Anaesthesia for the ruptured aortic aneurysm

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Abstract

The perioperative management of ruptured abdominal aortic aneurysms (RAAA) remains a core anaesthetic competency. Changes such as service centralization, aneurysm screening and the developing role of emergency endovascular aneurysm repair (EVAR) are altering the demands upon anaesthetists. Whereas previously on-site general anaesthesia for resuscitative open aneurysm repair (OAR) was standard, now transfer, choice of surgical technique and options for anaesthetic management may need to be considered. We present the key components of emergency anaesthesia for both OAR and EVAR and describe clinical dilemmas arising at preoperative and intraoperative stages.

Keywords Abdominal aortic aneurysm; coagulopathy; emergency anaesthesia; endovascular aneurysm repair; massive haemorrhage; transfusion

Royal College of Anaesthetists CPD Matrix: 2A03, 2A04, 2A05, 2C01, 3A05

Learning objectives

After reviewing this article, you should be able to:

- understand key determinants of surgical and anaesthetic strategies for ruptured abdominal aortic aneurysm (RAAA)
- evaluate patients with RAAA and appreciate the limited scope for patient optimization
- prepare a theatre for emergency open aneurysm repair
- describe key physiological implications of aortic occlusion and tissue reperfusion
- list perioperative complications of both open and endovascular aneurysm repair

Abdominal aortic aneurysm (AAA): the clinical problem

Overview

Ruptured abdominal aortic aneurysm (RAAA) requires urgent management to reduce mortality and major morbidity. Traditional open aneurysm repair (OAR) has only yielded limited incremental improvements in outcome for several decades.[1] Attempts to improve outcomes have focussed on avoiding rupture through screening programmes, service centralization in centres of excellence and expansion of endovascular aneurysm repair (EVAR) as technology has allowed.

Pathophysiology

Aneurysms are abnormal focal dilatations of greater than 50%; thus AAA diameters exceed 3 cm. Amongst AAAs, 90% are infrarenal and 10% juxtarenal or suprarenal. About 10% of aneurysms have an inflammatory cause.[2]

Established aneurysms progressively enlarge although expansion trajectories vary widely. Greater diameter is associated with higher rupture risk, for example 50 mm aneurysms confer a 1% annual rupture rate whereas those greater than 70 mm exceed 20%. More rapidly expanding and symptomatic aneurysms are higher risk, and given aneurysm dimensions represent greater risk in females than males.[3]

Presentation with pain from dissection before frank rupture confers the best chance of survival. With frank rupture around 75% mortality is expected with intraperitoneal bleeds faring worse than where retroperitoneal haematoma benefits from some tamponade effect. Around 50% of patients survive to presentation, the sub-group not shocked on arrival have a greater than 80% survival rate at 2 hours post-presentation (suggesting opportunity for imaging and transfer in selected individuals).[1,4]

Epidemiology

RAAA is a predominantly male condition (male:female 4:1),[1] the highest incidence of rupture occurs in those aged 65–85 years. Smoking, hypertension and hypercholesterolaemia increase incidence, as do some connective tissue disorders (e.g. Marfan's syndrome and Ehlers-Danlos syndrome); the association of diabetes with occlusive vascular disease is not mirrored in aneurysmal pathophysiology.

Service organization

Two key developments have altered AAA management in the UK in the past decade, as follows.

1. Centralization of services – recognition that greater throughput is associated with improved outcomes led to centralization into larger centres with sub-specialized staffing and infrastructure. However, in many centres the 'middle of the night AAA' will be managed by open repair involving generalist anaesthetists. The mortality benefit of centralization outweighs adverse outcomes caused by secondary transfer.[1]
2. The National Abdominal Aortic Aneurysm Screening Programme offers ultrasound imaging to males aged 65. Depending on aortic diameter patients are discharged, listed for episodic imaging, or (if diameter exceeds 55 mm) referred for vascular assessment. However, it leaves patients beyond the screening age un-imaged, and with a 1% annual rupture rate at 50 mm, some ruptures still occur in the screened group.

Patients with RAAA therefore either present at a large centre with on-site vascular services frequently managing this scenario, or to smaller non-vascular centres where communication (with

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the regional centre), decision making and transfer challenges will be greater.

Clinical management

RAAA management is characterized by complexity and time pressure; therefore, communication, delegation and concurrent activity, and pragmatic decision making are key to success.[2] Depending on where the patient presents, different demands may be faced; for example, from immediate theatre transfer and induction for resuscitative OAR, to participation in decision making that intervention or transfer is futile.

EVAR versus OAR

EVAR has an expanding role in elective and emergency AAA management.[1] Greater experience and technological improvement allows use for more challenging anatomy with faster procedure times that is more tolerable in the awake patient (allowing fewer general anaesthesia (GA) cases) and reduces ischaemic time. Cases previously judged too unstable for endovascular management may now be initiated with transfemoral balloon occlusion before angiographic assessment of the aneurysm with the option to progress to EVAR or OAR.

The potential benefits of EVAR include:

- potential avoidance of GA and ventilation-associated complications
- less stimulation requires a lighter plane of GA if this technique is chosen
- avoiding the 'dash to clamp' at induction by prior balloon occlusion
- avoiding laparotomy incision
- compared with OAR potentially, quicker, less blood loss, less tissue handling and dissection and easier avoidance of hypothermia.

EVAR is not universally applicable; factors influencing technique include availability of interventional services, surgical preferences and patient comorbidities. Aneurysm factors: site, size, length, neck angulation, tortuosity, landing zone characteristics, presence of intramural thrombus and access vessels also determine suitability.

Evidence remains inconclusive regarding relative merits of EVAR and OAR.[4] The literature recognizes that previous claims for marked EVAR superiority based on observational and registry case series are probably too subject to bias to be reliable. However, more recent studies have prospectively examined the outcomes of randomized patients and have allowed sub-group analysis by suitability for EVAR or age. Unfortunately these patient series are too recent to provide long-term outcome.

Preoperative phase

Anaesthetic preoperative priorities involve patient evaluation, optimization, and team preparation of theatre and equipment.

Patient evaluation: In unstable patients this occurs alongside resuscitation although normal responses to ABC findings may be modified (e.g. blood pressure should not be normalized). Unstable patients should not be anaesthetized for transfer/imaging as loss of tamponade and sympathetic drive risks irretrievable hypotension.

Sufficiently stable patients may undergo diagnostic confirmation typically with abdominal CT, which also evaluates the aneurysm anatomy and refines surgical decisions regarding technique.

A focussed anaesthetic history should be obtained; likely co-morbidities include those associated with causing the pathology. There is increased incidence of pathologies associated with atherosclerotic disease (i.e. coronary heart disease, heart failure, cerebrovascular disease and chronic kidney disease).[2]

Initial tests include cross-match (with activation of major haemorrhage pathways), full blood count, urea and electrolytes, liver function tests, calcium and coagulation. Arterial blood gases (ABG) and an ECG should ideally be performed.

Prognosis prediction at presentation has limited success. Scoring systems, including the Hardman Index and Glasgow Aneurysm Score, are insufficiently accurate alone but are useful adjuncts to current cardiovascular status, co-morbid condition and anatomical considerations when senior decision makers evaluate the patient.

Optimization: Time constraints limit opportunities for optimization; however, several useful measures can be undertaken.

Permissive hypotension improves mortality — higher blood pressure accelerates haemorrhage. However, systolic pressures below 70 mm Hg are also associated with increased 30 days mortality.[4,5] Limiting aortic wall stress requires control of significant hypertension through incremental doses of short acting analgesics (where pain drives the hypertension), β-blockers and vasodilators.[2]

Volume status should be carefully evaluated. Normovolaemia is not sought, although in markedly hypovolaemic and hypotensive patients sequential fluid boluses (e.g. 100—250 ml) may be appropriate. Iatrogenic coagulopathy is reversed (e.g. human prothrombin complex for warfarin); the increasing use of anti-platelet drugs and new oral anti-coagulants creates challenges that will require haematology advice.[2]

Hypothermia increases mortality — exacerbating acidosis and coagulopathy and measures to avoid this should be initiated early.

Analgesia may be beneficial for humanitarian and physiological reasons. Pain can drive both tachycardia with cardiac ischaemia and hypertension, however unthinking administration of opioids can obtund sympathetic drive which may be maintaining cardiovascular stability. Administration should therefore be in titrated small doses.

Preparation: Clear leadership and task allocation is required, where possible initially with two anaesthetists and two operating department practitioners involved in preparation — this allows one anaesthetist to remain with the patient while the other focusses on theatre preparation as described in emergency EVAR and open RAAA repair flowcharts (Figures 1 and 2).

All decisions need to be balanced against benefits of 'placement now' against time cost. Choices depend upon patient stability, (e.g. our practice is to gain arterial access pre-induction but not to persist if this delays gaining proximal control in an unstable patient).

Flowchart / checklist for anaesthetic management of emergency EVAR

	Preoperative
	High-flow oxygen
	IV access ×2
	Tests: Cross-match, FBC, U&E, Ca, Coags, ABG, ECG
	Activate blood bank
	Monitoring: ECG, SpO2 NIBP
	Arterial line if time
	Permissive hypotension: SBP 80–100 mmHg Low BP – fluid bolus 100–200 ml High BP – consider cautious analgesia, GTN, β-blocker
	Evaluate: anaesthetic history, allergies, medications, co-morbidities, functional status
	Warming: limit exposure, environment temperature, warming blankets, fluids warmed
	Urinary catheter
	Anaesthetic team: machine checks, drugs prepared, rapid infusor, blood products
	Confirm surgical plan
	Brief surgeon on anaesthetic plan
	Expedite transfer to EVAR suite
	Surgical team: instruments checked, surgeon scrubbed, patient draped

	Intraoperative
	Nominate dedicated communication person (if not GA)
	Monitoring: five-lead ECG, SpO_2, arterial line, periodic temperature, ABG, Hemacue, TEG, bloods
	Antibiotics
	Anaesthesia before proximal control Surgical LA Remifentanil if required (0.01–0.02 μg/kg/minute)
	Anaesthesia with proximal control As per preoperative briefing
	Optimize volume status
	Targeted transfusion aiming for Hb>80–100 g/L PTR<1.5 Fib>2 g/L or normal TEG Plt>100 × 10^9/L APTTR<1.5 Ca^{2+}>1 mmol/L
	Consider Tranexamic Acid 15 mg/kg
	During closing Contact ITU and prepare for transfer Intra-abdominal pressure measurement Analgesic plan Complete documentation

Figure 1

Operative phase: Induction to securing proximal control

OAR: Traditional OAR requires a cardiostable GA induction facilitating rapid surgical access to the aneurysm allowing proximal clamp placement; the aim is to minimize delay – incision immediately follows intubation. Combined effects of loss of muscle tone, reduced sympathetic drive and hypotensive effects of anaesthetic drugs may reduce blood pressure significantly with vasopressors and volume resuscitation used to mitigate the effect. Gross instability often ensues until cross-clamp application.

Induction recipes for unstable patients vary between experts and are not prescriptive, remembering that standard doses of induction agents risk gross cardiovascular instability and therefore opioid- or benzodiazepine-heavy cocktails are often preferred.

EVAR: With EVAR, options are more varied for both anaesthetist and surgeon. Surgical strategies sometimes involve placing an occluding balloon proximal to the aneurysm before or following anaesthesia (also an option in OAR). The anaesthetist is faced with several options:

- de novo induction of GA with similar risks as listed above
- Starting with local anaesthetic (LA) infiltration or ilio-inguinal regional anaesthesia (RA) to allow surgical access to groin vessels with subsequent transfemoral passage of an occluding balloon proximal to the aneurysm for inflation[5–7]

Flowchart / checklist for anaesthetic management of emergency open AAA repair

	Preoperative
	High-flow oxygen
	IV access ×2
	Tests: Cross-match, FBC, U&E, Ca, Coags, ABG, ECG
	Activate blood bank
	Monitoring: ECG, SpO2 NIBP
	Arterial line if time
	Permissive hypotension: SBP 80–100 mmHg Low BP – fluid bolus 100–200 ml High BP – consider cautious analgesia, GTN, β-blocker
	Evaluate: anaesthetic history, allergies, medications, co-morbidities, functional status
	Warming: limit exposure, environment temperature, warming blankets, fluids warmed
	Urinary catheter
	Anaesthetic team: machine checks drugs prepared blood products rapid infusor cell salvage Expedite transfer to theatre
	Surgical team: instruments checked, surgeon scrubbed, patient draped

	Intraoperative
	Monitoring: 5-lead ECG, SpO_2, arterial line, temperature, ABG, Hemacue, TEG, bloods
	Antibiotics
	Cardiostable GA induction
	Central line
	Optimize volume status
	Targeted transfusion aiming for Hb >80–100 g/L PTR <1.5 Fib >2 g/L or normal TEG Plt >100 × 10^9/L APTTR <1.5 Ca^{2+} >1 mmol/L
	Consider Tranexamic Acid 15 mg/kg
	During closing Contact ITU and prepare for transfer Intra-abdominal pressure measurement Analgesic plan Complete documentation

Figure 2

- Neuroaxial techniques have been described but are reserved for cases of cardiovascular stability without evidence of coagulopathy
- Non-GA methods may be supplemented with sedation or analgesia as required with the option of conversion to GA once proximal control is gained.[3]

Anaesthetic technique decisions involve many factors and are partly dependent on surgical plan – it is therefore essential that surgeon and anaesthetist exchange plans before beginning. Considerations for anaesthetic technique are listed in Box 1. Increasingly an LA/RA with sedation technique is initiated until proximal control is obtained. Thereafter continuation or GA conversion can occur as indicated.[2]

'Clamped' phase

Occluding the abdominal aorta with cross-clamp or endovascular balloon causes rapid and potentially dramatic changes in haemodynamics. Effects are generally greater with more proximal aneurysms. Traditional teaching emphasizes the instantaneous elevation in flow resistance causing increased mean arterial pressure (MAP) above the clamp which is beneficial for cerebral perfusion but increases left ventricular afterload and can significantly increase cardiac work risking reduced ejection fraction or cardiac ischaemia. MAP is also improved by continuing venous return from unperfused tissues, reduction in haemorrhage rate, sympathetic response to clamp application and activation of the renin–angiotensin–aldosterone axis although these factors take longer to become evident.

Distal to the occlusion, tissues progressively use anaerobic metabolism with reduced whole body oxygen consumption and elevated levels of acid, lactate, cytokines, potassium, prostaglandins and free radicals.

Proximal aneurysm control allows resuscitation to rapidly normalize physiology and prepare for clamp removal. Refilling the

Considerations for emergency EVAR anaesthesia technique

- Favouring local/peripheral regional anaesthesia
  - Avoids muscle relaxants and loss of abdominal tone
  - Avoids hypotensive effects of anaesthetic
  - Provides analgesia for access site (misses abdominal or ischaemic leg)
  - Avoids intubation and weaning
- Favouring GA
  - Still patient
  - No anxiety/agitation/intraoperative pain
  - Tolerance of long procedures
  - Easier respiratory manipulation, e.g. breath hold or hyperventilation
  - No need for conversion of technique mid-case
  - Allows transoesophageal echocardiography
- Factors contraindicating local/regional techniques
  - Local anaesthetic allergy
  - Anticipated prolonged duration
  - Severe abdominal pain
  - Agitation
  - Gross physiological instability, e.g. hypotension causing airway impairment
  - Anticipated secondary procedures, e.g. embolectomy or femoral–femoral crossover graft
- Other considerations
  - Patient preference
  - Initial haemodynamic status
  - Cardiopulmonary reserve
  - Coagulopathy (contraindicating neuroaxial techniques)

Box 1

patient limits the effects of prior hypovolaemia and hypotension. Transfusion is often required and massive transfusion protocols may be initially used, switching to targeted product administration as point of care and laboratory tests provide information.

Further preparation for clamp removal includes reduction or stopping of vasopressors as hypovolaemia is corrected – indeed some advocate use of vasodilators to allow greater filling to mitigate the vasodilator response to clamp removal. Evaluation of volume status can be difficult at this stage and some monitoring may not be of use e.g. oesophageal Doppler.

Increasing minute ventilation partially compensates for any metabolic acidosis. Metabolic products from ischaemic tissues alter cellular milieu and may promote dysrhythmia. Repeating ABG and bloods at this stage indicates metabolic status, guides administration of blood products and any electrolyte replacement.

A systematic review of the patient should occur shortly before unclamping which should occur only when anaesthetist and surgeon are content. An anaesthetic checklist for this phase would include:

- patient relatively hyperventilated
- intravascular volume optimized
- vasopressors running ± bolus dose immediately prior to unclamping
- fluids ready for rapid administration – however, the administration of large fluid volumes risks dilution of haemoglobin, platelets and coagulation factors
- having calcium and bicarbonate ready for administration if required.

'Unclamping' phase

Re-establishing distal perfusion on clamp removal or balloon deflation triggers cardiovascular changes that can be anticipated and modified. Immediately aortic MAP reduces with a consequent reduction in cardiac afterload and improvement in left ventricular ejection fraction. Usually central venous return falls, which impairs cardiac output. Previously anaerobic tissues now regain perfusion with transfer of metabolic products to the central circulation causing generalized vasodilation and myocardial depression. In summary, an acute fall in BP is accompanied by a reduction in arterial and mixed venous saturations, subsequently a further reduction in MAP is anticipated.

These physiological changes are partly dependent on factors over which anaesthetists have limited influence (e.g. cardiorespiratory reserve of the patient, level of clamp application and duration of clamping). Effects can be ameliorated through the following measures:

- optimizing the patient (as detailed above) prior to clamp removal
- continuing hyperventilation to limit acidosis
- supporting BP with vasoconstrictors
- maintaining euvolaemia as the patient vasodilates
- staged unclamping/deflation
- re-clamping/re-inflation if cardiovascular instability is particularly marked or surgical bleeding is observed.

Closing phase

Restoration of flow to ischaemic tissues induces a period of expected instability; following this a further set of predictable problems may become evident.

Bleeding may be either surgically identified or occult – only becoming evident through haemodynamic deterioration. Bleeding may be surgically remediable and can require re-clamping of vessels. Alternatively coagulopathy may be due to platelet or factor deficiency, or secondary to profound acidosis or hypothermia. Clinical assessment, thrombelastography and laboratory coagulation tests guide therapy. Coagulopathy can also exist in the absence of evident bleeding so these assessments may be routinely performed.

Ischaemia-reperfusion injury complicates tissue re-oxygenation; generation of reactive oxygen species and interleukins promotes an inflammatory response. This contributes to the vasodilated systemic inflammatory response syndrome (SIRS) state arising from initial pathology, surgical trauma and presence of ischaemic metabolic products.

Instrumentation of atherosclerotic vessels may have dislodged embolic debris impairing circulation in tissues to which they travel, these effects may only become evident later.

Observation of tight abdominal closure and any elevated ventilation pressures should prompt discussion regarding the benefits of laparostomy.

Alongside immediate clinical priorities preparation for transfer to critical care is undertaken.

Postoperative issues

Detailed critical care management of RAAA patients is beyond this article's scope; however, knowledge of likely issues informs intraoperative management and is relevant if patients require follow up surgery.

Intubated patients are usually transferred to critical care for further ventilation. All RAAA patients may experience a SIRS with progression of multi-organ failure and acute respiratory distress syndrome. The SIRS response in EVAR has been named post-stent implantation syndrome.[2,3,8]

Cardiovascular issues initially involve volume optimization and observation for signs of occult bleeding or tissue ischaemia secondary to vascular occlusion. Coagulopathy can develop later. A potential but rare cause of hypotension postoperatively is adrenal insufficiency due to clamp-associated hypoperfusion. Cardiac ischaemia remains a risk throughout the perioperative period.[8]

Neurological problems relate to pain (especially in OAR) and delirium which is common.[2]

Gut hypoperfusion and surgical handling can precipitate ileus and ischaemic colitis is possible. Patients with prolonged periods without nutritional intake fare worse and total parenteral nutrition may be required.[8]

Intra-abdominal pressures may be elevated and tight surgical closures are associated with abdominal compartment syndrome – this is due to direct tissue trauma and subsequent oedema, bleeding and haematoma, and organ failure with altered microcirculation in affected tissues. Intra-abdominal pressure monitoring may be indicated.[2,8]

Apart from vessel damage, EVAR-specific complications include spinal ischaemia due to covering of the perforating spinal arterial supply direct from the aorta, and large retroperitoneal haematomas which can cause raised intra-abdominal pressure and provide a rich site for infection.[2,3]

Finally, acute kidney injury may result from multiple factors. Renal ischaemia can be absolute, e.g. with supra-renal clamping (even clamps below the renal arteries can alter renal blood flow dynamics adversely), or relative resulting from low MAP, haemoglobin or oxygen levels. Additionally patients have contrast exposure and for many this is in addition to chronic kidney disease. Any rhabdomyolysis, ureteric injury or use of nephrotoxic drugs compounds these effects. Anaesthetic management minimizes the extent and duration of the above insults. Unfortunately there is no level 1 evidence supporting use of agents previously proposed for renal protective properties, examples include acetylcysteine, mannitol, dopamine and furosemide.[8] ◆

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