Anaesthesia for elective open abdominal aortic aneurysm repair

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Key points
Smoking is the most important modifiable risk factor in the formation, expansion, and rupture of abdominal aortic aneurysm (AAA).

The incidence of AAA increases with age.

Patients with AAA are typically elderly with significant co-morbidities.

Mortality rate after elective open AAA repair is significantly lower than that after emergency repair.

Effective teamwork and communication is essential, especially during aortic clamping and unclamping.

The word ‘aneurysm’ comes from the Greek *aneurysma*, meaning dilatation or widening. An aortic aneurysm is a permanent dilatation (>30 mm) anywhere along the path of the aorta (ascending, arch, thoracic, or abdominal).

This article focuses on the elective open surgical repair of infra-renal abdominal aortic aneurysms (AAA).

The reported population incidence of AAA is 4.9–9.9%. The overall mortality of open repair of infra-renal AAA varies between centres and ranges between 1% and 6%. In comparison, the overall mortality after ruptured AAA is almost 90%; 75% of patients die before reaching the operative theatre and of those undergoing surgery, a further 40% die. The Glasgow aneurysm score (GAS), designed to predict outcome after open AAA repair, is based on the patient’s age and co-morbidities. Until recently, the GAS was considered the most useful and consistently validated scoring system for open AAA repair, but it is becoming outdated with the increasing practice of endovascular aneurysm repair (EVAR). A review of the available AAA risk prediction and scoring models showed that the Medicare and the North West Vascular Governance model correlated the closest with outcome after AAA and would be suitable for risk prediction after elective AAA surgery in the UK.

In 2008, the VASCUNET database reported that the UK mortality rate after elective open repair of AAA (7.5%) was among the highest in Europe and Australasia. This led to the development of the Abdominal Aortic Aneurysm Quality Improvement Programme (AAAQIP) aiming to halve mortality by 2014. The latest QIP report of surgery performed in the UK between 2009 and 2010 found a 2.4% overall mortality rate after elective repair of AAA (4.3% after open repair and 0.9% after EVAR).

Aetiology, pathophysiology, and natural history

The strongest predictor of AAA formation is positive family history. Women are less likely to develop AAA than men of similar age. However, if an AAA has developed, women have an increased risk of aneurysm rupture and a higher mortality rate, especially where there is coexisting cardiovascular morbidity. Smoking is the most important modifiable risk factor in the formation, progression, and rupture risk of AAA. Epidemiological data indicate that there is a decreased prevalence of AAA in Black and Asian ethnic groups compared with Caucasians. The number of collagen and elastic fibres is reduced within the aneurysmal segment of the aorta with poor quality fibre cross-links; vascular wall strength is further compromised by several factors:

(i) local elastin resorption caused by increased elastase activity;
(ii) localized wall inflammatory changes;
(iii) increased protease activity;
(iv) mural thrombus formation in the arterial wall and plasminogen activation.

An AAA will expand with time and eventually rupture; the strongest predictors of rupture are the maximum diameter and the annual expansion rate. The annual risk of rupture for large AAA > 5.5 cm in diameter is 18% in women [95% confidence interval (CI), 8–26%] and 12% in men (95% CI, 5–20%). Reduced expansion rates are seen in patients with diabetes mellitus. The risk of aneurysm rupture increases in a non-linear fashion when aneurysms expand; the risk of rupture becomes clinically significant when the aneurysm diameter reaches 5 cm, but there is considerable variation between published studies (Fig. 1). Abdominal ultrasound is the first-line imaging tool in the diagnosis and
Important medical management steps are as follows: able risk factors and the control of coexisting diseases. The most optimum medical management should be directed towards modification of these risk factors. The surveillance of AAA with a detection specificity and sensitivity of almost 100%.

Management strategies
Non-surgical management and surveillance
Optimum medical management should be directed towards modifiable risk factors and the control of coexisting diseases. The most important medical management steps are as follows:

(i) Smoking cessation can slow down aneurysmal growth by 15–20% and decrease perioperative morbidity relating to wound healing and cardiorespiratory complications.
(ii) Statins can minimize perioperative myocardial ischaemia and may alter aneurysmal growth.
(iii) According to recent recommendations, low-dose aspirin should be started when an AAA is diagnosed and continued indefinitely. The evidence for this recommendation is based on meta-analyses in the primary and secondary prevention of coronary events; although not conducted in patients with AAA, the effects of secondary prevention of major coronary events were clear in patients with significant vascular disease. β-Blockers and angiotensin-converting enzyme (ACE) inhibitors may be considered for patients with high cardiovascular risk.

The UK Small Aneurysm Trial and the Aneurysm Detection and Management Study (ADAM–US) concluded that AAA between 4.0 and 5.5 cm in diameter had less than a 1% annual rupture rate and that early surgery for those patients conferred no long-term survival benefit. These studies concluded that a regular surveillance strategy is safe in compliant patients with small aneurysms. The aims of surveillance are to monitor aneurysmal size, identify patients with a high risk of rupture, and monitor the rate of AAA expansion.

The National Health Service AAA Screening Programme initiated population screening in the UK for men over 65 yr old and accepts self-referrals from older patients. The evidence for this recommendation is based on meta-analyses in the primary and secondary prevention of coronary events; although not conducted in patients with AAA, the effects of secondary prevention of major coronary events were clear in patients with significant vascular disease. β-Blockers and angiotensin-converting enzyme (ACE) inhibitors may be considered for patients with high cardiovascular risk.

Fig 1 Estimated annual rupture risk according to aneurysm size. The vertical lines represent the range of mean values for the annual risks of AAA rupture from published series; the curved line indicates the polynomial trend of these mean values. Figure created using data from Brewster et al.

Surgical management
The aim of surgery is to replace the weak aneurysmal segment of the abdominal aorta with a synthetic graft, with minimum perioperative mortality and morbidity. Two main surgical techniques are available for AAA repair: open repair and endovascular repair (EVAR). The choice of technique depends on aneurysmal morphology, patient’s co-morbidities, and age. In many centres, EVAR is becoming the preferred technique for surgical management of AAA, but in a significant proportion of patients, the aneurysm morphology (site, shape, angulation, involvement of renal arteries, and size of iliac vessels) precludes EVAR. With advances in device technology, operator experience, and lower morbidity and mortality rates, EVAR is likely to increase, but for some patients, open repair remains the only feasible option. EVAR has been discussed previously in this journal (Contin Educ Anaesth Crit Care Pain 2004; 4: 91–4) and this article is confined to open surgical repair.

The surgical approach can be either transperitoneal (TP) (via a transverse or a longitudinal abdominal incision) or retroperitoneal (RP). The TP approach is the most widely practiced and familiar technique for open AAA repairs and provides rapid and effective surgical access. The RP approach usually involves a left flank incision that may be considered in patients with ‘hostile’ abdomen, multiple previous abdominal operations, stoma, horseshoe kidney, or inflammatory aneurysms. Laparoscopic-assisted AAA repair has been reported but is not widely practiced.

After surgical exposure of the aneurysm, the surgeon applies a cross-clamp to the abdominal aorta. It is sometimes challenging to find a safe and practical site to apply the clamp due to the close proximity of the aneurysmal segment to the renal/mesenteric arteries. The application of the clamp may dislodge atheromatous plaques within the aortic wall, leading to vascular embolization and organ ischaemia.

When the aorta is cross-clamped, the anterior aortic wall is incised and a graft is sutured to both ends of the aorta to replace the aneurysmal segment. Grafts can be straight ‘tube’ or bifurcated ‘trouser’ grafts. Iliac vascular flow (at least one) should be maintained wherever possible to prevent pelvic organ ischaemia. A femoral–femoral crossover or other graft is sometimes needed to re-vascularize ischaemic limbs at the end of surgery.

Preoperative assessment, management, and investigation
The broad aims of preoperative assessment are to stratify and minimize perioperative mortality and morbidity risks. This is
particularly important before AAA surgery because it is a high-risk procedure performed in a high-risk population and patients must be optimized wherever possible. All patients should commence statins and antiplatelet medications. Arterial pressure should be well controlled and lifestyle advice offered, particularly smoking cessation.9

According to recent recommendations,9 patients should continue taking β-blockers (if already taking these), aspirin, and statins before surgery. Diuretics and ACE inhibitors should be considered on a case-by-case basis. Decisions regarding continuation of clopidogrel and newer antiplatelet agents (prasugrel, ticagrelor) through the perioperative period are more complex and depend on the indication for these agents; acute cessation in the presence of drug-eluting coronary stents predisposes to catastrophic stent thrombosis and discussion with the prescribing specialist (cardiologist) is required. Although there is an increased risk of perioperative bleeding, recent data suggest that continuation of clopidogrel may not increase transfusion requirements or the incidence of reoperation for bleeding after AAA repair.

Operative mortality risk is often quoted to patients as low (1–3%), moderate (3–7%), or high (5–10%).9 The GAS includes cardiovascular disease [myocardial infarction (MI), angina, cerebrovascular disease, and renal disease as predictors of mortality and morbidity with variable significance. Coronary artery disease is the leading cause of early and late morbidity after AAA repair; moreover, chronic obstructive pulmonary disease (COPD), diabetes mellitus, and renal insufficiency can all increase postoperative morbidity.9

COPD, anaemia, and low haematocrit should be addressed and optimized before operation as they are associated with increased mortality and morbidity when poorly treated.9

When EVAR is not possible and the risks of open repair are high, surgery can be deferred until the estimated risk of acute rupture renders surgery urgent, and when the perceived benefits outweigh the risks. Alternatively, surgical treatment may not be offered if operative risks are too high, after due discussion with the patient.

Vascular anaesthetists should assess all patients and review their investigations before operation. Preoperative investigations include all standard investigations as per local policies and national guidelines (full blood count, electrolytes, electrocardiogram, chest X-ray, urinalysis, and others as indicated). In the authors’ unit, echocardiogram and lung spirometry are performed routinely. Other cardiac investigations are requested on a case-by-case basis (cardiac catheterization, dobutamine stress testing, etc.). Functional capacity can be assessed subjectively based on the patient’s ability to perform activities of daily living that require sustained aerobic metabolism, or objectively by cardiopulmonary exercise testing where facilities are available.

As for other major surgical procedures, the presence of ‘major’ cardiac risk factors [decompensated heart failure, acute coronary syndrome, significant arrhythmias, severe valvular disease, worsening ischaemic heart disease (IHD), or recent MI] mandates that AAA surgery is deferred for investigation and management. The American College of Cardiology/American Heart Association guidelines on the management of the surgical patient with cardiovascular disease provide a useful template for how to proceed, depending on medical conditions, physical fitness, and type of surgery.10 The main indications for referral for a cardiology opinion are to establish whether a patient’s cardiac condition is optimized or whether coronary revascularization is indicated before AAA repair. Patients with COPD may benefit from regular nebulizers and chest physiotherapy before surgery to decrease the incidence of respiratory complications.

Aortic cross-clamping and physiological considerations

The physiological effect of aortic cross-clamping during surgery varies with the level of the clamp in relation to the main aortic branches. Perfusion to the lower half of the body is therefore dependent on collateral circulation while the clamp is applied.

Clamp application increases the afterload of the heart and a sudden increase in arterial pressure proximal to the clamp; this can be attenuated with vasodilators [e.g. glyceryl trinitrate (GTN), sodium nitroprusside], opioids, or deepening of anaesthesia. These measures may also allow fluid loading in preparation for clamp release; however, the effect of vasoactive drugs is unpredictable; they may change haemodynamics without improving cardiac output and tissue perfusion due to blood redistribution.10

Increased afterload and left ventricular end-diastolic volume both increase myocardial contractility and oxygen demand. This increase in myocardial oxygen demand is usually met by an increase in coronary blood flow and oxygen supply, but can cause myocardial ischaemia.

After aortic cross-clamp release, peripheral vascular resistance decreases by 70–80%, causing a decrease in arterial pressure. Hypotension can also be caused by blood sequestration in the lower half of the body, ischaemia–reperfusion injury, and the washout of anaerobic metabolites causing metabolic (lactic) acidosis. This can cause direct myocardial suppression and profound peripheral vasoconstriction. Coronary blood flow and left ventricular end-diastolic volume also decrease (almost 50% from pre-clamp levels) after clamp release.

Strategies to manage hypotension after aortic cross-clamp release include gradual release of the clamp, volume loading, vasoconstrictors, or positive inotropic drugs (e.g. ephedrine, meteraminol, phenylephrine, epinephrine, and norepinephrine). It is important to be aware that vasoactive drugs should only be used after adequate volume repletion.10 Management of aortic cross-clamp application and release requires excellent communication with the surgeon in order to anticipate and manage the physiological effects.

Intraoperative management

The core aim of anaesthesia for elective open AAA repair is that the patient is managed to the end of surgery so as to be haemodynamically stable, comfortable, normothermic, not bleeding, and with no immediate need for multi-organ support after operation. Acid–base status and gas exchange values should be kept within
Acceptable limits, aiming for extubation immediately or in the early postoperative period.

Conduct of anaesthesia

Monitoring

Minimum standard monitoring should be placed before induction of anaesthesia. A five-lead ECG is more sensitive in detecting myocardial ischaemia. Invasive arterial pressure monitoring should be established before but central venous access is usually secured after induction of anaesthesia. Urinary catheterization and temperature monitoring should be initiated. Different cardiac output monitoring strategies have their limitations and may respond slowly to haemodynamic changes with aortic cross-clamp application and release. Oesophageal Doppler uses flow velocity in the aorta to calculate cardiac output and is unreliable when the aorta is clamped. Pulse wave contour analysis cardiac output and other monitors are gaining popularity, but their use has not yet been fully evaluated in aortic surgery.

Anaesthesia and analgesia

Anaesthetic management focuses on the acute haemodynamic changes with aortic cross-clamping and unclamping, maintaining organ perfusion and oxygenation, attenuating ischaemic reperfusion injury, and providing intra- and postoperative analgesia. Anaesthesia is usually maintained by a balanced volatile/opioid technique (fentanyl, remifentanil, morphine).

A thoracic epidural catheter is usually placed before induction of anaesthesia at a level corresponding to the upper dermatomal level of the incision (usually T8–T10) for postoperative analgesia. Stability during induction of anaesthesia must be maintained, avoiding massive swings in arterial pressure and heart rate. Tracheal intubation and artificial ventilation is routine practice, a ventilator capable of administering PEEP is preferable. Antibiotic prophylaxis should be administered within 30 min of skin incision.

Intraoperative analgesia can be provided using opioids or by using epidural analgesia; however, high doses of epidural local anaesthetics can cause profound hypotension after aortic cross-clamp release due to sympathetic blockade. It is common practice to limit epidural local anaesthetic administration until after cross-clamp release and haemostasis has been achieved.

Heparin 75–150 units kg$^{-1}$ is given i.v. before aortic cross-clamp application. Activated clotting time can be used to guide heparin therapy (2–3 times more than baseline). Cell salvage equipment should be used when available. Serial arterial blood gas samples are usually analysed to monitor respiratory and metabolic status. Facilities for the rapid infusion of warm fluids and blood should be available for immediate use. All efforts should be made to maintain normothermia; however, lower body warming during aortic cross-clamp application is discouraged.

The hypertensive response seen with aortic cross-clamp application can be managed by an infusion of short-acting vasodilators (e.g. GTN), increasing the administered dose of inhalation anaesthetic agents or by administering i.v. opioids. The mean arterial pressure should be maintained within the autoregulation limits of vital organs. In preparation for aortic unclamping, vasodilators can be weaned down with adequate fluid loading. Vasoconstrictors and positive inotropes should be available for immediate use.

Blood, blood products, and fluids

Haemoglobin should be maintained $>9–10$ g d$^{-1}$ as IHD is common in these patients. Blood products (FFP, platelets, and cryoprecipitate) are usually given according to the clinical need when haemostasis is secured and aortic cross-clamp is removed. Thromboelastography testing can be used to monitor and help to manage coagulopathy. Fluid loading while the aortic cross-clamp is applied is usually achieved using crystalloids and colloids. While the limitations of central venous pressure (CVP) as a measure of intravascular volume are increasingly recognized, it is common practice to titrate fluids to maintain a CVP of $\geq 12–15$ cm H$_2$O before cross-clamp release. Other monitors or measures of cardiac output or fluid responsiveness may also be used, although few specific data are available in aortic surgery.

Organ protection

The physiological basis of organ protection is to maintain vital organ perfusion, oxygen delivery, and euvolaemia.

Myocardial damage may be minimized by maintaining myocardial oxygen supply and minimizing demand. The basic principles of myocardial and other organ protection are to maintain good oxygen saturation, haemoglobin concentrations, and blood flow. Excessive tachycardia and hypotension should be avoided when possible. Vasodilators can help to lower the left ventricular end-diastolic volume, decreasing ventricular wall tension, decreasing the force of contraction, and improving endocardial perfusion. These effects will tend to improve the balance of myocardial oxygen supply and demand.

The main cause of renal complications after AAA repair is the decrease in renal blood flow, decreased renal perfusion pressure (outside autoregulation) augmented by the increasing renal vascular resistance (by 30%) associated with aortic clamping. Myoglobin release from ischaemic tissues may contribute to acute tubular necrosis by decreasing local nitric oxide release. Acute kidney injury (AKI) may also be linked to ischaemia–reperfusion injury, decreased renal cortical blood flow, prostaglandin imbalance, and increased activity of renin–angiotensin system. Postoperative dialysis rates are similar in patients who have undergone either suprarenal or infra-renal aortic cross-clamping. Intraoperative urine output does not correlate with the degree of...

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decrease in glomerular filtration rate (GFR) or the incidence of postoperative AKI.

Several drugs (dopamine, N-acetyl cysteine, mannitol, furosemide) have been used in an attempt to protect against AKI, although none has been shown consistently to be beneficial, and all diuretics should be used only after adequate fluid replacement and volume loading. Loop diuretics potentially decrease renal tubular reabsorption and oxygen demand. Mannitol can increase renal blood flow during aortic cross-clamp; however, both mannitol and dopamine use fail to return GFR to baseline levels after operation.\(^{10}\)

### Postoperative management

After surgery, the patient is nursed in a high dependency or intensive care area. Epidural analgesia is widely used after operation (using a mixture of local anaesthetics and opioids). Early postoperative mobilization and physiotherapy should occur as soon as practically possible and tolerated. Prophylaxis against deep vein thrombosis (DVT) (hydration, compression stockings, and heparin therapy) should be started perioperatively and continued until the patient is fully mobile and no longer considered at risk of DVT.\(^{9}\)

There are many potential complications after open AAA repair (Table 1) and close monitoring for these is required.

### Conclusions

Open repair of AAA is a major high-risk surgical procedure undertaken in patients with significant co-morbidities and poor physiological reserve. In order to achieve good outcomes, risk factors should be optimized, the surgical intervention planned, pathophysiology understood, and organ protection strategies used. Effective communication and team work are essential.

### Declaration of interest

None declared.

### References


Please see multiple choice questions 17–20.