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# How best to diagnose and manage abdominal aortic aneurysms

## The evidence summarized here can help guide your approach to this life-threatening condition that often goes undetected until rupture.

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### PRACTICE RECOMMENDATIONS

› *Perform a one-time abdominal aortic aneurysm (AAA) screening ultrasound in men ages 65 to 75 years who have ever smoked.* (B)

› *Consider performing a one-time AAA screening ultrasound in women ages 65 to 75 years who have ever smoked.* (C)

› *Prescribe high-intensity statin therapy for men and women with atherosclerotic AAA.* (A)

**Strength of recommendation (SOR)**

(A) Good-quality patient-oriented evidence

(B) Inconsistent or limited-quality patient-oriented evidence

(C) Consensus, usual practice, opinion, disease-oriented evidence, case series

Ruptured abdominal aortic aneurysms (AAAs) caused about 6000 deaths annually in the United States between 2014 and 2020[1] and are associated with a pooled mortality rate of 81%.[2] They result from a distinct degenerative process of the layers of the aortic wall.[2] An AAA is defined as an abdominal aorta whose dilation is > 50% normal (more commonly, a diameter > 3 cm).[3,4] The risk for rupture correlates closely with size; most ruptures occur in aneurysms > 5.5 cm[3,4] (**TABLE 1**[5]).

Most AAAs are asymptomatic and often go undetected until rupture, resulting in poor outcomes. Because of a low and declining prevalence of AAA and ruptured AAA in developed countries, screening recommendations target high-risk groups rather than the general population.[4,6-8] This review summarizes risk factors, prevalence, and current evidence-based screening and management recommendations for AAA.

## Who's at risk?

Age is the most significant nonmodifiable risk factor, with AAA rupture uncommon in patients younger than 55 years.[9] One retrospective study found the odds ratio (OR) for diagnosing AAA was 9.41 in adults ages 65 to 69 years (95% CI, 8.76-10.12; $P < .0001$) and 14.46 (95% CI, 13.45-15.55; $P < .0001$) in adults ages 70 to 74 years, compared to adults younger than 55 years.[10]

Smoking is the most potent modifiable risk factor for AAA. Among patients with AAA, > 90% have a history of smoking.[4] The association between smoking and AAA is dose dependent, with an OR of 2.61 (95% CI, 2.47-2.74) in patients with a pack-per-year history < 5 years and 12.13 (95% CI, 11.66-12.61) in patients with a pack-per-year history > 35 years, compared to nonsmokers.[10] The risk for AAA increases with smoking duration but decreases with cessation duration.[4,10] Smoking cessation remains an important intervention, as active smokers have higher AAA rupture rates.[11]

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**TABLE 1**

## Annual aneurysm rupture risk based on aortic diameter at baseline[5]

| Aneurysm size | Annual rupture risk |
|---|---|
| 3.0-3.9 cm | Nearly 0 |
| 4.0-4.9 cm | Up to 1% |
| 5.0-5.9 cm | 1%-11% |
| 6.0-6.9 cm | 10%-22% |
| > 7 cm | 30%-33% |

Other risk factors for AAA include concomitant cardiovascular disease (CVD) such as coronary artery disease (CAD), cerebrovascular disease, atherosclerosis, dyslipidemia, and hypertension.[10] Factors associated with reduced risk for AAA include African American race, Hispanic ethnicity, Asian ethnicity, diabetes, smoking cessation, consuming fruits and vegetables > 3 times per week, and exercising more than once per week.[6,10]

## Prevalence declines but sex-based disparities in outcomes persist

The prevalence of AAA has declined in the United States and Europe in recent decades, correlating with declining rates of smoking.[4,12] Reports published between 2011 and 2019 estimate that AAA prevalence in men older than 60 years has declined over time, with a prevalence of 1.2% to 3.3%.[6] The prevalence of AAA has also decreased in women,[6,13,14] estimated in 1 study to be as low as 0.74%.[13] Similarly, deaths from ruptured AAA have declined markedly in the United States—by 70% between 1999 and 2016 according to 1 analysis.[9]

One striking difference in the male-female data is that although AAAs are more common in men, there is a 2- to 4-fold higher risk for rupture in women, who account for nearly half of all AAA-related deaths.[9,10,15-17] The reasons for this heightened risk to women despite lower prevalence are not fully understood but are likely multifactorial and related to a general lack of screening for AAA in women, tendency for AAA to rupture at smaller diameters in women, rupture at an older age in women, and a history of worse surgical outcomes in women than men (though the gap in surgical outcomes appears to be closing).[9,10,18]

While declines in AAA and AAA-related death are largely attributed to lower smoking rates, other likely contributing factors include the implementation of screening programs, incidental detection during cross-sectional imaging, and improved surgical techniques and management of CV risk factors (eg, hypertension, hyperlipidemia).[9,10]

### The benefits of screening older men

Randomized controlled trials (RCTs) have demonstrated the benefits of AAA screening programs. A meta-analysis of 4 population-based RCTs of AAA screening in men ≥ 65 years demonstrated statistically significant reductions in AAA rupture (OR = 0.62; 95% CI, 0.55-0.70) and death from AAA (OR = 0.65; 95% CI, 0.57-0.74) over 12 to 15 years, with a number needed to screen (NNS) of 305 (95% CI, 248-411) to prevent 1 AAA-related death.[18] The study also found screening decreases the rate of emergent surgeries for AAA (OR = 0.57; 95% CI, 0.48-0.68) while increasing the number of elective surgeries (OR = 1.44; 95% CI, 1.34-1.55) over 4 to 15 years.[18]

Only 1 study has demonstrated an improvement in all-cause mortality with screening programs, with a relatively small benefit (OR = 0.97; 95% CI, 0.94-0.99).[19] Only 1 of the studies included women and, while underpowered, showed no difference in AAA-related death or rupture.[20] Guidelines and recommendations of various countries and professional societies focus screening on subgroups at highest risk for AAA.[4,6-8,18]

## Screening recommendations from USPSTF and others

The US Preventive Services Task Force (USPSTF) currently recommends one-time ultrasound screening for AAA in men ages 65 to 75 years who have ever smoked (commonly defined as having smoked > 100 cigarettes) in their lifetime.[6] This grade "B" recommendation, initially made in 2005 and reaffirmed in the 2014 and 2019 USPSTF updates, recommends screening the highest-risk segment of the population (ie, older male smokers).[6]

In men ages 65 to 75 years with no smoking history, rather than routine screening, the USPSTF recommends selectively offering screening based on the patient's medical history, family history, risk factors, and personal values (with a "C" grade).[6] The USPSTF continues to recommend against screening for AAA in women with no smoking history and no family history of AAA.[6] According to the USPSTF, the evidence is insufficient to recommend for or against screening women ages 65 to 75 years who have ever smoked or have a family history of AAA ("I" statement).[6]

**One critique of the USPSTF recommendations** is that they fail to detect a significant portion of patients with AAA and AAA rupture. For example, in a retrospective analysis of 55,197 patients undergoing AAA repair, only 33% would have been detected by the USPSTF grade "B" recommendation to screen male smokers ages 65 to 75 years, and an analysis of AAA-related fatalities found 43% would be missed by USPSTF criteria.[9,21]

Screening guidelines from the Society for Vascular Surgery (SVS) are broader than those of the USPSTF, in an attempt to capture a larger percentage of the population at risk for AAA-related disease by extrapolating from epidemiologic data. The SVS guidelines include screening for women ages 65 to 75 years with a smoking history, screening men and women ages 65 to 75 years who have a first-degree relative with AAA, and consideration of screening patients older than 75 years if they are in good health and have a first-degree relative with AAA or a smoking history and have not been previously screened.[4] However, these expanded recommendations are not supported by patient-oriented evidence.[6]

Attempts to broaden screening guidelines must be tempered by potential risks for harm, primarily overdiagnosis (ie, diagnosing AAAs that would not otherwise rise to clinical significance) and overtreatment (ie, resulting in unnecessary imaging, appointments, anxiety, or surgery). Negative psychological effects on quality of life after a diagnosis of AAA have not been shown to cause significant harm.[6,18]

A recent UK analysis found that screening programs for AAA in women modeled after those in men are not cost effective, with an NNS to prevent 1 death of 3900 in women vs 700 in men.[15,18] Another recent trial of ultrasound screening in 5200 high-risk women ages 65 to 74 years found an AAA incidence of 0.29% (95% CI, 0.18%-0.48%) in which only 3 large aneurysms were identified.[22]

In the United States, rates of screening for AAA remain low.[23] One study has shown electronic medical record–based reminders increased screening rates from 48% to 80%.[24] Point-of-care bedside ultrasound performed by clinicians also could improve screening rates. Multiple studies have demonstrated that screening and diagnosis of AAA can be performed safely and effectively at the bedside by nonradiologists such as family physicians and emergency physicians.[25-28] In 1 study, such exams added < 4 minutes to the patient encounter.[26] Follow-up surveillance schedules for those identified as having a AAA are summarized in **TABLE 2**.[4]

## Management options: Immediate repair or surveillance?

After diagnosing AAA, important decisions must be made regarding management, including indications for surgical repair, appropriate follow-up surveillance, and medications for secondary prevention and cardiovascular risk reduction.

### EVAR vs open repair

The 2 main surgical strategies for aneurysm repair are open repair and endovascular repair (EVAR). In the United States, EVAR is becoming the more common approach and was used to repair asymptomatic aneurysms in > 80% of patients and ruptured aneurysms in 50% of patients.[6] There have been multiple RCTs assessing EVAR and open repair for large and small aneurysms.[29-34] Findings across these studies consistently show EVAR is associated with lower immediate (ie, 30-day) morbidity and mortality but no longer-term survival benefit compared to open repair.

EVAR procedures require ongoing long-term surveillance for endovascular leakage and other complications, resulting in an increased need for re-intervention.[31,33,35] For

**Smoking is the most potent modifiable risk factor for abdominal aortic aneurysm.**

**TABLE 2**

## Society for Vascular Surgery surveillance imaging recommendations[4]

| Aortic diameter on ultrasound | Recommended surveillance interval |
|---|---|
| Aorta > 2.5 but < 3.0 cm | Repeat ultrasound in 10 y |
| AAA 3.0 to 3.9 cm | Repeat ultrasound every 3 y |
| AAA 4.0 to 4.9 cm | Repeat ultrasound every 1 y |
| AAA 5.0 to 5.4 cm | Repeat ultrasound every 6 mo |

AAA, abdominal aortic aneurysm.

**TABLE 3**

## Indications for surgical repair of abdominal aortic aneurysm[4,7,34]

| Surgical indication | Evidence |
|---|---|
| Fusiform AAA with a diameter ≥ 5.5 cm[4] | Strong data from randomized controlled trials[4,7,34]; dependent on surgical risk and life expectancy |
| Women with an AAA 5.0-5.4 cm[4] | Weak/observational evidence based on the observed risk for rupture in women compared to men. |
| Saccular AAA identified at any size[4] | Less common presentation limits evidence; generally repaired upon detection, regardless of size. |
| AAA > 4 cm but expanding > 1 cm/y[4] | Considered an indication for repair in some randomized trials. |
| Symptomatic AAA[4] | Includes rupture and acute onset of abdominal or back pain that is otherwise unexplained and thought to be aneurysm related. |

AAA, abdominal aortic aneurysm.

**Although abdominal aortic aneurysms are more common in men, there is a 2- to 4-fold higher risk for rupture in women.**

these reasons, the National Institute for Health and Care Excellence (NICE) guidelines suggest open repair as the preferred modality.[7] However, SVS and the American College of Cardiology Foundation/American Heart Association guidance support either EVAR or open repair, noting that open repair may be preferable in patients unable to engage in long-term follow-up surveillance.[36]

**Indications for repair.** In general, repair is indicated when an aneurysm reaches or exceeds 5.5 cm.[4,7] Both SVS and NICE also recommend clinicians consider surgical repair of smaller, rapidly expanding aneurysms (> 1 cm over a 1-year period).[4,7] Based on evidence suggesting a higher risk for rupture in women with smaller aneurysms,[14,37] SVS recommends clinicians consider surgical repair in women with an AAA ≥ 5.0 cm. Several RCTs evaluating the benefits of immediate repair for smaller-sized aneurysms (4.0-5.5 cm) favored surveillance.[38,39] Accepted indications for surgical repair are summarized in **TABLE 3**.[4,7,34]

Surgical repair recommendations also are based on aneurysm morphology, which can be fusiform or saccular (**FIGURE**). More than 90% of AAAs are fusiform.[40] Although saccular AAAs are less common, some studies suggest they are more prone to rupture than fusiform AAAs, and SVS guidelines suggest surgical repair of saccular aneurysms regardless of size.[4,41,42]

**Perioperative and long-term risks.** Both EVAR and open repair of AAA carry a high perioperative and long-term risk for death, as patients often have multiple comorbidities. A 2019 trial comparing EVAR to open repair with 14 years of follow-up reported death in 68% of patients in the EVAR group and 70% in the open repair group.[31] Among these deaths, 2.7% in the EVAR group and 3.7% in the open repair group were aneurysm related.[31] The study also found a second surgical intervention was required in 19.8% of patients in the open repair group and 26.7% in the EVAR group.[31]

When assessing perioperative risk, SVS guidelines recommend clinicians employ a

shared decision-making approach with patients that incorporates Vascular Quality Initiative (VQI) mortality risk score.[4] (VQI risk calculators are available at https://qxmd.com/vascular-study-group-new-england-decision-support-tools.[43])

### Medication management

Based on the close association of aortic aneurysm with atherosclerotic CVD (ASCVD), professional societies such as the European Society of Cardiology and European Atherosclerosis Society (ESC/EAS) have suggested aortic aneurysm is equivalent to ASCVD and should be managed medically in a similar manner to peripheral arterial disease.[44] Indeed, many patients with AAA may have concomitant CAD or other arterial vascular diseases (eg, carotid, lower extremity).

**Statins.** In its guidelines, the ESC/EAS consider patients with AAA at "very high risk" for adverse CV events and suggest pharmacotherapy with high-intensity statins, adding ezetimibe or proprotein convertase subtilisin/kexin type 9 (PCSK9) inhibitors if needed, to reduce low-density lipoprotein cholesterol ≥ 50% from baseline, with a goal of < 55 mg/dL.[44] Statin therapy additionally lowers all-cause postoperative mortality in patients undergoing AAA repair but does not affect the rate of aneurysm expansion.[45]

**Aspirin and other anticoagulants.** Although aspirin therapy may be indicated for the secondary prevention of other cardiovascular events that may coexist with AAA, it does not appear to affect the rate of growth or prevent rupture of aneurysms.[46,47] In addition to aspirin, anticoagulants such as clopidogrel, enoxaparin, and warfarin are not recommended when the presence of AAA is the only indication.[4]

**Other medications.** Angiotensin-converting enzyme inhibitors, angiotensin receptor blockers, beta-blockers, and antibiotics (eg, doxycycline) have been studied as a treatment for AAA. However, none has shown benefit in reducing aneurysm growth or rupture and they are not recommended for that sole purpose.[4,48]

**Metformin.** There is a negative association between diabetes and AAA expansion and rupture. Several cohort studies have indicated that this may be an independent effect driven primarily by exposure to metformin. While it is not unreasonable to consider this another important indication for metformin use in patients with diabetes, RCT evidence has yet to establish a role for metformin in patients without diabetes who have AAA.[48,49] JFP

FIGURE

## Fusiform vs saccular aneurysms: How they look

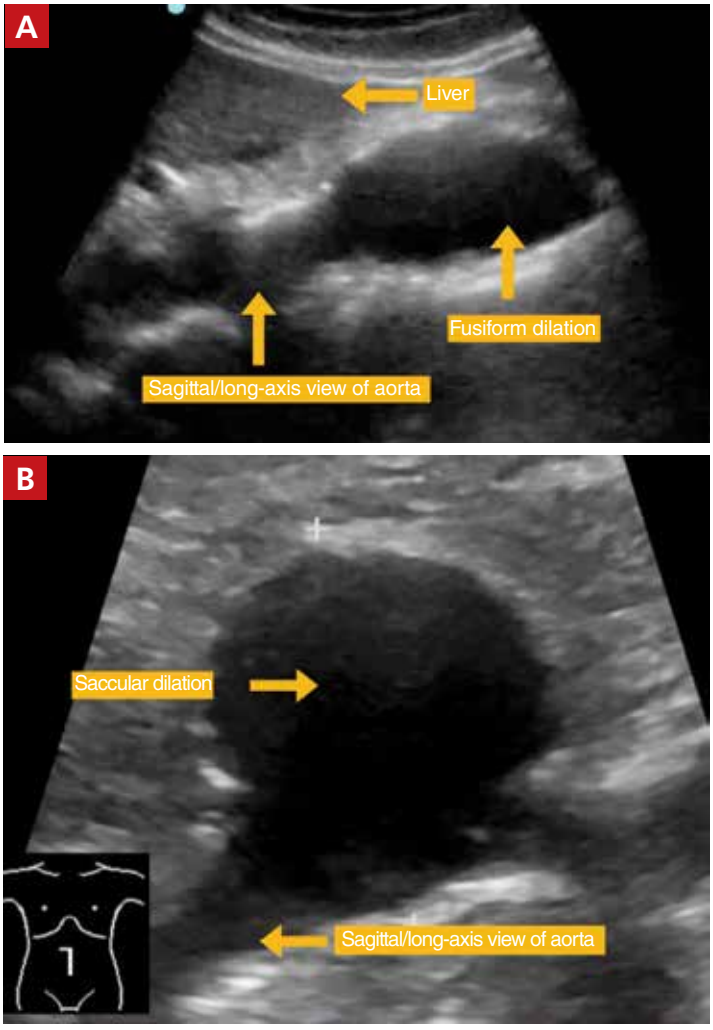


IMAGE COURTESY OF MIKAEL HÄGGSTRÖM, CC0, VIA WIKIMEDIA COMMONS

Long-axis views showing fusiform dilation (**A**) and saccular dilation (**B**) of the abdominal aorta.

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**The USPSTF continues to recommend against screening in women with no smoking history and no family history of abdominal aortic aneurysm.**

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# EDITORIAL

CONTINUED FROM PAGE 324

got a long way to go.

Might salt substitution at the population level be a way to simultaneously reduce our sodium intake and increase our potassium intake?[12] The closest I found to a population-wide substitution study was a cluster randomized trial conducted in 6 villages in Peru.[13] In a stepped-wedge design, households had 25% of their regular salt replaced with potassium salt. Small shops, bakeries, community kitchens, and food vendors also had salt replacement. The intention-to-treat analysis showed a small reduction in systolic BP (1.3 mm Hg) among those with hypertension at baseline (n = 428) and a 51% reduced incidence of developing hypertension among the other 1891 participants over the 4673 person-years of follow-up.

I found this study interesting and its results compelling, leading me to wonder: In the United States, where most of our sodium comes from the food industry, should we replace even a small amount of the sodium in processed foods with potassium? We're not getting there with DASH alone. JFP

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