivalent, the goal low-density lipoprotein level should be the same as for patients with established coronary

Guidelines for AAA screeening:
Men age 60-85
Women 60-85 with cardiovascular risk factors
Men and women age 50+ with family history of AAA

artery disease, ie, lower than 100 mg/dL, with an optional goal of lower than 70 mg/dL.[55]

Antioxidants have not been shown to reduce the incidence or rupture rate of small AAAs,[56] but available trial data fall short of proving or disproving that they are beneficial.

Can medications retard AAA growth?

Antimetalloproteases such as doxycycline[57,58] and roxithromycin[59] may halt aneurysm expansion, but data are insufficient to recommend their routine use at this time.

Nonsteroidal anti-inflammatory drugs have similarly been suggested to be beneficial in small studies.

Beta-blockers have numerous benefits in patients with cardiovascular disease. They reduce aortic complications in patients with Marfan syndrome, and they slow the progression of AAAs in hypertensive patients. Three randomized trials[60–62] found that beta-blockers did not slow the growth rate of most small aneurysms, although fewer patients in the beta-blocker groups had rapid expansion rates (defined as > 0.75 cm/year).[60] Also, slower growth rates were noted in a beta-blocker subgroup who had aneurysms larger than 3.9 cm at enrollment,[61] suggesting that the medication had a small effect.

42% of patients with ruptured AAAs die before reaching the emergency room

In the absence of other indications for beta-blockers, the evidence is insufficient to recommend using them routinely for the sole purpose of slowing atherosclerotic aneurysm growth. We do use them, however, to control blood pressure in hypertensive AAA patients who tolerate them.

Preoperative assessment before AAA repair

Although advanced age is a risk factor for operative mortality,[63–65] the ADAM trial suggested that "biological age," as reflected by the condition of the vital organs such as the lungs, kidneys, and heart, was more important than the chronological age as a determinant of operative outcome.[31]

Preoperative cardiac testing may not identify all patients at higher operative risk from AAA repair.[66] Patients with poor renal and pulmonary function clearly have worse operative outcomes.[63,66–68] The mortality rate in one study was more than 20 times higher in patients with chronic obstructive pulmonary disease, elevated creatinine concentrations, or electrocardiographic evidence of ischemia than in patients without these abnormalities.[69]

Steyerberg et al[68] developed a preoperative risk score based on a meta-analysis of 16 trials and the experience of University Hospital Leiden, Netherlands. The score from different risk factors is added to the center-specific mortality score to estimate the risk of dying during AAA surgery.

Aortic factors implicated in postoperative morbidity and mortality include extensive atheromatous disease, mural calcification, thrombosis, juxtarenal extension of aneurysm, and inflammatory aortic aneurysms.[30] The increased risk resulted from longer suprarenal clamping time, need for complex dissection, and increased hemodynamic stresses.

When is the best time for AAA repair?

Elective repair is advocated in appropriate patients to avoid the catastrophic effects of AAA rupture. Forty-two percent of patients with ruptured AAAs die before reaching the emergency room, and an additional 50% don't make it from the emergency room to the operating room.[6,70] Moreover, the 30-day mortality rate after emergency surgical repair is as high as 50%.[71]

Aneurysmal size and the patient's fitness for surgery are the main determinants of timing and method of elective repair. Small aneurysms (4.0–5.5 cm) should be followed with serial ultrasound scans until they are large enough to justify repair (5.5 cm in men and 5 cm in women). All aneurysms larger than 6 cm should be repaired, if the patient consents to the procedure and his or her condition permits.[72]

TABLE 3 summarizes the two landmark randomized trials of elective surgical repair vs ultrasound surveillance for small aneurysms (< 5.5 cm). Even though surgical outcomes were better in the ADAM study[31] than in the UK Small Aneurysm Trial,[63] no significant difference in survival between the surgery and surveillance groups was noted. Notably, most patients in the ADAM surveillance group eventually required surgery: up to 81% of those with aneurysms between 5.0 and 5.4 cm on the initial ultrasound examination.[31]

TABLE 3

Randomized trials of surveillance vs repair of asymptomatic small abdominal aortic aneurysms

	UK SMALL ANEURYSM TRIAL[34,37]	ANEURYSM DETECTION AND MANAGEMENT (ADAM) VA COOPERATIVE STUDY[31]
Aneurysm size	4–5.5 cm	4–5.5 cm
No. of subjects	1,090 (902 men, 188 women)	1,136 (98–99.6% men)
Age range (years)	60–76	50–79
Mean follow-up (years)	Initial: 4.6 (range 2.6–6.9) Extended: 8 (range 6–10)	4.9 (range 3.5–8.0)
Analysis	Intention to treat	Intention to treat
Results	No difference in overall survival in short term or long term Possible benefit of early surgery for younger patients and those with larger aneurysms Lower total mortality with early surgery after 8 years, possibly because more patients in the early surgery group adopted life-style modifications	No difference in overall survival No benefit in any age group, aneurysm size
Remarks	High postoperative mortality rate (5.8%) may have diminished the benefit of early surgery 75% of the patients had AAAs < 5 cm At 10 years, 74% of early surveillance group had undergone open repair 27 patients had endovascular repair; censoring their data did not affect the survival curves	Operative mortality rate 2.7%

In women the decision regarding time of repair is more complex, since women have smaller aortic wall diameters compared with men,[73] have a three to four times higher risk of rupture with AAAs smaller than 5.5 cm,[34,74] and have worse outcomes following elective and emergency surgical repair. The available data are not enough to justify setting the threshold diameter for surgery lower for women, but some authors advocate using the infrarenal-to-suprarenal diameter ratio as measured by imaging techniques other than ultrasonography.[27]

With the uncertainties surrounding rupture risk, operative risk, and the individual patient's life expectancy, the decision of timing and type of repair should involve the patient's preference and true informed consent. The joint committee of the Joint Council of the American Association of Vascular Surgery and the Society for Vascular Surgery recommended careful surveillance for aneurysms smaller than 5.5 cm that are growing slower than 1 cm/year.[30] Although aneurysms smaller than 5.5 cm may be electively treated in younger patients, women, and short men, it is reasonable to watch an aneurysm of the same size in high-risk patients, especially if endovascular repair is not feasible.

Open surgery or endovascular repair?

The choice of open surgery vs endovascular repair (ie, catheter-based placement of a stent graft to exclude the AAA) depends on the patient's condition, preference, and life expectancy, and the surgeon's experience.

Advances in endovascular repair make it

an attractive option, particularly for patients deemed poor candidates for open surgery. Endovascular repair is safe and effective for AAAs and is gaining acceptance from patients and physicians.[75–79] Although there was no overall mortality benefit of endovascular repair in trials of older devices, newer devices are showing an encouraging trend. The early lack of overall mortality benefit was likely due to selection bias (sicker, older patients were enrolled in the initial trials). As endovascular devices and procedures are refined, durability is likely to improve and the cost and psychological burden of the mandatory surveillance may be decreased, making it a standard procedure.

In two recent large randomized trials,[81,82] patients who underwent endovascular repair fared better than those who underwent open repair with regard to operative (30-day) mortality and other complications. The jury is still out, however, regarding the long-term outcomes, as the baseline characteristics of enrollees and the skill of the operator and the center appear to have a lot to do with such outcome measures. As such, it is important to take into consideration the expertise and results at the individual medical center.

High-risk patients with cardiac or pulmonary comorbidities are candidates for endovascular repair, which is better tolerated than open repair.[82] Endovascular repair is also an option for the "biologically old" or those with "hostile abdomens" or other clinical conditions that increase the risk of postoperative complications with open surgery, provided that the anatomy is appropriate. It is important to note that nephrotoxicity associated with use of contrast material is a real risk, particularly in patients with borderline renal function.

Although endovascular repair has lower early rates of operative morbidity and mortality, the results with open surgery have proven durability. Therefore, open surgery may be preferred for younger patients at low operative risk.[83] Although aneurysm-related deaths will likely be reduced with endovascular repair, the absence of an overall mortality benefit and the need for indefinite surveillance are perceived barriers to its widespread use beyond the special populations outlined above. Advances in endovascular devices and technique, however, will enhance its role.

The availability of endovascular repair should not change the thresholds for AAA repair beyond those recommended by the available guidelines.

REFERENCES

1. **Johnston KW, Rutherford RB, Tilson MD, Shah DM, Hollier L, Stanley JC.** Suggested standards for reporting on arterial aneurysms. J Vasc Surg 1991; 13:452–458.
2. **Pyo R, Lee JK, Shipley JM, et al.** Targeted gene disruption of matrix metalloproteinase-9 (gelatinase B) suppresses development of experimental abdominal aortic aneurysms. J Clin Invest 2000; 105:1641–1649.
3. **Lopez-Candales A, Holmes DR, Liao S, Scott MJ, Wickline SA, Thompson RW.** Decreased vascular smooth muscle cell density in medial degeneration of human abdominal aortic aneurysms. Am J Pathol 1997; 150:993–1007.
4. **Lederle FA, Johnson GR, Wilson SE, et al.** Prevalence and associations of aortic abdominal aneurysm detected through screening. Aneurysm Detection and Management (ADAM) Veterans Affairs Cooperative Study Group. Ann Intern Med 1997; 126:441–449.
5. **Melton LJ 3rd, Bickerstaff LK, Hollier LH, et al.** Changing incidence of abdominal aortic aneurysm: a population-based study. Am J Epidemiol 1984; 120:379–386.
6. **Bickerstaff LK, Hollier LH, Van Peenen HJ, Melton LJ III, Pairolero PC, Cherry KJ.** Abdominal aortic aneurysm: the changing natural history. J Vasc Surg 1984; 1:6–12.
7. **Melton LJ, Ilstrup DM, Riggs BL, et al.** Fifty-year trend in hip fracture incidence. Clin Orthop 1982; 162:144–149.
8. **Lederle FA, Johnson GR, Wilson SE, et al, for the Aneurysm Detection and management (ADAM) Veterans Affairs Cooperative Study Group.** The Aneurysm Detection and Management Study screening program validation cohort and final results. Arch Intern Med 2000; 160:1425–1430.
9. **Lederle FA, Johnson GR, Wilson SE.** Aneurysm Detection and Management Veterans Affairs Cooperative Study. Abdominal aortic aneurysm in women. J Vasc Surg 2001; 34:122–126.
10. **Fisher DF Jr, Yawn DH, Crawford ES.** Preoperative disseminated intravascular coagulation associated with aortic aneurysms. A prospective study of 76 cases. Arch Surg 1983; 118:1252–1255.
11. **Lederle FA, Simel DL.** Does this patient have abdominal aortic aneurysm? JAMA 1999; 281:77–82.
12. **Fink HA, Lederle FA, Roth CS, Bowles CA, Nelson DB, Haas MA.** The accuracy of physical examination to detect abdominal aortic aneurysm. Arch Intern Med 2000; 160:833–836.
13. **Scott R, Ashton HA, Kay DN.** Abdominal aortic aneurysm in 4237 screened patients: prevalence, development, and management over 6 years. Br J Surg 1991; 78:1122–1125.
14. **Petersen M, Cambria RP, Kaufman JA, et al.** Magnetic resonance angiography in the preoperative evaluation of abdominal aortic aneurysms. J Vasc Surg 1995; 21:891–898.
15. **Strachan DP.** Predictors of death from aortic aneurysm among middle-aged men: the Whitehall study. Br J Surg 1991; 78:401–404.
16. **Wilmink TBM, Quick CRG, Day NE.** The association between cigarette smoking and abdominal aortic aneurysms. J Vasc Surg 1999; 30:1099–1105.
17. **Johansen K, Koepsell T.** Familial tendency of abdominal aortic aneurysms. JAMA 1986; 256:1934–1936.
18. **Darling RC 3rd, Brewster DC, Darling RC, et al.** Are familial abdominal aortic aneurysms different? J Vasc Surg 1989; 10:39–43.
19. **Webster MW, Ferrel RE, St Jean PL, Majumder PP, Fogel SR, Steed DL.** Ultrasound screening of first-degree relatives of patients with abdominal aortic aneurysm. J Vasc Surg 1991; 13:9–13.
20. **Basnyat PS, Aiono S, Warsi AA, Magee TR, Galland RB, Lewis MH.**

Natural history of the ectatic aorta. Cardiovasc Surg 2003; 11:273–276.
21. **Chang JB, Stein TA, Liu JP, Dunn ME.** Risk factors associated with rapid growth of small abdominal aortic aneurysms. Surgery 1997; 121:117–122.
22. **MacSweeney STR, Ellis M, Worrell PC, Greenhalgh RM, Powell JT.** Smoking and growth rate of small abdominal aortic aneurysms. Lancet 1994; 344:651–652.
23. **Vardulaki KA, Prevost TC, Walker NM, et al.** Growth rates and risk of rupture of abdominal aortic aneurysms [erratum appears in Br J Surg 1999; 86:280]. Br J Surg 1998; 85:1674–1680.
24. **Vardulaki KA, Prevost TC, Walker NM, et al.** Incidence among men of asymptomatic abdominal aortic aneurysms: estimates from 500 screen detected cases. J Med Screen 1999; 6:50–54.
25. **Schewe CK, Schweikart HP, Hammel G, Spengel FA, Zollner N, Zoller WG.** Influence of selective management on the prognosis and the risk of rupture of abdominal aortic aneurysms. Clin Invest 1994; 72:585–591.
26. **Norman P, Spencer CA, Lawrence-Brown MM, Jamrozik K.** C-reactive protein levels and the expansion of screen-detected abdominal aortic aneurysms in men. Circulation 2004; 110:862–866.
27. **Brown LC, Powell JT, The UK Small Aneurysm Trial Participants.** Risk factors for rupture in patients kept under ultrasound surveillance. Ann Surg 1999; 230:289–297.
28. **Guirguis EM, Barber GG.** The natural history of abdominal aortic aneurysms. Am J Surg 1991; 162:481–483.
29. **Limet R, Sakalihassan N, Albert A.** Determination of the expansion rate and incidence of rupture of abdominal aortic aneurysms. J Vasc Surg 1991; 14:540–548.
30. **Brewster DC, Cronenwett JL, Hallett JW Jr, et al.** Guidelines for the treatment of abdominal aortic aneurysms. Report of a subcommittee of the Joint Council of the American Association for Vascular Surgery and Society for Vascular Surgery. J Vasc Surg 2003; 37:1106–1117.
31. **Lederle FA, Wilson SE, Johnson GR, et al.** Immediate repair compared with surveillance of small abdominal aortic aneurysms. N Engl J Med 2002; 346:1437–1444.
32. **Lederle FA, Johnson GR, Wilson SE, et al.** Rupture rate of large abdominal aortic aneurysms in patients refusing or unfit for elective repair. JAMA 2002; 287:2968–2972.
33. **Bengtsson H, Ekberg O, Aspelin P, Takolander R, Bergqvist D.** Abdominal aortic dilatation in patients operated on for carotid artery stenosis. Acta Chir Scand 1988; 154:441–445.
34. **The UK Small Aneurysm Trial Participants.** Mortality results for randomised controlled trial of early elective surgery or ultrasonographic surveillance for small abdominal aortic aneurysms. Lancet 1998; 352:1649–1655.
35. **Faggioli GL, Stella A, Gargiulo M, Tarantini S, D'Addato M, Ricotta JJ.** Morphology of small aneurysms: definition and impact on risk of rupture. Am J Surg 1994; 168:131–135.
36. **Wolf YG, Thomas WS, Brennan FJ, Goff WG, Sise MJ, Bernstein EF.** Computed tomography scanning findings associated with rapid expansion of abdominal aortic aneurysms [erratum appears in J Vasc Surg 1995; 21:295]. J Vasc Surg 1994; 20:529–535.
37. **Stenbaek J, Kalin B, Swedenborg J.** Growth of thrombus may be a better predictor of rupture than diameter in patients with abdominal aortic aneurysms. Eur J Vasc Endovasc Surg 2000; 20:466–469.
38. **Vorp DA, Lee PC, Wang DH, et al.** Association of intraluminal thrombus in abdominal aortic aneurysm with local hypoxia and wall weakening. J Vasc Surg 2001; 34:291–299.
39. **Kazi M, Thyberg J, Religa P, et al.** Influence of intraluminal thrombus on structural and cellular composition of abdominal aortic aneurysm wall. J Vasc Surg 2003; 38:1283–1292.
40. **Kent KC, Zwolak RM, Jaff MR, et al.** Screening for abdominal aortic aneurysm: A consensus statement. J Vasc Surg 2004; 39:267–269.
41 **U.S. Preventive Services Task Force.** Screening for abdominal aortic aneurysm: recommendation statement. Ann Intern Med 2005; 142:198–202.
42. **Alcorn HG, Wolfson SK Jr, Sutton-Tyrrell K, Kuller LH, O'Leary D.** Risk factors for abdominal aortic aneurysms in older adults enrolled in the Cardiovascular Health Study. Arterioscler Thromb Vasc Biol 1996; 16:963–970.
43. **Blanchard JF, Armenian HK, Friesen PP.** Risk factors for abdominal aortic aneurysm: results of a case-control study. Am J Epidemiol 2000; 151:575–583.
44. **Plumeekers HJ, Hoes AW, van der Does E, et al.** Aneurysms of the abdominal aorta in older adults. The Rotterdam Study. Am J Epidemiol 1995; 142:1291–1299.
45. **Naydeck BL, Sutton-Tyrrell K, Schiller KD, Newman AB, Kuller LH.** Prevalence and risk factors for abdominal aortic aneurysms in older adults with and without isolated systolic hypertension. Am J Cardiol 1999; 83:759–764.
46. **Newman AB, Arnold AM, Burke GL, O'Leary DH, Manolio TA.** Cardiovascular disease and mortality in older adults with small abdominal aortic aneurysms detected by ultrasonography: a cardiovascular health study. Ann Intern Med 2001; 134:182–190.
47. **Brady AR, Fowkes FG, Thompson SG, Powell JT.** Aortic aneurysm diameter and risk of cardiovascular mortality. Arterioscler Thromb Vasc Biol 2001; 21:1203–1207.
48. **MacSweeney STR, O'Meara M, O'Malley MK, Powell JT, Greenhalgh RM.** High prevalence of unsuspected abdominal aortic aneurysm in patients with confirmed symptomatic peripheral or cerebral arterial disease. Br J Surg 1993; 80:582–584.
49. **Carty GA, Nachtigal T, Magyar R, Herzler G, Bays R.** Abdominal duplex ultrasound screening for occult aortic aneurysm during carotid arterial evaluation. J Vasc Surg 1993; 17:696–702.
50. **Karanjia PN, Madden KP, Lobner S.** Coexistence of abdominal aortic aneurysm in patients with carotid stenosis. Stroke 1994; 25:627–630.
51. **Kang SS, Littooy FN, Gupta SR, et al.** Higher prevalence of abdominal aortic aneurysms in patients with carotid stenosis but without diabetes. Surgery 1999; 126:687–692.
52. **Diwan A, Sarkar R, Stanley JC, Zelenock GB, Wakefield TW.** Incidence of femoral and popliteal artery aneurysms in patients with abdominal aortic aneurysms. J Vasc Surg 2000; 31:863–869.
53. **Davi R, Marchese F, Borghesi MR, Romanelli R.** Multiple coronary artery aneurysms of an atherosclerotic nature in a patient with an abdominal aortic aneurysm: presentation of an anatomo-clinical case. Pathologica 1997; 89:304–309.
54. **Peter O, Ozisik K, Islamoglu F, Posacioglu H, Demircan M.** Multiple coronary artery aneurysms combines with abdominal aortic aneurysm. Jpn Heart J 2001; 42:135–141.
55. **Grundy SM, Cleeman JI, Merz CN, et al; Coordinating Committee of the National Cholesterol Education Program.** Implications of recent clinical trials for the National Cholesterol Education Program Adult Treatment Panel III Guidelines. J Am Coll Cardiol 2004; 44:720–732.
56. **Tornwall ME, Virtamo J, Haukka JK, Albanes D, Huttunen JK.** Alpha-tocopherol (vitamin E) and beta-carotene supplementation do not affect the risk for large abdominal aortic aneurysm in a controlled trial. Atherosclerosis 2001; 157:167–173.
57. **Baxter BT, Pearce WH, Waltke EA, et al.** Prolonged administration of doxycycline in patients with small asymptomatic abdominal aortic aneurysms: report of a prospective (phase II) multicentre study. J Vasc Surg 2002; 36:1–12.
58. **Mosorin M, Juvonen J, Biancari F, et al.** Use of doxycycline to decrease the growth rate of abdominal aortic aneurysms: a randomized, double-blinded, placebo-controlled pilot study. J Vasc Surg 2001; 34:606–610.
59. **Vammen S, Lindholt JS, Ostergaard L, Fasting H, Henneberg EW.** Randomized double-blind controlled trial of roxithromycin for prevention of abdominal aortic aneurysm expansion. Br J Surg 2001; 88:1066–1072.
60. **Propranolol Aneurysm Trial Investigators.** Propranolol for small abdominal aortic aneurysms: results of a randomized trial. J Vasc Surg 2002; 35:72–79.
61. **Wilmink ABM, Hubbard CSFF, Day NE, Quick CRG.** Effect of propranolol on the expansion of abdominal aortic aneurysms: a randomized study [abstract]. Br J Surg 2000; 87:499.
62. **Lindholt JS, Henneberg EW, Juul S, Fasting H.** Impaired results of a randomised double blinded clinical trial of propranolol versus place-

bo on the expansion rate of small abdominal aortic aneurysms. Int Angiol 1999; 18:52–57.
63. **Brady AR, Fowkes FG, Greenhalgh RM, Powell JT, Ruckley CV, Thompson SG.** Risk factors for postoperative death following elective surgical repair of abdominal aortic aneurysm: results from the UK Small Aneurysm Trial. Br J Surg 2000; 87:742–749.
64. **Akkersdijk GJM, van der Graaf Y, van Bockel JH, de Vries AC, Eikelboom BC.** Mortality rates associated with operative treatment of infrarenal abdominal aortic aneurysms in the Netherlands. Br J Surg 1994; 81:706–709.
65. **Dardik A, Lin JW, Gordan TA, Williams GM, Perler BA.** Results of elective abdominal aortic aneurysm repair in the 1990s: a population-based analysis of 2335 cases. J Vasc Surg 1999; 30:985–995.
66. **D'Angelo AJ, Puppala D, Farber A, Murphy AE, Faust GR, Cohen JR.** Is preoperative cardiac evaluation for abdominal aortic aneurysm repair necessary? J Vasc Surg 1997; 25:152–156.
67. **Yasuhara H, Ishiguro T, Muto T.** Factors affecting late survival after elective abdominal aortic aneurysm repair. Br J Surg 1999; 86:1047–1052.
68. **Steyerberg EW, Kievit J, de Mol Van Otterloo JC, van Bockel JH, Eijkemans MJ, Habbema JD.** Perioperative mortality of elective abdominal aortic aneurysm surgery: a clinical prediction rule based on literature and individual patient data. Arch Intern Med 1995; 155:1998–2004.
69. **Johnston KW.** Multicenter prospective study of nonruptured abdominal aortic aneurysm. Part II. Variables predicting morbidity and mortality. J Vasc Surg 1989; 9:437–447.
70. **Dueck AD, Johnston KW, Alter D, Laupacis A, Kucey DS.** Predictors of repair and effect of gender on treatment of ruptured abdominal aortic aneurysm. J Vasc Surg 2004; 39:784–787.
71. **Bradbury AW, Makhdoomi KR, Adam DJ, Murie JA, Jenkins AM, Ruckley CV.** Twelve-year experience of the management of ruptured abdominal aortic aneurysm. Br J Surg 1997; 84:1705–1707.
72. **Powell JT GR, Ruckley CV, Fowkes FGR.** Prologue to a surgical trial. Lancet 1993; 342:1473–1474.
73. **Simpson CF.** Sotalol for the protection of turkeys from the development of aminopropionitrile-induced aortic ruptures. Br J Pharmacol 1972; 45:385–390.
74. **The United Kingdom Small Aneurysm Trial Participants.** Long-term outcomes of immediate repair compared with surveillance for small abdominal aortic aneurysms. N Engl J Med 2002; 346:1445–1452.
75. **Matsumura JS, Brewster DC, Makaroun MS, Naftel DC, for the Excluder Bifurcated Endoprosthesis Investigators.** A multicenter controlled clinical trial of open versus endovascular treatment of abdominal aortic aneurysm. J Vasc Surg 2003; 37:262–271.
76. **Brewster DC, Geller SC, Kaufman JA, et al.** Initial experience with endovascular aneurysm repair: comparison of early results with outcome of conventional open repair. J Vasc Surg 1998; 27:992–1003.
77. **May J, White GH, Yu W, et al.** Concurrent comparison of endoluminal versus open repair in treatment of abdominal aortic aneurysms: analysis of 303 patients by life-table method. J Vasc Surg 1998; 27:213–221.
78. **Zarins CK, White RA, Schwarten D, et al.** AneuRx stent graft versus open surgical repair of abdominal aortic aneurysms: multicenter prospective clinical trial. J Vasc Surg 1999; 29:292–308.
79. **Moore WS, Brewster DC, Bernhard VM, for the EVT/Guidant Investigators.** Aorto-uni-iliac endograft for complex aortoiliac aneurysms compared with tube/bifurcation endografts: results of the EVT/Guidant trials. J Vasc Surg 2001; 33:S11–S20.
81. **Greenhalgh RM, Brown LC, Kwong GP, Powell JT, Thompson sG;** EVAR trial participants. Comparison of endovascular aneurysm repair with open repair in patients with abdominal aortic aneurysm (EVAR trial 1), 30-day operative mortality results: randomised controlled trial. Lancet 2004; 364:843–848.
82. **Prinssen M, Verhoeven EL, Buth J, et al;** Dutch Randomized Endovascular Aneurysm Management (DREAM) Trial Group. A randomized trial comparing conventional and endovascular repair of abdominal aortic aneurysms. N Engl J Med 2004; 351:1607–1618.
82. **Hertzer NR, Mascha EJ, Karafa MT, O'Hara PJ, Krajewski LP, Beven EG.** Open infrarenal abdominal aortic aneurysm repair: the Cleveland Clinic experience from 1989 to 1998. J Vasc Surg 2002; 35:1145–1154.
83. **Schermerhorn ML, Finlayson SR, Fillinger MF, Buth J, van Marrewijk C, Cronenwett JL.** Life expectancy after endovascular versus open abdominal aortic aneurysm repair: results of a decision analysis model on the basis of data from EUROSTAR. J Vasc Surg 2002; 36:1112–1120.

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