Anaesthesia for open abdominal aortic surgery

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Abstract

The prevalence of abdominal aortic aneurysm (AAA) and the number of patients undergoing aneurysm repair is increasing. The UK has worked tirelessly to reduce its operative mortality rates for elective open AAA repair with the introduction of a quality improvement programme. Reducing death from ruptured aortic aneurysm has been the focus of the national screening programme. Despite the increased prevalence of disease and intervention, the popularity of open repair has diminished since the advent of endovascular repair (EVAR). The short-term benefits of EVAR when compared to open repair are well described; however, the long-term survival benefits, freedom form re-intervention and cost effectiveness of EVAR are not proven. The choice of technique for emergency AAA repair is contentious, with the more traditional approach of open repair being rapidly overtaken by endovascular options. In this article we provide an overview of the evidence supporting the different treatment options, outline current approaches to risk stratification, describe the key physiological changes that occur during open repair and describe an overview of the approach to perioperative management.

Keywords AAA Quality Improvement Programme (AAAQIP); abdominal aortic aneurysm (AAA); cross-clamping; endovascular aneurysm repair (EVAR); National AAA Screening Programme (NAAASP)

Royal College of Anaesthetists CPD Matrix: 3A05, 2A03, 2A04, 2A05, IL05

Learning objectives

After reading this article, you should be able to:

- describe the incidence and risk factors associated with abdominal aortic aneurysm
- discuss the rationale for the national AAA screening programme
- define the indications for intervention and identify the factors that govern whether open or endovascular repair is the technique of choice
- outline the risk-stratification scoring systems applicable to aneurysm surgery and their merits
- list the key perioperative considerations for open abdominal aortic surgery

Epidemiology, risk factors and natural history

The prevalence of abdominal aortic aneurysm (AAA) has increased steadily over the past 50 years. It now affects between 4% and 8% of men aged 65–80. The comparatively low prevalence found in women (1.3%), led to their exclusion from screening trials as cost effectiveness could not be demonstrated.[1] This was despite their increased risk of aneurysm rupture and associated mortality rates.

Aside from advancing age and gender, important risk factors for the development of an AAA include a positive family history and chronic tobacco use. Smokers are four times more likely to develop an AAA when compared to non-smokers. Smoking is also associated with an increased rate of aneurysm growth.

Aortic aneurysms are usually asymptomatic and increase in size at varying rates over time. The annual risk of aneurysm rupture increases exponentially when the anteroposterior diameter exceeds 5.5 cm. On this basis, elective intervention is considered in all those meeting this criterion. Ruptured AAA accounts for the death of around 7000 men in England and Wales every year and is associated with a mortality rate in excess of 75%.

Anatomical classification of AAA

Diagnosis of an AAA requires evidence of a dilatation ≥50% of the normal aortic diameter. An infrarenal aortic diameter of ≥3.0 cm is considered aneurysmal. AAAs are commonly described based on their relationship to the renal arteries. Most AAAs occur in the segment between the renal and inferior mesenteric arteries. Only around 5% of AAAs involve the renal or visceral arteries. Up to 40% of AAAs are associated with iliac artery aneurysms.

The most common cause of AAA is atherosclerosis. The tensile strength of the vascular wall is weakened as increased elastase and protease activity results in decreased elastin and collagen fibres. Associated inflammatory changes and thrombus formation also contribute to aneurysm formation.[2]

National screening programme

A National AAA Screening Programme (NAAASP) was set up in 2009 on the background of evidence presented by the Multicentre Aneurysm Screening Study (MASS) group. This suggested that screening could halve AAA-related deaths in men aged 65–74 and presented evidence supporting surgical intervention in AAAs ≥5.5 cm.[3]

Screening is currently offered to all men aged 65 in the UK. These men are screened by ultrasound and managed according to a nationally agreed clinical pathway. It is recommended that patients identified via the NAAASP should undergo intervention within 8 weeks of diagnosis, with those diagnosed incidentally, following a similar timeframe. The screening programme's success hinges on the premise that the risk associated with

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intervention is less than the risk of harm associated with natural disease progression and the risk of aneurysm rupture.

AAA quality improvement framework and centralization of services

The UK was reported to be a significant outlier compared to our European counterparts when crude mortality rates following elective open AAA repair was reported in 2008. In response to this data, a national quality improvement framework was conceived and a nationwide programme promoting best practice was delivered. This coincided with the national screening programme. In addition to encouraging standards of best practice, the framework aimed to reduce variance in clinical practice and reduce operative mortality rates.[4]

The AAA Quality Improvement Programme (AAAQIP) emphasizes the importance of a multidisciplinary team (MDT) approach to preoperative assessment. A discussion between surgical, radiology and vascular anaesthetic team members should take place after formal CT angiography, regarding patient suitability for elective AAA intervention. Patients considered for intervention should be involved in this process and given consistent advice about the potential risks involved to ensure they can make an informed decision about their management.

The centralization of services aims to utilize the volume-outcome relationship, which demonstrates favourable outcomes for patients undergoing intervention in centres undertaking higher caseloads. High-volume centres demonstrated reduced length of stay and improved survival after complications. Initial recommendations suggested that vascular centres should be performing a minimum of 100 elective interventions over a 3-year period. They should be equipped with the necessary staff and facilities to be able to provide both open and endovascular repair. Both the Royal College of Anaesthetists (RCoA) and the Vascular Society have published guidelines supporting these recommendations.

Clinical and process data on aneurysm repairs should be entered into the national clinical audit via the National Vascular Registry (formerly the National Vascular Database). This should be done in real time or within 2 weeks of discharge or death.

The screening programme and quality improvement recommendations appear to be having the desired effect as the overall mortality rate for AAA repair (open and EVAR) in the UK has fallen from 2.4% in 2010 to 1.3% between January 2015 and December 2017. For open repair the NVR reported 30-day mortality at 3.2%, with an average in hospital length of stay of 8 days.[5]

Indications for intervention

In the elective setting, the decision regarding when to operate is guided by the size and rate of growth of the aneurysm. Current evidence supports intervention when AAAs $\geq$5.5 cm. Aneurysms $\leq$5.5 cm have a relatively low annual rupture rate ($\leq$1%). Soon to be published data from the NHS AAA Screening Programme may question whether rupture rates at 5.5 cm are indeed high enough to support that threshold for surgery. Studies from both sides of the Atlantic showed no long-term survival benefit between surveillance and surgery for patients with an AAA between 4.0 and 5.5 cm in diameter.[6]

Despite the less invasive nature of endovascular repair, neither the CAESAR nor the PIVOTAL trials could demonstrate a benefit to quality of life or survival when comparing surveillance to EVAR for patients with an aneurysm $\geq$4.0 cm.

The indication for intervention in patients presenting with symptomatic or ruptured AAA is clinical rather than radiological. The opportunity for thorough preoperative assessment, multidisciplinary discussion and optimization is limited under these circumstances. A rapid evaluation of the appropriateness of surgery is required.

Open versus endovascular repair

Since its introduction in the late 1980s, EVAR has seen its popularity grow for both elective and emergency AAA repair. The proportion of elective repairs performed endovascularly has increased from 54% in 2009 to 68% in 2017. The characteristics of patients undergoing endovascular repair is also changing. They tend to be older and have a greater burden of comorbid disease. The continuing trend towards endovascular repair is based on the evidence of studies comparing EVAR to open aneurysm repair. Decisions made are both on anatomical grounds but on the better (short term) survival rates with EVAR.

EVAR I, the Dutch DREAM trial and the American OVER trial concluded that although EVAR offered lower operative and 30-day mortality rates there was no survival advantage at the 5-year endpoint. Furthermore, EVAR was associated with increased risks of complications, reinterventions and costs.[6]

Interestingly, a follow-up study of EVAR-1 demonstrated increased all cause and aneurysm-related mortality figures in the EVAR group from 8 years following follow up. This was in part attributed to secondary aneurysm sac rupture.

The most recent trial comparing elective open repair to EVAR (the ACE trial) demonstrated similar 30-day mortality rates between the two interventions. The authors concluded that in patients with low-to-intermediate risk, there is no difference in perioperative or midterm survival or in complication rates.[7]

In 2014, the IMPROVE trial failed to prove any significant difference in 30-day mortality between EVAR and open surgery in patients with ruptured AAA. However, those that survived who underwent EVAR had a significantly reduced length of stay and were more likely to be discharged directly home and more likely to lead an independent life.[8]

Data from the National Vascular Registry reports an increased length of hospital stay (8 days) for patients undergoing elective open AAA repair compared to those undergoing EVAR (2 days). The overall mortality rate for open aneurysm repair was 3.2% compared to 0.7% with EVAR.[5]

Risk stratification

Individualized risk stratification prior to elective intervention starts with a MDT discussion for each patient with an aneurysm $\geq$5.5 cm. Risk models have been used but are somewhat limited in their prediction for an individual. A review of the available risk prediction scoring systems for elective AAA repair showed that the British Aneurysm Repair (BAR) score, Medicare and Vascular Governance North West models provided the most accurate correlation between intervention and outcome. These scoring systems calculate patients' mortality risk based on a number of factors including the type of repair, age, sex and coexisting disease.[9]

Scoring systems all have their limitations and should only be used to supplement the clinical judgement of experienced vascular surgeons and anaesthetists involved in discussions with the patient and their family. The current risk models are subjective, with variations in the definitions of diagnoses. They rarely reflect the severity or the duration that an individual has been exposed to a disease (e.g. diabetes mellitus). They tend to have low positive predictive value and were modelled at a time when infra-renal open surgery was the most commonly performed intervention. This is important because the patient cohort in today's 'endovascular era' who are not suitable for EVAR tend to have by definition more complex anatomy. If not suitable for complex EVAR (e.g. fenestrated or branched stent grafting) then an open repair will be more technically challenging. This is coupled with the fact that more challenging anatomy is often associated with greater cardiovascular disease burden (i.e. a high-risk patient).

Cardiopulmonary exercise testing (CPET) is increasingly being used in the preoperative assessment of patients undergoing major surgery. Low or sub-threshold values of certain CPET variables are associated with reduced life expectancy after elective AAA repair (open or EVAR). A cohort of patients with reduced survival at 3 years post-procedure can be identified using these variables. This information may help guide discussions with the patient regarding the options for intervention. For patients predicted to have a relatively short life expectancy (e.g. less than 5 years) open surgery will lead to a significant proportion of what remains of their life being spent in hospital. Coupling this with their increased perioperative risk (which may also be in part determined using CPET) may influence the final decision on choice of management.

The use of serum markers to assist in risk stratification is becoming more prevalent. For example, studies have demonstrated that raised serum N-terminal pro-brain natriuretic peptide (NT-proBNP) is a valuable predictor of perioperative cardiovascular complications after non-cardiac surgery. This is particularly true where the raised serum level is secondary to cardiac failure. There is supporting evidence now that preoperative NT-proBNP measurement out performs clinical risk indices and may supersede clinical risk factor scoring systems in the future particularly in vascular surgery, where the evidence base is strongest.

It is important to recognize that risk prediction models do not predict the outcome for an individual patient (and hence not used to do so), but they will provide an estimate of the risk for a population of patients with similar characteristics undergoing the same procedure.

In patients who present with symptomatic ruptured or unruptured AAA the goal is to ensure that emergency open repair is appropriate and in the best interest of the patient. Where surgery is futile it is necessary to provide urgent end of life care. This decision can be made after a targeted assessment of the patient's medical history, current condition, and suitability for an endovascular procedure has been made. It is important to remember that some patients may be known to the vascular team and may have declined to undertake elective intervention. Nonetheless, once rupture or impending rupture becomes evident it may now be reasonable (shift in risk–benefit balance) to attempt a high-risk life-saving operation. This re-emphasizes the importance of involving senior clinicians in the decision making process.

Preoperative evaluation and optimization

Stratifying perioperative risk and identifying areas of disease management requiring optimization is a cornerstone of preoperative assessment. Counselling the patient is another key element of this process as their understanding of the diagnosis and treatment pathway can vary enormously. In certain circumstances, the decision not to operate could give the patient the longest and best quality of life.

Ideally, a vascular anaesthetist should assess all patients listed for aneurysm repair. Specific features associated with outcome following AAA repair should be explored. This includes the presence of coronary artery disease, chronic obstructive pulmonary disease (COPD), diabetes mellitus, peripheral vascular disease and renal insufficiency. The assessment of a patient's functional capacity can be measured by objective cardiopulmonary exercise testing or incremental 6-minute shuttle walk test.

Undiagnosed COPD should be addressed and treatment optimized with the assistance of a respiratory physician or the patient's general practitioner. Those on short acting inhaled bronchodilators may benefit from being switched onto longer acting agents for the perioperative period. Those with steroid responsive disease may benefit from a short course of oral steroid therapy prior to surgery and to operate outside of the winter months if time permits. Smoking cessation advice, incentive spirometry and perioperative physiotherapy are particularly important adjuncts, which should be explained and taught in advance of the date for surgery.

Diet and exercise advice can reaffirm the importance of preoperative aerobic conditioning to the patient. A number of enhanced recovery programmes are underway in the UK to assist and educate patients in the importance of improving fitness prior to surgery.

Patients with AAA have an indication for antiplatelets and statin therapy. If no contraindication exists they should commence therapy as soon as possible. Statins are protective both in the long term (reducing cardiovascular risk) but also in the perioperative period. There is also evidence that they slow the progression of aneurysm growth. Cessation of statin therapy during the perioperative period is not advised and this should be communicated to the patient. Aspirin is continued but thienopyridine derivatives such as clopidogrel should generally be stopped in advance of open surgery due to the association with excessive bleeding and contraindication to neuraxial blockade. Patients who have recently undergone percutaneous coronary intervention (PCI) and coronary stenting should have their antiplatelet therapy management discussed with an interventional cardiologist at the earliest opportunity. They can advise on the risk–benefit balance of deferring surgery, which will help when planning the optimal timing of surgery.

There has been much discussion surrounding the risk–benefit analysis of preoperative β-blockers in patients undergoing non-cardiac vascular surgery. Original recommendations suggested that β-blockers be commenced in all those undergoing vascular surgeries that are at a higher perioperative cardiac risk. It could be argued that all patients undergoing aneurysm repair should be considered to be part of this high-risk group due to their associated increased incidence of coronary artery disease. Despite this assumption, the current recommendations state that

initiation of β-blockers should not be considered routine in those undergoing vascular surgery and should be only started if indicated on a case-by-case basis.

Evidence of mild to moderate cardiac ischaemia on non-invasive stress testing (e.g. dobutamine stress echocardiography) may necessitate initiation of therapy with anti-ischaemia medication. This is based upon the fact that the therapy is indicated regardless of the need for aortic surgery. Higher grade ischaemia (e.g. occurring at relatively low heart rate) will necessitate further investigation in the form of coronary angiography because the patient may benefit from cardiac revascularization. This category of patient is uncommon, though more common in vascular patients than any other non-cardiac surgical cohort. This finding has significant implications for a patient undergoing open aortic surgery and requires a multi-professional approach. β-Blockers should be continued in those already taking them and up titrated in patients with evidence of low grade cardiac ischaemia on stress testing.

Anaesthetic technique

The anaesthetic technique for open AAA repair is built on the foundations of maintaining haemodynamic stability, normothermia, haemostasis and adequate analgesia. A variety of modifications to the anaesthetic technique can be applied, which will achieve the same outcome for the patient. These variations may be due to personal preference of the anaesthetist, the patient's comorbid state or the specific surgical approach.

Monitoring

A five-lead ECG is recommended in addition to standard monitoring. The group of patients undergoing aneurysm repair have a high incidence of coronary artery disease and this lead configuration is associated with improved detection of myocardial ischaemia.

Significant haemodynamic changes can occur on induction and at any time throughout the perioperative period. Invasive arterial blood pressure monitoring is recommended before induction to allow for prompt recognition and management of these fluctuations. An arterial line also provides access for rapid acid-base analysis, haemoglobin measurement and point of care testing of the patient's coagulation status.

A central venous catheter can be inserted after induction in preparation for the use of any vasopressors or inotropes that may be required. Urine output and temperature monitoring are important in this group of patients, particularly when considering the pathophysiological effects of cross clamping.

Cardiac output monitoring in combination with goal-directed fluid therapy was shown to improve postoperative outcome in high-risk general surgical patients. To date, the evidence supporting the use of cardiac output monitoring in open aneurysm repair is lacking. The reliability of the oesophageal Doppler and arterial pressure waveform analysis is invalidated when the aorta is clamped. Despite the lack of evidence and difficulty interpreting results during cross-clamping, many anaesthetists will employ one form of cardiac output monitoring perioperatively. Perioperative transoesophageal echocardiography (TOE) allows for real-time, dynamic assessment of cardiac function but is heavily dependent on operator ability and interpretation.

Bispectral index monitoring (BIS) has garnered some support in recent times. It has been suggested that monitoring depth of anaesthesia using electroencephalographic data allows more accurate titration of anaesthetic agents without increasing the risk the awareness. Avoiding excessive doses of anaesthetic agents would be beneficial in this group of patients prone to significant haemodynamic changes, but this has yet to be proven. Evidence is emerging that suggests BIS-guided anaesthesia may be associated with a reduced incidence of postoperative cognitive dysfunction (POCD). Patients undergoing aneurysm repair tend to be older and have associated vascular disease. These factors in combination with the nature and duration of surgery, places them at high risk of developing POCD. Any technique that could reduce this risk would be desirable.

Anaesthesia

Open AAA repair is carried out under general anaesthesia. The potential cardioprotective effects of volatile anaesthetics found in cardiac surgery were not replicated when volatiles were compared with total intravenous anaesthesia (TIVA) for patients undergoing elective AAA repair. Despite this, a volatile anaesthetic agent combined with a systemic opioid remains the technique employed by the majority of anaesthetists.

Postoperative analgesia is usually managed with a thoracic epidural that is sited before induction. Some anaesthetists prefer not to commence the epidural infusion until the end of surgery, after haemostasis and haemodynamic stability has been achieved. The sympathetic blockade associated with epidural analgesia will necessitate vasopressor therapy if commenced early after aortic clamp release. Intraoperatively, systemic opioids can be used as a bridging regimen until the epidural analgesia is commenced.

When compared to systemic opioids, thoracic epidural analgesia has been shown to reduce the duration of postoperative tracheal intubation, provide superior analgesia and reduce the incidences of postoperative myocardial infarction and gastric or renal complications. In isolation, aspirin does not confer any additional increased risk of spinal canal haematoma but careful consideration must be given to those patients on dual or more novel antiplatelet therapies.

Tracheal intubation is routine and artificial ventilation using lung-protective strategies is favoured. Low tidal volume ventilation with high PEEP is associated with a reduction in postoperative pulmonary complications. The recent PROVAR trial failed to demonstrate an improvement in intraoperative or postoperative respiratory function when using different lung protective ventilatory strategies in patients undergoing open abdominal surgery.[10]

Active warming measures should be employed to avoid the associated risks of hypothermia (oxygen consumption, myocardial dysrhythmias, coagulopathies, and postoperative wound infections). Lower body warming during aortic cross-clamping should be avoided. Broad-spectrum antibiotic prophylaxis is routine for AAA surgery and is usually administered prior to incision.

The debate surrounding the optimal fluid therapy extends throughout anaesthetic subspecialties. A Cochrane review published in 2010 failed to identify the best fluid replacement to use

during and following abdominal aortic surgery. It suggested a combination of crystalloids and colloids with supplemental blood products were not only the most common choice, but also the most rational.

The anaesthetic management of emergency open AAA surgery is time sensitive and the opportunity for preoperative optimization is lost. Patients with symptomatic unruptured AAA may be permitted to delay surgery for insertion of a thoracic epidural catheter, but in any more time sensitive scenarios this is not advocated. Massive haemorrhage protocols should be initiated whilst blood is sent for full crossmatch (minimum 8–10 units). Permissive hypotension and the avoidance of aggressive fluid resuscitation prior to cross-clamping minimizes surges in BP that could cause dislodgement of an intact thrombus and avoids further dilution of already depleted clotting factors. A systolic pressure of 70 mmHg is advocated provided the patient shows no signs of altered conscious state or cardiac ischaemia. Recent subset analysis and results from the IMPROVE Trial seemed to suggest that this value may be too low for the elderly comorbid population but more research is needed.

Induction of anaesthesia usually takes place in theatre with rapid sequence induction, ideally with invasive blood pressure monitoring, and the patient prepped and draped. The aim is to minimize the time between induction and application of the aortic cross-clamp. Heparinization is not usually required. As with elective surgery certain facilities, devices and point of care testing equipment is necessary to successfully manage emergency open AAA repair (Box 1).

Blood and blood products

Blood loss during open aneurysm repair is highly variable. Contributing factors include those relating to the aneurysm and those relating to the surgery. Larger aneurysms, those with more complex anatomy and those associated with a hostile abdomen are associated with an increased risk of bleeding. Haemorrhage may result from retrograde bleeding from lumbar vessels, malpositioned aortic clamps, leaking anastomoses and dilutional or consumptive coagulopathies. Patients on clopidogrel will invariably require greater volumes of blood products and anticoagulation with warfarin will need to be addressed using local protocols.

Preoperative anaemia should have been identified, investigated, and treated prior to elective surgery. Cross-matched blood, rapid infusion devices, point-of-care coagulation testing, and intraoperative cell salvage should be available before starting surgery. Adequate intravenous access is essential to allow for rapid administration of fluids and blood products.

A transfusion threshold should be set on an individualized basis. This group of patients often has coexisting coronary artery disease and may benefit from a relatively higher haemoglobin concentration to maximize oxygen-carrying capacity.

Transfusion of blood products is dependent on the clinical picture, results of point of care coagulation tests, formal laboratory coagulation studies and occasionally advice from a haematologist. Thromboelastography (TEG) and rotational thromboelastometry (ROTEM) are the most recent advances in targeted transfusion of blood products. Their use is almost universal in vascular centres. Ideally, blood products should be transfused when haemostasis has been achieved and after the aortic cross-clamp has been released. Ideally the anaesthetist should aim for a platelet count greater than 100×10^9/L, fibrinogen concentration greater than 2 g/dL and INR less than 1.5.

Cross-clamping

In the elective case, prior to aortic cross-clamping, a dose of unfractionated heparin is given intravenously to reduce the risk of thrombosis. At the author's NHS Trust, an empirical dose of 100 IU/kg is administered based on the patient's ideal body weight. A recent systematic review has questioned the evidence supporting the beneficial effect of heparin in open AAA surgery and recommended the need for further research.

When the cross-clamp is applied, perfusion to the lower half of the body is entirely reliant upon collateral circulation and there is a sudden increase in arterial pressure proximal to the clamp. This increases the afterload and left ventricular wall tension of the heart. Resulting in an increase in myocardial workload and oxygen demand. In normal circumstances, this would be met by an increase in coronary blood flow and oxygen supply, but in patients with coronary artery disease it can cause myocardial ischaemia and impaired cardiac output. Management of this iatrogenic physiological change relies on vasodilatation. This can be achieved with vasodilators such as glyceryl trinitrate, opioids, or by increasing the depth of anaesthesia (Box 2).

Unclamping

A second physiological insult occurs when the aortic cross-clamp is removed. Peripheral vascular resistance decreases by three quarters resulting in potentially profound hypotension. This

Essential equipment in theatre for ruptured AAA

- White board to record blood and blood products administered
- Rapid infusion device
- Cell salvage machine and personnel
- Point of care testing equipment (e.g. rotational thromboelastometry, haemacue, ACT, ABG, glucometer)

Box 1

Factors affecting haemodynamic changes with aortic cross-clamp

- Level of clamp
- Blood volume redistribution
- LV function
- Presence of CAD
- Extent of collateral circulation
- Type of aortic disease (AAA vs. aorto-occlusive)
- Intravascular volume status at the time
- Anaesthesia
- Duration of cross clamp

Box 2

decrease in arterial pressure may be compounded by blood sequestration in the lower half of the body, ischaemia-reperfusion injury, and the release of anaerobic metabolites. The severity of hypotension is proportional to the cross-clamp time.

Prior to unclamping, the authors suggest a haemostatic pause. During this time, the aims should be to achieve adequate volume resuscitation and cardiovascular stability, correct acid–base and electrolyte disturbances and normalize the temperature. Increased minute ventilation during the period of cross-clamping may help to minimizes the effects of the ensuing metabolic acidosis. Epidural loading and infusion may also be considered at this time.

These measures in conjunction with a graduated release of the cross-clamp can reduce the degree of hypotension observed. Vasoconstrictors and positive inotropes may be required even after fluid replacement. In refractory hypotension, the cross-clamp may have to be reapplied. The gradual unclamping of the common iliac arteries one at a time, over a few minutes may also be helpful. Successful navigation through these physiological insults relies on close communication between the surgeon and anaesthetist.

Postoperative management

Open aortic surgery necessitates a period of postoperative observation in a critical care environment. In most cases, the aim is for primary extubation at the end of surgery and transfer to the high dependency unit for level II care. Indications for a period of postoperative ventilation include profound metabolic acidosis, refractory hypothermia, respiratory failure and high vasopressor or inotropic support. The requirement for organ support often depends on the complexity of the intervention, intraoperative complications, and the patient's pre-morbid state.

Postoperative complications

Open aneurysm surgery carries with it significant morbidity. Postoperative organ dysfunction is particularly common in the cardiovascular, respiratory, and renal systems.

Increased perioperative demands on the myocardium may manifest itself as delayed myocardial ischaemia and infarction. This commonly occurs within the first 72 hours following surgery. Respiratory failure may occur secondary to atelectasis, pneumonia or pulmonary thromboembolism.

The overall incidence of acute kidney injury (AKI) following open aortic surgery is approximately 5–10%. A predictably higher incidence is found in those patients in whom a supra-renal aortic clamp was required. Reduced renal blood flow and perfusion pressure contribute directly to the development of acute tubular necrosis. There is no evidence to support the use of renoprotective drugs such as dopamine, N-acetyl cysteine, furosemide, or mannitol.

Although rare, neurological complications following open abdominal aortic surgery can occur. Disruption of the arterial supply to the spinal cord during cross-clamping can result in ischaemia and paraplegia. Preoperative placement of a cerebrospinal fluid (CSF) drain may be deemed necessary in patients deemed to be high risk or spinal cord ischaemia. In general, the risk is extremely low and maintenance of adequate perfusion pressure with postoperative monitoring for evolving neurology is sufficient.

Other less common postoperative complications include bowel ischaemia, abdominal compartment syndrome, cerebrovascular disease, and associated surgical complications. ◆

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