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PERIPHERAL VASCULAR DISEASE (WS JONES, SECTION EDITOR)

Contemporary Management of Abdominal Aortic Aneurysms

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Abstract

Purpose of Review Abdominal aortic aneurysms (AAA) can carry extremely high mortality rates and most will only present with symptoms with impending rupture. We present an overview of management of this disease process starting with screening, to medical management, surveillance and treatment options currently available, as well as those being studied for future use.

Recent Findings Screening has been proven to reduce the mortality rate. There still remains a paucity of data to support medical therapies to help mitigate the rate of aneurysm growth and prevent rupture. However, on the topic of repair, there have been advancements in endovascular devices which have broadened the scope of treatment for patients with anatomy not amenable to standard endovascular repair or those who are not suitable candidates for open surgical repair.

Summary Appropriate surveillance, risk factor modification, and operative repair, when indicated, are the cornerstones of contemporary management of AAAs. Advancements in endovascular technologies have allowed us to treat more patients. Further research is warranted on non-operative medical therapies.

Keywords Abdominal aortic aneurysm · AAA · Aneurysm rupture · Aneurysm growth · Aortic aneurysm · EVAR

Introduction

The natural history of abdominal aortic aneurysms (AAAs) can carry mortality rates upwards of 88% after rupture and in 2017 was the etiology of more than 9900 deaths in the USA [1, 2]. The management of AAAs has evolved significantly from the pioneering efforts of proximal ligation with Cooper and Matas, and arterial wrapping championed by Poppe [3]. Most aneurysms only present with symptoms with impending rupture. Outside of this, it is essentially a silent disease making early diagnosis and management critical to the goal of rupture prevention. Appropriate surveillance, risk factor modification, and operative repair, when indicated, are the cornerstones of contemporary management of AAAs.

Background

Aneurysms can be defined as any abnormal dilatation greater than 50% of normal arterial diameter. They are further defined by their morphology as fusiform, concentric saccular, or eccentric saccular. While the average aortic size varies by location of the aorta and by gender, the abdominal aorta has classically been viewed to have a normal diameter less than 3 cm [4]. The precise mechanism of aneurysmal degeneration is complex and not fully elucidated, but general understanding involves smooth muscle apoptosis and degeneration of the media [5].

The most feared complication of AAAs is rupture as it can lead to immediate mortality through rapid exsanguination into the retroperitoneal space and peritoneal cavity. The Law of Laplace articulates the relationship between wall stress, diameter, and pressure, suggesting that increased aneurysm size leads to increased risk of rupture due to increased wall stress placed on the already damaged endothelium [6].

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The prevalence of AAAs ranges from 4 to 8% in screening studies, affecting predominantly males [7, 8]. Some risk factors for the development of AAA include advanced age, male gender, presence of other arterial aneurysms, family history, as well as hypertension and smoking which are the most modifiable risk factors for development.

Screening

Early detection plays a critical role in reducing the associated mortality of AAAs due to the dramatically reciprocal survival outcomes observed after elective repair compared to that after rupture. The Multicenter Aneurysm Screening Study (MASS) is a commonly referenced multicenter randomized control trial including 67,770 patients which concluded that ultrasound screening for AAAs in men ages 65 to 74 leads to a reduction in number of deaths related to AAA after 10 years [9].

The US Preventive Task Force recommends a one-time screening with duplex ultrasound for all men ages 65 to 75 who have a smoking history and selectively to those of the same age who have never smoked [10]. While they cite insufficient evidence to offer screening to women, the Society of Vascular Surgery (SVS) carries broader recommendations in both gender and age. They recommend a one-time screening ultrasound to both men and women ages 65 to 75 with a history of smoking, those over the age of 75 with a history of smoking, but in good health, as well as those over the age of 65 with a first-degree relative with an AAA [11•]. It should be noted that the latter two groups carry weaker recommendations with lower quality of evidence.

Medical Management

Once the diagnosis of an AAA is made, either through a screening ultrasound or as an incidental finding on another imaging study, medical management to help reduce the risk of aneurysm growth and rupture becomes paramount. The two main modifiable risk factors for this are smoking and hypertension. While there is strong evidence that demonstrates smoking increases the risk of aneurysm development and growth, there have been no studies that have proven the degree of efficacy of smoking cessation's effect on the mitigation of growth once an aneurysm is present [12, 13].

Similarly, despite the wealth of evidence that hypertension is strongly associated with aneurysm development, a 2019 meta-analysis found no difference in the growth rate of aneurysms between patients with hypertension and those without [14]. The limitation of this study is that the determination of which patients were in the hypertension cohort versus non-hypertension cohort appears to be based solely on a diagnosis. This does not confirm that the patients in the hypertension group had adequate control of their blood pressure, nor does it rule out undiagnosed hypertension patients being present in the non-hypertension cohort. Thus, it is still our practice to recommend smoking cessation and management of hypertension with lifestyle modifications and antihypertensive medications when indicated in patients with aneurysmal disease.

There are currently no medications that have been proven to reduce the rate of growth or prevent rupture. While there has been some data that supports this concept in non-human subjects, this has not been reproducible in humans. Data recently published from a randomized clinical trial demonstrated no benefit in reducing the rate of aneurysm growth in patients treated with doxycycline [15]. An observational study found no association between aneurysm growth in patients taking beta blockers, angiotensin-converting enzyme (ACE) inhibitors and angiotensin II receptor blockers (ARBs), though there was a negative association found in patients with a concomitant diagnosis of diabetes mellitus [16]. Further research is warranted to investigate other non-invasive medical therapies to treat aneurysmal disease.

Surveillance and Treatment Indications

Regular surveillance is required to monitor for aneurysm growth. This follow-up is critical in rupture prevention as the risk of presenting emergently with a rupture was six times higher in patients who have gaps in their surveillance [17]. Intervals vary based on AAA size with current guidelines recommending surveillance imaging every 3 years for aneurysms between 3.0 cm and 3.9 cm, every 12 months for those between 4.0 cm and 4.9 cm, and every 6 months for those between 5.0 cm and 5.4 cm [11•], Evidence has shown that smaller aneurysms grow slower than larger aneurysms with a median growth rate of 1.9 mm per year for aneurysms under 4 cm, 2.7 mm per year for those between 4.0 cm and 4.5 cm, and 3.5 mm per year for aneurysms measuring 4.6 cm or greater [18].

As aneurysms increase in size, the risk for rupture increases as well. Respectively, aneurysms measuring 4 cm to 5 cm carry an annual rupture risk of 0.5 to 5%, 5 to 6 cm with 3 to 15% risk, 6 to 7 cm with 10 to 20% risk, 7 to 8 cm with 20 to 40% risk, and aneurysms measuring greater than 8 cm carry an annual rupture risk of 30 to 50% [19]. Rapid growth of an aneurysm is defined by an increase in size of 0.5 cm within 6 months or 1 cm within 12 months. If there is evidence of rapid growth, or an aneurysm reaches the size of 5.5 cm in males or 5.0 cm in females, elective repair is recommended. The difference in threshold size for repair between genders is due to the increased risk for rupture

in women, notwithstanding the smaller size of the normal abdominal aorta in women [20].

The threshold of 5.5 cm and 5.0 cm in men and women, respectively, has held up to further evaluation in the era of endovascular repair. We still do not see any survival benefit of repairing smaller aneurysms. Both the Aneurysm Detection and Management Veterans Affairs Cooperative Study (ADAM) and the UK Small Aneurysm Trial (UKSAT) demonstrated that there was no benefit noted for repair of aneurysms smaller than 5.5 cm [21, 22]. This was also seen in the Comparison of Surveillance Versus Aortic Endografting for Small Aneurysm Repair Trial (CAESAR) that examined endovascular repair of smaller aneurysms [23]. Again, in all these trials, an early survival disadvantage to the operative group due to the risks of surgery versus surveillance was noted. However, UKSAT noted survival curves of the surveillance group and the operative group of aneurysms under 5.5 cm eventually crossed at the 3-year mark [24].

Aside from patients with evidence of rapid growth or those who eventually reach the threshold size for repair, there are several other indications for treatment regardless of the aneurysm size. These include patients presenting with rupture or symptoms concerning for impending rupture, those with thromboembolic events secondary to mural thrombus within the aneurysm, patients experiencing mass effect and compression of vital structures in the abdomen, or those with fistulization and hemorrhage into other hollow viscus structures (i.e. bowel, ureter, etc.). There are two types of treatment available — open repair and endovascular aortic repair (EVAR). The decision to pursue one over the other can be impacted by a variety of factors including the indication to treat, anatomy, concomitant comorbidities, and patient preference.

Patients presenting with rupture or impending rupture should be treated with the most expedited repair modality possible, understanding that not every facility will have endovascular capabilities readily available. It is recommended that all hospitals have a protocol in place to streamline management and treatment of ruptured AAA patients with a goal of door-to-OR time less than 90 min [11•].

Open Repair

Overview

Prior to the advent of endovascular repair, open repair was the only modality available for the treatment of AAAs. This entails accessing the abdominal aorta through either a midline transperitoneal approach or left retroperitoneal exposure, and replacement of the diseased segment with either a prosthetic or homograft conduit. The graft is sewn to non-diseased aorta proximal to the aneurysmal segment. A beveled anastomosis can be used to preserve the native visceral vessels for aneurysms that extend into this region. For those that involve a significant portion of, or the entire visceral segment, individual visceral bypasses may be indicated. The location of the distal anastomosis can also vary depending on the caudal extent of aneurysmal disease. For aneurysms that terminate within the abdominal aorta, a tube graft may be used with the distal anastomosis being placed just proximal to the aortic bifurcation. For aneurysms that abut or entail the bifurcation, or in patients with concomitant iliac aneurysms, a bifurcated graft may be used with the distal anastomoses being placed in the distal iliac or proximal femoral system. The redundant aneurysm sac is repurposed to provide full coverage of the graft, serving as a biologic barrier to reduce the risk of aortoenteric fistula formation.

Outcomes

To fully elucidate the outcomes of open AAA repair, it should be noted that multiple patient risk factors and intraoperative techniques must be considered. The SVS, through use of the Vascular Quality Initiative (VQI), has outlined risk factors that one must be cognizant of when assessing overall perioperative morbidity and mortality. These include patient risk factors of advanced age, chronic obstructive pulmonary disease (COPD), renal dysfunction and prior aortic surgery, as well as operative risk factors of suprarenal or supraceliac proximal clamp placement [11•].

Historically, mortality rates from elective openAAA repair have been as high as 8%, but contemporary analyses demonstratemortality rates from 2% to 5% in multi-institutional studies as outlined in Table 1 [25–33]. These findings are similar to the open arms found in the large randomized controlledtrials comparing open versus endovascular aortic repair [34–36].

Many of these patients have significant baseline comorbidities and thus perioperative complications are inevitable. In an analysis of Medicare claims data comparing endovascular versus open repair of AAAs, Schermerhorn et al. found that of the 22,830 patients who had undergone open repair, 9.4% sustained a postoperative myocardial infarction (MI) and 10.9% suffered acute renal failure with 0.5% needing dialysis [30]. The rate of acute renal failure and need for dialysis increases up to 20% and 3.5%, respectively, for those with aneurysms involving the visceral segment [37]. Colonic ischemia is an uncommon, but well-known complication that needs to be monitored for as it can be found at a rate of 0.2 to 6% [38–42].

After AAA repair, patients will still require some degree of surveillance. For those treated with open repair, it is recommended that a computed tomography angiography (CTA) be obtained five years postoperatively. Long term, there is the risk of developing anastomotic aneurysms and

Table 1 Mortality of elective open AAA repair

Authors	Publication year	Number of patients	In-hospital mortality (non-ruptured)
Akkersdijk et al. [25]	1994	1289	6.8%
Bradbury et al. [26]	1998	1515	6.1%
Dardik et al. [27]	1999	2335	3.5%
Bayly et al. [28]	2001	933	7.3%
Rigberg et al. [29]	2006	12,406	3.8%
Schermerhorn et al. [30]	2008	22,830	4.8%
Teixeira et al. [31]	2016	3530	2.7%
Latz et al. [32]	2021	957	4.6% for juxtarenal; 9.5% for suprarenal; 14.7% for type IV TAAA
Sharma et al. (33)	2021	3078	4.1% 30 day

degeneration of the native aorta adjacent to the graft. These can occur at a rate of 1% at 5 years, 5% at 10 years, and 20% at 15 years [43, 44].

Endovascular Repair

Overview

Unlike open repair where the aneurysm sac is removed, the principal of endovascular repair is based on exclusion of the aneurysm sac. This is accomplished by deployment of an intraluminal stent graft that seals proximal and distal to the aneurysm therefore diverting flow through the graft and removing pressure off the sac that could lead to rupture. Wire access is first obtained via the bilateral common femoral arteries, either percutaneously or through open exposure. Sheaths are placed in the groins through which the stent grafts are delivered and subsequently deployed. Balloon angioplasty can be utilized to ensure good apposition at the proximal and distal landing zones. Completion aortogram is performed to identify any endoleaks (Table 2). If a Type I or Type III endoleak is identified, those should be addressed at that time. If percutaneous access was utilized, these sites are closed with a closure device, while open exposures require primary repair of the arteriotomy.

Table 2 Types of endoleaks

Type	Definition
Type I	IA – Leak from inadequate the proximal seal of graft IB – Leak from inadequate the distal seal of graft IC – Leak at the distal seal of side branch graft
Type II	Leak into aneurysm sac from branch collateral vessels
Type III	IIIA – Leak from separation or inadequate seal of overlapping modular components IIIB – Leak from a tear of defect in the fabric IIIC – Leak from the junction between aortic graft and side branch graft
Type IV	Leak into the aneurysm sac due to the porosity of the graft fabric/material
Type V	Aneurysm sac growth with of evidence of endoleak

Prior to proceeding with endovascular aortic repair, it is prudent to obtain multi-dimensional imaging to ensure suitable anatomy. This is often performed with a CTA, specifically with thin arterial slices assessing the distal thoracic aorta down to the femoral vessels; magnetic resonance angiography (MRA) can also be used if there are contraindications to the patient obtaining a CTA [45]. Once images are obtained, a detailed analysis is performed through a variety of three-dimensional reconstruction software. In our institution we prefer Aquarius TeraRecon© in order to adequately size the endograft. Centerline measurements are obtained to ensure there is adequate neck length, angle and diameter in the proximal neck, as well as in the distal iliac landing zones. While there are several commercially available grafts with varying Instructions for Use (IFU) for anatomic criteria, in general the proximal neck length should be at least 10 to 15 mm in length with an angulation of less than 60° and a diameter of 16 to 32 mm. For the distal iliac landing zones, a length of 20 mm and a diameter of 6 to 25 mm is generally acceptable.

For patients treated with endovascular repair, postoperative surveillance intervals are more frequent than those treated with open repair. A CTA is recommended one month postoperatively, followed by a repeat scan 6 months later, then annually thereafter. These scans are critical in diagnosing endoleaks that may necessitate repair. The imaging modality may be switched from CTA to duplex ultrasound in the absence of an endoleak or sac expansion, and when feasible with the patient's body habitus [11•].

Outcomes

The most common complication after EVAR is an endoleak and this serves as the most frequent indication for reintervention [46]. Rates of endoleaks can vary based on differences between standards for reporting, follow up intervals and study lengths. That being said, it's widely accepted that

Type II endoleaks are the most common with a rate of up to 20% of patients being diagnosed on a postoperative CTA [47, 48]. These only require repair if they are contributing to residual sac size growth. The natural history of Type II endoleaks tends to be more benign compared to Type I or Type III endoleaks which are found to occur at a lower rate [49]. Interestingly, a meta-analysis of 28,862 patients who had undergone EVAR found a pooled estimate of 10.5% of patients that had developed a Type I endoleak over the lifetime of their follow up [50]. Other complications from EVAR include graft limb occlusion which can impact up to 7.2% of patients and stent graft migration which has previously been found to occur in 8.6% of patients after EVAR, though it should be noted that patients with less favorable proximal landing zone anatomy and those treated with older generation stent grafts were at higher risk [51, 52]. Similar to the rate of endoleaks, reintervention rates also vary greatly between studies. Wanken et al. performed a systematic review and meta-analysis of 32,126 patients who had undergone endovascular repair and found reintervention rates of 19% at 5 years, 30% at 10 years and 35% at 14 years [53•]. When stratified by year, these rates improved with later implantation dates, likely reflective of advancements with newer stent grafts.

Several landmark studies have demonstrated favorable 30-day mortality rates with EVAR including the Endovascular Aneurysm Repair Trial 1 (EVAR-1) at 1.7%, the Dutch Randomized Endovascular Aneurysm Management Trial (DREAM) at 1.2%, and the Lifeline Endovascular Registry at 1.7% [34, 36, 54]. One recent meta-analysis found a higher pooled estimate of 30-day mortality at a rate of 3.3% [50]. Multiple randomized controlled trials comparing survival in open repair versus endovascular aortic repair demonstrated that endovascular repair reduces perioperative mortality [35, 55, 56]. However, a recent meta-analysis which included these landmark trials, found the early EVAR group advantage was eroded progressively and by 3 years after aneurysm repair, aneurysm-related mortality was five times higher in the EVAR group, mainly due to secondary rupture or reinterventions [57].

Advancements in Endovascular Repair

Juxtarenal and paravisceral abdominal aortic aneurysms were previously precluded from endovascular repair due to coverage of visceral vessels and insufficient proximal and distal landing zones. Furthermore, there is data to support that aggressive use of infrarenal devices in patients with hostile landing zone anatomy results in worse outcomes [58, 59]. To that end, the advent of commercially available fenestrated endografts has allowed for an endovascular approach to complex aortic pathologies such as aneurysms with short aortic neck length and visceral involvement.

In the US, there is currently only one FDA-approved commercially available fenestrated endograft which is manufactured by Cook Medical (Bloomington, Indiana). These grafts are custom designed to patient specific anatomy and thus require meticulous preoperative planning. The use of three-dimensional reconstruction software, such as Aquarius TeraRecon©, is critical as it allows for exacting measurements of the distance between the visceral vessels, their diameters, and radial orientation. While there are limitations to the number, location, and size of the fenestrations, as well as the maximal angulation of the aorta, such grafts are becoming increasingly more utilized. These custom endografts often require several weeks to produce, and thus are not readily available in emergent situations. The reader is directed to other sources for detailed understanding of the complexity of implantation of such endografts [60, 61].

Given the relatively new adoption of fenestrated endografts, multicenter randomized controlled trials regarding their efficacy are limited. Motta et al. evaluated 150 patients undergoing fenestrated endovascular aortic repair (FEVAR) and found an early mortality rate of 2.7% and a branch patency rate of 97% at two years [62]. This suggests endovascular repair of complex aneurysms is safe and effective when performed in a high aortic disease volume center. Jones et al. published a meta-analysis in 2019 comparing short-term and long-term outcomes of FEVAR and open repair of juxtarenal aortic aneurysms [63]. Twenty-seven studies were identified, involving 2974 patients. Early postoperative mortality rate following FEVAR was 3.3%, compared with 4.2% after open repair. They found FEVAR to have better outcomes of renal insufficiency (16.2% vs 23.8%) and major early complication (23.1% vs 43.5%), but noted a significantly higher incidence of reintervention (11.1% vs 2%) when compared to open repair. This suggests that short-term mortality is improved with FEVAR, but questions remain about its long-term durability and reintervention rate. With the increasing use of fenestrated endografts, more multicenter and subsequently randomized trials will need to be performed to further characterize long-term outcomes, but current results do seem promising.

With the initial success of endograft implantation for treatment of aortic aneurysms, it is no surprise that a similar technique has been employed for common iliac, external iliac, and internal iliac artery aneurysms. These iliac-branched endografts, commonly referred to as "IBEs", have been designed exclusively for the treatment of iliac artery aneurysms. There is currently only one FDA-approved commercially available device that is manufactured by W. L. Gore and Associates (Flagstaff, Arizona). A recent study assessing the GORE® EXCLUDER® Iliac Branch Endoprosthesis illustrated a primary patency rate of 91% coupled with a low reintervention rate of 3% at 1 year [64]. Further research is warranted to evaluate long-term efficacy of such endografts and the role they may play in mitigating the incidence of complications that may be a result of reduced

pelvic perfusion, such as gluteal claudication, sexual dysfunction and spinal cord collateralization. The latter is beneficial to patients who have aneurysmal disease elsewhere in the aorta who have had or may need further treatment.

Conclusion

Contemporary management of AAAs has evolved over the decades with improvements in early detection, routine surveillance, risk profile modification, and advances in repair modalities. As a result, the mortality rate associated with AAAs has been declining over the past two decades and at a faster rate than ever before [65•]. There has been an increase in AAA detection with the implementation of screening programs. Furthermore, there is evidence supporting even broader screening criteria to include women and those with first-degree relatives with history of an aneurysm. While the technical aspect of open repair remains largely unchanged, we have made advancements in perioperative management of these patients and the associated complications. The area of endovascular repair has seen the greatest amount of change as the technology surrounding these endografts has evolved rapidly. This has allowed for a wider range of patients with complex aortic aneurysms to be eligible for endovascular repair than previously allowed. Similar to other areas of surgery, trends are moving toward more minimally invasive therapeutics. As such, advancements in endovascular technology will eventually lead to complete endovascular solutions for aortic pathology. Ultimately, the goal is to find ways to prevent or treat aneurysmal disease non-operatively, akin to the way peptic ulcer disease is now prevented and treated medically, despite previously requiring surgical intervention. Until this is achieved, continued screening, surveillance, risk factor management and operative repair when indicated is paramount in the management of AAAs.

Compliance with Ethical Standards

Conflict of Interest The authors declare that they have no conflict of interest.

Human and Animal Rights and Informed Consent This article does not contain any studies with human or animal subjects performed by any of the authors.

References

Papers of particular interest, published recently, have been highlighted as:
• Of importance

1. Virani SS, Alonso A, Benjamin EJ, Bittencourt MS, Callaway CW, Carson AP, et al. Heart disease and stroke statistics-2020 update: a report from the American Heart Association. Circulation. 2020;141(9):e139–596.
2. Bengtsson H, Bergqvist D. Ruptured abdominal aortic aneurysm: a population-based study. J Vasc Surg. 1993;18(1):74–80.
3. Tracci M, Rishi, RA, Upchurch, GR. Aortoiliac Aneurysms: Evaluation, Decision Making, and Medical Management. In: Sidawy A, Perler, BA, editor. Rutherford's Vascular Surgery and Endovascular Therapy. 9th ed: Elsevier, Inc. 2019;884–93.e3.
4. Erbel R, Eggebrecht H. Aortic dimensions and the risk of dissection. Heart. 2006;92(1):137–42.
5. Quintana RA, Taylor WR. Cellular mechanisms of aortic aneurysm formation. Circ Res. 2019;124(4):607–18.
6. Vorp DA. Biomechanics of abdominal aortic aneurysm. J Biomech. 2007;40(9):1887–902.
7. Norman PE, Jamrozik K, Lawrence-Brown MM, Le MT, Spencer CA, Tuohy RJ, et al. Population based randomised controlled trial on impact of screening on mortality from abdominal aortic aneurysm. BMJ. 2004;329(7477):1259.
8. Ashton HA, Gao L, Kim LG, Druce PS, Thompson SG, Scott RA. Fifteen-year follow-up of a randomized clinical trial of ultrasonographic screening for abdominal aortic aneurysms. Br J Surg. 2007;94(6):696–701.
9. Ashton HA, Buxton MJ, Day NE, Kim LG, Marteau TM, Scott RA, et al. The Multicentre Aneurysm Screening Study (MASS) into the effect of abdominal aortic aneurysm screening on mortality in men: a randomised controlled trial. Lancet. 2002;360(9345):1531–9.
10. Force USPST, Owens DK, Davidson KW, Krist AH, Barry MJ, Cabana M, et al. Screening for abdominal aortic aneurysm: US preventive services task force recommendation statement. JAMA. 2019;322(22):2211–8.
11.• Chaikof EL, Dalman RL, Eskandari MK, Jackson BM, Lee WA, Mansour MA, et al. The Society for Vascular Surgery practice guidelines on the care of patients with an abdominal aortic aneurysm. J Vasc Surg. 2018;67(1):2–77 e2. **Aside from serving as a comprehensive outline of AAA management, these updated guidelines provide updates and new recommendations on surveillance intervals, antibiotic prophylaxis and postoperative surveillance imaging modalities.**
12. Buckley C, Wyble CW, Borhani M, Ennis TL, Kobayashi DK, Curci JA, et al. Accelerated enlargement of experimental abdominal aortic aneurysms in a mouse model of chronic cigarette smoke exposure. J Am Coll Surg. 2004;199(6):896–903.
13. Blanchard JF, Armenian HK, Friesen PP. Risk factors for abdominal aortic aneurysm: results of a case-control study. Am J Epidemiol. 2000;151(6):575–83.
14. Takagi H, Umemoto T, Group A. Association of hypertension with abdominal aortic aneurysm expansion. Ann Vasc Surg. 2017;39:74–89.
15. Baxter BT, Matsumura J, Curci JA, McBride R, Larson L, Blackwelder W, et al. Effect of doxycycline on aneurysm growth among patients with small infrarenal abdominal aortic aneurysms: a Randomized Clinical Trial. JAMA. 2020;323(20):2029–38.
16. Lederle FA, Noorbaloochi S, Nugent S, Taylor BC, Grill JP, Kohler TR, et al. Multicentre study of abdominal aortic aneurysm measurement and enlargement. Br J Surg. 2015;102(12):1480–7.
17. Mell MW, Baker LC, Dalman RL, Hlatky MA. Gaps in preoperative surveillance and rupture of abdominal aortic aneurysms among Medicare beneficiaries. J Vasc Surg. 2014;59(3):583–8.
18. Brady AR, Thompson SG, Fowkes FG, Greenhalgh RM, Powell JT, Participants UKSAT. Abdominal aortic aneurysm expansion: risk factors and time intervals for surveillance. Circulation. 2004;110(1):16–21.

19. Brewster DC, Cronenwett JL, Hallett JW, Jr., Johnston KW, Krupski WC, Matsumura JS, 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(5):1106–17.
20. Norman PE, Powell JT. Abdominal aortic aneurysm: the prognosis in women is worse than in men. Circulation. 2007;115(22):2865–9.
21. Lederle FA, Wilson SE, Johnson GR, Reinke DB, Littooy FN, Acher CW, et al. Immediate repair compared with surveillance of small abdominal aortic aneurysms. N Engl J Med. 2002;346(19):1437–44.
22. Powell JT, Brown LC, Forbes JF, Fowkes FG, Greenhalgh RM, Ruckley CV, et al. Final 12-year follow-up of surgery versus surveillance in the UK Small Aneurysm Trial. Br J Surg. 2007;94(6):702–8.
23. Cao P, De Rango P, Verzini F, Parlani G, Romano L, Cieri E, et al. Comparison of surveillance versus aortic endografting for small aneurysm repair (CAESAR): results from a randomised trial. Eur J Vasc Endovasc Surg. 2011;41(1):13–25.
24. Brown LC, Thompson SG, Greenhalgh RM, Powell JT, Participants UKSAT. Fit patients with small abdominal aortic aneurysms (AAAs) do not benefit from early intervention. J Vasc Surg. 2008;48(6):1375–81.
25. Akkersdijk GJ, van der Graaf Y, van Bockel JH, de Vries AC, Eikelboom BC. Mortality rates associated with operative treatment of infrarenal abdominal aortic aneurysm in The Netherlands. Br J Surg. 1994;81(5):706–9.
26. Bradbury AW, Adam DJ, Makhdoomi KR, Stuart WP, Murie JA, Jenkins AM, et al. A 21-year experience of abdominal aortic aneurysm operations in Edinburgh. Br J Surg. 1998;85(5):645–7.
27. Dardik A, Lin JW, Gordon 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(6):985–95.
28. Bayly PJ, Matthews JN, Dobson PM, Price ML, Thomas DG. In-hospital mortality from abdominal aortic surgery in Great Britain and Ireland: Vascular Anaesthesia Society audit. Br J Surg. 2001;88(5):687–92.
29. Rigberg DA, Zingmond DS, McGory ML, Maggard MA, Agustin M, Lawrence PF, et al. Age stratified, perioperative, and one-year mortality after abdominal aortic aneurysm repair: a statewide experience. J Vasc Surg. 2006;43(2):224–9.
30. Schermerhorn ML, O'Malley AJ, Jhaveri A, Cotterill P, Pomposelli F, Landon BE. Endovascular vs. open repair of abdominal aortic aneurysms in the Medicare population. N Engl J Med. 2008;358(5):464–74.
31. Teixeira PG, Woo K, Abou-Zamzam AM, Zettervall SL, Schermerhorn ML, Weaver FA. The impact of exposure technique on perioperative complications in patients undergoing elective open abdominal aortic aneurysm repair. J Vasc Surg. 2016;63(5):1141–6.
32. Latz CA, Boitano L, Schwartz S, Swerdlow N, Dansey K, Varkevisser RRB, et al. Editor's choice - mortality is high following elective open repair of complex abdominal aortic aneurysms. Eur J Vasc Endovasc Surg. 2021;61(1):90–7.
33. Sharma G, Madenci AL, Wanis KN, Comment LA, Lotto CE, Shah SK, et al. Association and interplay of surgeon and hospital volume with mortality after open abdominal aortic aneurysm repair in the modern era. J Vasc Surg. 2021;73(5):1593–602 e7.
34. Greenhalgh RM, Brown LC, Kwong GP, Powell JT, Thompson SG, participants Et. 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(9437):843–8.
35. Lederle FA, Freischlag JA, Kyriakides TC, Padberg FT Jr, Matsumura JS, Kohler TR, et al. Outcomes following endovascular vs open repair of abdominal aortic aneurysm: a randomized trial. JAMA. 2009;302(14):1535–42.
36. Prinssen M, Verhoeven EL, Buth J, Cuypers PW, van Sambeek MR, Balm R, et al. A randomized trial comparing conventional and endovascular repair of abdominal aortic aneurysms. N Engl J Med. 2004;351(16):1607–18.
37. Nathan DP, Brinster CJ, Jackson BM, Wang GJ, Carpenter JP, Fairman RM, et al. Predictors of decreased short- and long-term survival following open abdominal aortic aneurysm repair. J Vasc Surg. 2011;54(5):1237–43.
38. Zwolak RM, Sidawy AN, Greenberg RK, Schermerhorn ML, Shackelton RJ, Siami FS, et al. Lifeline registry of endovascular aneurysm repair: open repair surgical controls in clinical trials. J Vasc Surg. 2008;48(3):511–8.
39. Bjorck M, Troeng T, Bergqvist D. Risk factors for intestinal ischaemia after aortoiliac surgery: a combined cohort and case-control study of 2824 operations. Eur J Vasc Endovasc Surg. 1997;13(6):531–9.
40. Brewster DC, Franklin DP, Cambria RP, Darling RC, Moncure AC, Lamuraglia GM, et al. Intestinal ischemia complicating abdominal aortic surgery. Surgery. 1991;109(4):447–54.
41. Longo WE, Lee TC, Barnett MG, Vernava AM, Wade TP, Peterson GJ, et al. Ischemic colitis complicating abdominal aortic aneurysm surgery in the U.S. veteran. J Surg Res. 1996;60(2):351–4.
42. Becquemin JP, Majewski M, Fermani N, Marzelle J, Desgrandes P, Allaire E, et al. Colon ischemia following abdominal aortic aneurysm repair in the era of endovascular abdominal aortic repair. J Vasc Surg. 2008;47(2):258–63. Discussion 63.
43. Edwards JM, Teefey SA, Zierler RE, Kohler TR. Intraabdominal paraanastomotic aneurysms after aortic bypass grafting. J Vasc Surg. 1992;15(2):344–50. Discussion 51–3.
44. Ylonen K, Biancari F, Leo E, Rainio P, Salmela E, Lahtinen J, et al. Predictors of development of anastomotic femoral pseudoaneurysms after aortobifemoral reconstruction for abdominal aortic aneurysm. Am J Surg. 2004;187(1):83–7.
45. Goshima S, Kanematsu M, Kondo H, Kawada H, Kojima T, Sakurai K, et al. Preoperative planning for endovascular aortic repair of abdominal aortic aneurysms: feasibility of nonenhanced MR angiography versus contrast-enhanced CT angiography. Radiology. 2013;267(3):948–55.
46. Schlosser FJ, Muhs BE. Endoleaks after endovascular abdominal aortic aneurysm repair: what one needs to know. Curr Opin Cardiol. 2012;27(6):598–603.
47. Sheehan MK, Ouriel K, Greenberg R, McCann R, Murphy M, Fillinger M, et al. Are type II endoleaks after endovascular aneurysm repair endograft dependent? J Vasc Surg. 2006;43(4):657–61.
48. Buth J, Harris PL, van Marrewijk C, Fransen G. The significance and management of different types of endoleaks. Semin Vasc Surg. 2003;16(2):95–102.
49. van Marrewijk C, Buth J, Harris PL, Norgren L, Nevelsteen A, Wyatt MG. Significance of endoleaks after endovascular repair of abdominal aortic aneurysms: the EUROSTAR experience. J Vasc Surg. 2002;35(3):461–73.
50. Franks SC, Sutton AJ, Bown MJ, Sayers RD. Systematic review and meta-analysis of 12 years of endovascular abdominal aortic aneurysm repair. Eur J Vasc Endovasc Surg. 2007;33(2):154–71.
51. Cochennec F, Becquemin JP, Desgranges P, Allaire E, Kobeiter H, Roudot-Thoraval F. Limb graft occlusion following EVAR: clinical pattern, outcomes and predictive factors of occurrence. Eur J Vasc Endovasc Surg. 2007;34(1):59–65.
52. Spanos K, Karathanos C, Saleptsis V, Giannoukas AD. Systematic review and meta-analysis of migration after endovascular abdominal aortic aneurysm repair. Vascular. 2016;24(3):323–36.

53.• Wanken ZJ, Barnes JA, Trooboff SW, Columbo JA, Jella TK, Kim DJ, et al. A systematic review and meta-analysis of long-term reintervention after endovascular abdominal aortic aneurysm repair. J Vasc Surg. 2020;72(3):1122–31. **This article reinforces the importance of lifelong surveillance for patients who have had endovascular repair. It also highlights the impact that advancements in endovascular technology has had with newer stent grafts demonstrating lower reintervention rates.**
54. Lifeline Registry of EPC. Lifeline registry of endovascular aneurysm repair: long-term primary outcome measures. J Vasc Surg. 2005;42(1):1–10.
55. United Kingdom ETI, Greenhalgh RM, Brown LC, Powell JT, Thompson SG, Epstein D, et al. Endovascular versus open repair of abdominal aortic aneurysm. N Engl J Med. 2010;362(20):1863–71.
56. De Bruin JL, Baas AF, Buth J, Prinssen M, Verhoeven EL, Cuypers PW, et al. Long-term outcome of open or endovascular repair of abdominal aortic aneurysm. N Engl J Med. 2010;362(20):1881–9.
57. Powell JT, Sweeting MJ, Ulug P, Blankensteijn JD, Lederle FA, Becquemin JP, et al. Meta-analysis of individual-patient data from EVAR-1, DREAM, OVER and ACE trials comparing outcomes of endovascular or open repair for abdominal aortic aneurysm over 5 years. Br J Surg. 2017;104(3):166–78.
58. Abbruzzese TA, Kwolek CJ, Brewster DC, Chung TK, Kang J, Conrad MF, et al. Outcomes following endovascular abdominal aortic aneurysm repair (EVAR): an anatomic and device-specific analysis. J Vasc Surg. 2008;48(1):19–28.
59. Chaikof EL, Fillinger MF, Matsumura JS, Rutherford RB, White GH, Blankensteijn JD, et al. Identifying and grading factors that modify the outcome of endovascular aortic aneurysm repair. J Vasc Surg. 2002;35(5):1061–6.
60. de Niet A, Reijnen MM, Tielliu IF, Lardenoije JW, Zeebregts CJ. Fenestrated endografts for complex abdominal aortic aneurysm repair. Surg Technol Int. 2016;29:220–30.
61. Oderich GS, Mendes BC, Kanamori KS. Technique of implantation and bail-out maneuvers for endovascular fenestrated repair of juxtarenal aortic aneurysms. Perspect Vasc Surg Endovasc Ther. 2013;25(1–2):28–37.
62. Motta F, Crowner JR, Kalbaugh CA, Marston WA, Pascarella L, McGinigle KL, et al. Outcomes and complications after fenestrated-branched endovascular aortic repair. J Vasc Surg. 2019;70(1):15–22.
63. Jones AD, Waduud MA, Walker P, Stocken D, Bailey MA, Scott DJA. Meta-analysis of fenestrated endovascular aneurysm repair versus open surgical repair of juxtarenal abdominal aortic aneurysms over the last 10 years. BJS Open. 2019;3(5):572–84.
64. van der Veen D, Holewijn S, Bellosta R, van Sterkenburg SMM, Heyligers JMM, Ficarelli I, et al. One year outcomes of an international multicentre prospective cohort study on the gore excluder iliac branch endoprosthesis for aorto-iliac aneurysms. Eur J Vasc Endovasc Surg. 2021;62(2):177–85.
65.• Png CYM, Wu J, Tang TY, Png IPL, Sheng TJ, Choke E. Editor's choice - decrease in mortality from abdominal aortic aneurysms (2001 to 2015): Is it decreasing even faster? Eur J Vasc Endovasc Surg. 2021;61(6):900–7. **This article highlights the successful efforts made in reducing AAA mortality, particularly over the past two decades and further supports continued work in management of this disease.**