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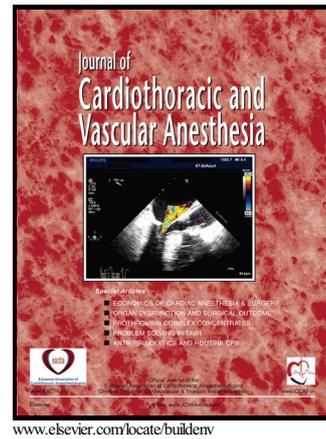
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TITLE PAGE

Modern Anaesthetic Management of Ruptured Abdominal Aortic Aneurysms

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INTRODUCTION

Ruptured abdominal aortic aneurysm (rAAA) is a surgical emergency for which anesthesia as a specialty has developed a more or less classic textbook approach.[1] However, recent initiatives such as the Abdominal Aortic Aneurysm Quality Improvement Programme (AAA QIP), [2] the centralization of vascular services in the United Kingdom (UK) and the Immediate Management of Patients with Ruptured Aneurysm: Open *Versus* Endovascular Repair (IMPROVE) trial [3] have challenged some of our traditional perceptions of the optimal anesthetic management of rAAA in contemporary practice. This review focuses on all the factors involved in modern day anesthesia for rAAA, including the impact of centralization, different surgical and anesthetic techniques, including the use of local anesthesia only for endovascular aneurysm repair (EVAR), perioperative resuscitation and fluid management.

EPIDEMIOLOGY

The prevalence of abdominal aortic aneurysm (AAA) in men between the ages of 65-74 is 5-7%, rising to >10% in those over 74. [4] Ruptured abdominal aortic aneurysm (rAAA) is the 13th commonest cause of death in the UK, responsible for 8,000 deaths per year. [5] In England and Wales alone, around 1200 repairs from rAAA are performed annually. [6] Despite a large reduction in mortality from rAAA at population level from 65.9 to 44.6 per 100,000 since the mid 1990's,[6]

(largely due to a reduction in smoking prevalence and improved medical treatment), mortality, once rupture has occurred, remains high. With variation

between different healthcare systems and countries, mortality ranges from 53 to 90%. [7] Without repair, the condition has almost 100% mortality within 3 days. [8]

DIAGNOSIS OF RUPTURED AAA

Ruptured AAA is an emergency that requires rapid and efficient preoperative evaluation and decision-making. Clinical signs and symptoms of rAAA vary from abdominal or back pain, in the presence of a pulsatile abdominal mass, to shock and cardiovascular collapse. [1] The diagnosis can be difficult, especially in elderly patients with multiple co-morbidities. A clinical diagnosis of rAAA by a senior emergency medicine clinician is sufficient to prompt referral and transfer to a specialist vascular center. Ultrasonography is helpful to diagnose the presence of an AAA, but it cannot diagnose rupture. Computed tomography (CT) is the most helpful special investigation to diagnose rAAA, but it should never delay patient transfer to a specialist vascular center. It is preferable to perform the CT in a vascular center rather than delay transfer. [9,10]

TRANSFER OF PATIENTS WITH rAAA TO SPECIALIST VASCULAR CENTRES

The best short and medium term outcomes in rAAA are seen in hospitals with larger bed numbers, [7] larger rAAA case volume (threshold of 15 rAAA's per year), [5,11] higher levels of surgical skill (surgeons maintaining higher volumes of vascular work have lower mortality rates) [12] and specialized vascular anesthetists. [13]

Centralization of specialist vascular services and recognition of vascular surgery as an independent specialty since 2012 are initiatives brought about as part of AAA QIP, set up by the Vascular Society of Great Britain and Ireland (VSGBI). [13] This was prompted by an international comparison of perioperative mortality after elective AAA surgery that showed that the UK had a high mortality rate compared to the rest of Western Europe. [14] More recent evidence from comparative studies confirms further international disparity also with regards to rAAA. [7] European data suggests that streamlined regional pathways improve outcomes for patients with rAAA.[15] Widely varied practices for transfer of patients with rAAA in England led to a three-round Delphi consensus approach [16] and also the publication of the joint best practice guidelines for the management and transfer of patients with a diagnosis of rAAA to a vascular center from the VSGBI, the College of Emergency Medicine and the Royal College of Radiologists.[17] Transfer of patients should be as swift and streamlined as possible; a paramedic crew is preferred but not essential; intravenous access and continuous vital sign monitoring is required before transfer; aggressive fluid resuscitation should be avoided and no blood products should be transferred with the patient. The clinical diagnosis of rAAA does not have to be supported by any investigations that could delay transfer to a specialist vascular center. Transfer is contra-indicated in patients who have had a cardiac arrest during the current admission. [9,10] Figure 1 is a suggested pathway for decision-making and management of the transfer of patients who present with rAAA. An audit

standard stating that appropriate patients with rAAA should be transferred to the nearest specialist vascular unit within 30 minutes after diagnosis, was introduced with the above guidelines.[10]

Treatment of patients with rAAA at specialist centers with significant volume and extensive experience may result in an increased acceptance of high-risk patients for surgical intervention. Although this might not reduce perioperative mortality, it could reduce overall mortality in rAAA, because no surgical intervention nearly always leads to death. [17] There is a need to measure the impact of centralization via clinical audit and prospective observational studies.

PRE-OPERATIVE MANAGEMENT

A ruptured AAA is a true surgical and anesthetic emergency and there is limited time for preoperative investigations or optimization of modifiable risk factors. [1] A rapid preoperative assessment is required to assess functional status, medical history and risk stratification as discussed below. In cases of significant cardiovascular collapse, immediate transfer to an operating theatre is required. However, in most cases there is time for CT angiography to confirm the diagnosis and also to assess the aneurysm morphology and possible suitability for EVAR. The patient should be monitored and accompanied by trained staff; there is a high risk of cardiovascular collapse and moving and rolling of the patient should be kept to a minimum. Successful resuscitation and anesthetic management often requires two anesthetists: in the initial phase one anesthetist

to treat the patient while the other oversees the preparation of the operating theatre, drugs and cell saver. The pathway from presentation to the operating theatre is illustrated in figure 2.

Risk Stratification & Patient Selection

Early assessment of co-morbidities and unmodifiable risk factors [18] must be carried out in all patients who present with rAAA, as well as establish whether the patient has an advanced directive. They are often elderly with premorbid conditions that contribute significantly to perioperative risk. [18] In some clinical scenarios attempted surgical intervention would be futile and in an attempt to stratify perioperative risk, scoring systems have been applied. [19] (Table 1)

1) **Glasgow Aneurysm Score (GAS).** A cut-off of 84 is currently used to signify high mortality; patients with a GAS <84 had a 30-day mortality of 28.2% compared to 65% in those with a score >84. [20]

2) **Hardman Index.** Most commonly used and seen as the gold standard in rAAA.[21] Systolic Blood Pressure (SBP) is not directly taken into account at presentation but the index is easy to calculate and well validated. It is also the only scoring system to be validated for rAAA for both operative and EVAR repair and for both immediate and 30-day mortality. [21]

3) **Acute Physiology & Chronic Health Evaluation (APACHE II).** This scoring system has long been used to assess disease severity in the critically ill. [22] It must have all variables submitted, therefore can be limiting or potentially cause delay. It is the only one of the scoring systems discussed here, which includes

an assessment of chronic disease, however points are only scored if the disease is severe. It is not specific to rAAA and studies are conflicting in its validation in this population. [23,24]

4) **Physiological and Operative Severity Score for the enUmeration of Mortality and morbidity (POSSUM).** This combines physiological and operative variables. Further POSSUM equations have been developed and validated specifically for vascular surgery and rAAA (V-POSSUM and RAAA-POSSUM respectively). [25]

Risk prediction scoring systems have limitations and should only be used in conjunction with clinical judgment. A recent paper has shown that GAS ≥ 96 was associated with poor long-term survival but > 20% of these patients survived 1 year. [26] None of the risk scoring systems currently in use provides reliable guidance to clinicians regarding decision to treat. It is also important to consider the contribution of factors, not included in current scoring systems, to prognosis, for example, the anatomical features of the aneurysm neck. The following features make the neck less suitable for repair by endovascular approach but unfortunately also increase mortality at open repair, therefore are independent predictors of mortality: 1) inappropriate zones for graft implantation (extreme calcification / thrombus); 2) proximal neck <10-15mm 3) angulation >60 degrees. [21,27-29]

Hemodynamic and fluid management

Traditionally, guidelines on the preoperative emergency management of patients with rAAA have always recommended the use of 'permissive hypotension' in

order to limit further blood loss. Target SBP being 50-100mmHg, unless signs of cerebral or myocardial hypoperfusion are seen. [30,31] However, recent evidence from the IMPROVE trial reported a significant association between the lowest SBP recorded pre-operatively and 30-day mortality, independent of fluid administration. Using a threshold SBP of 70mmHg, 30-day mortality below and above this was 51% and 34.1% respectively. [32] A recent meta-analysis of 29 studies, which defined hemodynamic instability as an SBP <80mmHg or shock index >1 also found a significant association with 30-day mortality. [33] Furthermore, a recent retrospective study showed that SBP <70mmHg was also associated with larger intraoperative blood loss. [34] These studies indicate that systolic blood pressure acts as an important prognostic indicator in patients who present with rAAA.

Guidelines and teaching on fluid management in rAAA have always been to restrict fluid administration pre-aortic clamping for fear of accelerating bleeding and dilution of clotting factors. [1,9] The most common advice being to give only 250ml fluid boluses to maintain SBP>70mmHg. [35] A recent retrospective study of IV fluids given to 248 patients with rAAA (96% underwent OR), showed that aggressive IV fluid administration pre-aortic clamping or endovascular sealing was associated with increased 30-day mortality (median volume 0.9L/hr pre-

op).[36] However, association does not prove causality. Also, in the IMPROVE trial, the effect of volume of fluid administered on 30-day mortality was not significant. [32] The potential benefit of decreased bleeding and lessening of further rupture should be balanced against the risk of end organ ischemia and dysfunction. More clinical studies are needed to provide evidence–based recommendations regarding the optimal blood pressure ranges, hemodynamic and fluid management for patients who present with rAAA.

INTRA-OPERATIVE MANAGEMENT

Surgical treatment for rAAA requires a relatively large multidisciplinary team to work efficiently. Therefore, good communication between the anesthetic team, vascular surgeons, interventional radiologists, emergency department doctors and theatre staff is crucial in the effective management of rAAA. The surgeon usually consults the interventional radiologist to make the decision whether the surgical approach should be via the open or EVAR route. Open repair is performed in an emergency operating theatre while EVAR is performed in a hybrid theatre. The immediate goal is control of hemorrhage by the surgical and/or interventional radiology team. The operating theatre should be prepared with a cell saver, pressurised warmed fluids, ideally through a rapid infuser device and ready to be administered through large bore iv cannulae. [1] Vasopressors and inotropes should also be prepared in advance.

Echocardiography may be useful as a method of detecting the rare complication of aortic dissection, which can occur during or after aortic surgery, sometimes as a consequence of aortic cross-clamping. Whilst transesophageal echocardiography (TEE) is the choice of technique for detecting aortic dissection, transthoracic echocardiography (TTE) can also provide useful information, as dissection is most often retrograde when caused by aortic clamping. [37] Both techniques require an experienced operator and in current UK practice, this is not a routinely used as a peri-operative investigative technique in non-cardiac surgery. It can however also provide information about dissection malperfusion (false and true lumens) by enabling visualization of blood flow. Cardiovascular instability may be an indication of dissection. However, evidence regarding the use of echocardiography in rAAA is limited. [38,39]

Anesthetic Technique

Induction of anesthesia in these patients is often associated with hemodynamic instability. This might be due the effects of anesthetic drugs, reduction in sympathetic tone and the loss of muscle tone maintaining tamponade, exacerbating intra-abdominal bleeding. Therefore, the patient is only induced once prepped and draped and with the surgeon ready to start immediately. Invasive blood pressure measurement before induction is ideal, but there should be no delay, especially if the patient is unstable. In the UK, a modified rapid sequence induction with carefully titrated doses of opiates, induction agents and vasopressors are often used. A variety of anesthetic techniques might be

suitable and there is currently no evidence that certain agents or a specific technique is superior. The main aim is to maintain hemodynamic stability and avoid hypotension. Central venous access should be established after induction and should not delay surgery.

Local vs. general anesthesia for EVAR

Studies have shown early on that it is possible to perform EVAR surgery under different types of anesthesia including general, neuro-axial and local anesthesia. [40,41] Currently, choice of anesthesia depends mostly on experience and personal choice of the anesthetist. There have been no prospective, randomized trials designed to specifically examine the outcomes between general anesthesia (GA) vs. local anesthesia (LA).

Analysis of over 5000 patients from the EUROSTAR database investigating the effect of the type of anesthesia on outcome showed a significantly lower mortality, morbidity and hospital stay in the group who received regional anesthesia (RA) / LA vs. GA. Further analysis of the data indicated that high-risk patients in particular, gain from less invasive anesthetic techniques with decreased mortality, morbidity, hospital stay and critical care admission in the regional/LA group vs. the GA group.[42,43]

A subgroup analysis of the IMPROVE trial showed that patients who had emergency EVAR surgery for ruptured AAA under LA had a significantly reduced odds of 30-day mortality compared to those who had GA (odds ratio 0.27, 95% CI 0.10 to 0.70) after adjustment for major potential confounders. [32] A systematic

review published in 2012 suggested differences in a number of outcomes between LA, RA and GA for EVAR, with LA appearing to be the safest option and GA the most hazardous. However, the review did not identify any RCTs comparing types of anesthesia and the authors were clear that the results could be due to confounding with higher risk patients receiving GA.[44] Theories about the potential causes of poorer outcomes under GA include relaxation of tissues and subsequent release of tamponade, as well as the significant hemodynamic effects of GA, including a loss in vascular tone, all of which may be exacerbated in shocked patients with ruptured AAA. Further studies have been published since the 2012 review and EVAR technology has also advanced significantly in recent years with the advent of narrow-gauge, "low-profile" grafts leading to

increased use of percutaneous techniques under LA. Therefore, there is a need for a high quality, adequately powered RCT, specifically comparing the influence of anesthetic technique for EVAR on outcomes in rAAA repair.

Surgical Approach: Open vs. EVAR

Endovascular approach to aneurysm repair was first reported in the early 1990's [45] and since then has increased in popularity as it avoids the major physiological insult of a laparotomy in this high-risk patient group. Observational studies from national datasets suggest that emergency EVAR for rAAA may be associated with a lower 30-day mortality rate. [11,46,47] The observational study outcomes may be explained by selection bias, whereby surgeons accepted more

hemodynamically stable patients to undergo EVAR. The recent IMPROVE trial was a prospective, 30-center trial, that randomized 600 patients with a clinical diagnosis of rAAA to undergo repair by either EVAR or an open approach. They reported no significant reduction between the 30-day mortality or cost of the two groups.[3] A recent individual-patient meta-analysis of three randomized controlled trials that compared EVAR versus open repair for rAAA, reported that woman might benefit more from the EVAR approach and that patients are discharged sooner after EVAR, but survival to ninety days was similar for both groups. [48] Despite this, earlier discharge from critical care, shorter hospital stay and a much higher proportion discharged directly home in the EVAR group means that the EVAR approach is likely to gain further support for use in rAAA, especially in specialist centers. Furthermore, the EVAR approach for rAAA would increasingly allow treatment of more elderly patients and those with significant comorbidities who would not be considered feasible candidates for open surgery.[7] Not all patients are anatomically suitable for EVAR (only about 65%) and even for patients who are suitable for EVAR, mortality remains high at about 25%.[3] The latest guidelines from the European Society for Cardiology recommend either open or EVAR surgical approach for patients who present with rAAA suitable for EVAR. [49]

Hemodynamic control in open AAA

Careful administration of short-acting vasodilators or opiates can be used to control increases in blood pressure that might occur after cross-clamping of the aorta. This also allows more aggressive fluid resuscitation via either crystalloid or blood products which, in turn, aids preparation for release of the aortic clamp. Central venous pressure (CVP), pulse pressure variation via the arterial line or non-invasive cardiac output monitoring can be used to guide to intravascular filling. [1] Aim for close to normotensive blood pressure.

Despite these strategies, release of the cross-clamp causes a sudden decrease in afterload and concurrent ischemia-reperfusion injury that often cause severe hypotension and acid-base disturbances with lactic acidosis. This can be managed by gradual release of the clamp and use of positive inotropic agents and vasoconstrictors. Optimal management of the application and release of the aortic cross-clamp requires good communication between the vascular surgical and anesthetic teams.

There is a high risk of peri-operative cardiac complications.[4,11] Intravenous nitrate infusions can be used if ischemic changes are visible on the ECG. Regular arterial blood gas analysis is used throughout to monitor resuscitation, acid-base and electrolyte balance.

Hematology

Every modern hospital should have a major hemorrhage protocol for the quick and appropriate issuing of blood and coagulation products. A recent

retrospective study showed that lower preoperative fibrinogen concentration and platelet count were associated with larger intraoperative blood loss. [34] Specifically, hypofibrinogenemia (<1.5 g/L) was associated with intraoperative blood loss >2L and higher 30-day mortality. [34]

Thromboelastography can reliably indicate fibrinolysis, hypercoagulability and / or hypofibrinogenaemia and is used commonly in larger arterial centers to guide transfusion practice. Where it is not available, formal laboratory testing of coagulation, including INR, platelets and fibrinogen is used. The use of a red cell salvage system has dramatically decreased the amount of donor blood commonly used in rAAA surgery. There is currently no consensus about red blood cell transfusion threshold in either elective or emergency vascular surgery and a remarkable lack of randomized controlled trials. [5,50] The UK based, National Institute for Health & Care Excellence (NICE) recently published guidance, recommending a red blood cell transfusion threshold of 80g/Liter and a post-transfusion target hemoglobin of 80-100g/Liter in patients with major hemorrhage. [51] However, each patient must be assessed on an individual basis.

Thermoregulation

Hypothermia caused by patient exposure, blood loss and administration of large amounts of blood products and resuscitation fluids can have further detrimental effects on coagulation. Every attempt should be made to achieve normothermia via upper body warm air blankets and warmed fluids. Warm air blankets should never be applied to the lower limbs as heat injury may occur during cross-clamping.

Renal Protection

Acute kidney injury after repair of rAAA is common with contributing factors including pre-existing renal impairment, perioperative hypovolemia, hypotension, aortic cross clamping and in the case of EVAR, intravenous contrast. A recent observational study reported the incidence of acute kidney injury after rAAA to be 43% after open surgery and 26% after EVAR. [52] There are different models for defining acute kidney injury. Although acute kidney injury defined by either AKIN (Acute Kidney Injury Network) or RIFLE (risk, injury, failure, loss and end-stage) criteria is associated with higher overall mortality, the AKIN criteria is a better predictor of mortality in patients who are undergoing AAA surgery. [53] The use of the AKIN criteria for defining acute kidney injury is also advocated by the Vascular Society of Great Britain and Ireland. Patients with acute kidney injury after surgical repair of rAAA have significantly higher mortality rates, independent of co-morbidities and other complications. [52-54] Therefore, it is important to do everything possible to minimize acute kidney injury. The use of drugs such as

dopamine, furosemide and mannitol to prevent kidney injury are not currently supported by any clinical evidence. The mainstay of the current renal protective strategy is to avoid nephrotoxic drugs, limit cross clamping time, especially supra-renal, and to maintain adequate perfusion by avoidance of hypotension and maintenance of adequate intravascular fluid volume.

POSTOPERATIVE CARE

All patients require postoperative transfer to ICU for further supportive care. Patients are at high risk of cardiac, respiratory and renal complications as well as intra-abdominal hypertension and ileus. Acid-base balance, electrolytes, renal function, coagulation and hemoglobin should be closely monitored. The lower limbs are closely monitored for signs of ischemia. Postoperative analgesia is a challenge and often an epidural is inserted after correction of the coagulopathy and before extubation. Neurological deficit due to spinal cord ischemia[55] is a rare, but devastating complication after rAAA and early diagnosis may be confounded by epidural use.

FUTURE PERSPECTIVES FOR rAAA

The endovascular revolution has changed the elective surgical management of patients with abdominal aortic aneurysms and the indication from recent trials is that it will continue to be used more widely for rAAA. [3,48,56] Ongoing development

of endovascular graft technology is expected to further enhance the use of this technique.[57] Future research needs to identify how the results from emergency EVAR for rAAA could be improved through the use of local anesthesia, fluid management, surgical technique such as the use of endovascular occlusion balloons or other measures. [48] The results from the IMPROVE trial indicated that women seem to benefit more than men from the endovascular approach for rAAA. [3] Further future challenges and areas for research include addressing the absence of information about target red blood cell transfusion threshold[50] in this patient group and whether indications and management of rAAA should be different in women and in the elderly. [48,57]

CONCLUSIONS

The treatment of patients who present with rAAA has recently gone through several changes and evolved to accommodate advancing surgical techniques, including the increased use of EVAR and the centralization of vascular services in the UK to specialist centers. Traditional perceptions of the optimal anesthetic management of rAAA are challenged by recent studies, such as the IMPROVE Trial [3,41] that has raised questions about the optimal mode of anesthesia for EVAR of rAAA and also the optimal hemodynamic management.

This patient group should benefit from the introduction of national clinical guidelines and audit standards. A continued, collaborative approach between surgeons and anesthetists should aid the collection of larger data sets. Along

with this, more prospective clinical research studies, including well designed, relevant randomized controlled trials are needed to inform anesthetic approach with regards to goal-directed hemodynamic and fluid management and also the optimal mode of anesthesia for EVAR of rAAA.

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FIGURE LEGENDS

Figure 1

Suggested pathway for decision-making and management of the transfer of patients who present with rAAA.

Figure 2

Pathway from presentation to the operating theatre.

FIGURE LEGENDS

Figure 1

Decision-making and transfer pathway for patients presenting with a ruptured abdominal aortic aneurysm (rAAA):

Figure 2

Patient pathway from presentation at vascular center to the operating theatre.

Table 1. Risk Stratification for rAAA
Table 1. Risk stratification for ruptured AAA

GAS		APACHE II		Hardman Index		POSSUM	
Variable	Score	Variable	Score	Variable	Score	Variable	Score
Age	No. years	Temperature (°C)	0 – 4	LOC	1		
Age >76	1			Age (years)	1, 2, 4 or 8		
		MAP (mmHg)		Cr >190 mmol/l	1	Cardiac signs	
Shock	17	Heart rate (bpm)		Hb <90g/l	1	Respiratory Signs	
Myocardial disease	7	Respiratory rate		Myocardial ischaemia	1	SBP (mmHg)	
Cerebrovascular disease	10	Oxygenation				Heart rate (bpm)	
Renal disease	14	Arterial pH				GCS	
		Na^+ (mmol/l)				Ur (mmol/l)	
		K^+ (mmol/l)				Na^+ (mmol/l)	
		Cr mmol/l				K^+ (mmol/l)	
		Haematocrit (%)				Hb (g/l)	
		WCC (x 10^9/l)				WCC (x 10^9/l)	
		GCS				ECG	
		Age (years)				Operation category	

Chronic Health Points	No. procedures
	Total blood loss (ml)
	Peritoneal soiling
	Malignancy
	Timing of operation

Score predicting high mortality risk		
>84 = 65% mortality	≥2 high mortality	
30-34 = 70% mortality	% predicted mortality	
	(≥3 unlikely to survive)	>35 = 90%
	calculated directly	

rAAA specific?	
Yes	Yes
No	Yes (if RAAA-POSSUM used)

Validated for open & EVAR repair?	
No	Yes
No	No

Myocardial disease: previous myocardial infarction and/or ongoing angina; Cerebrovascular disease: includes all grades of stroke including transient ischaemic attack; Renal disease: serum urea> 20 mmol/l and/or creatinine >150 mmol/l; LOC=loss of consciousness after presentation; Cr=serum creatinine; Hb=haemoglobin; acute myocardial ischaemia defined as depressed ST segments >1 mm and/or associated T wave changes on electrocardiogram (ECG); MAP=mean arterial pressure; GCS=Glasgow Coma Score; Ur-serum urea; Na=serum sodium; WCC=white cell count; Oxygenation: A-a PO_2 (FiO_2>50%) or PaO_2 (FiO_2<50%), A-aPO_2=Alveolar-arterial gradient of partial pressure of oxygen; FiO_2=fraction of inspired oxygen; PaO_2=partial pressure of arterial oxygen; Chronic Health Points: if patient has history of severe organ system insufficiency, points are awarded: for non-operative or emergency postoperative patients 5, for elective postoperative patients 2.

CLINICAL DIAGNOSIS OF RUPTURED AAA

➡ Immediately inform on-call Consultant Vascular Surgeon, Anaesthetist & Radiologist
➡ Move patient to resuscitation area
➡ Large bore IV access
➡ Brief targeted pre-operative assessment
➡ Activate major haemorrhage protocol - blood products cross matched / available
➡ Aim to maintain SBP > 70mmHg
➡ Inform 2nd anaesthetist to prepare theatre, drugs & equipment
➡ If possible insert arterial line

PATIENT UNSTABLE

PATIENT STABLE

Radiological diagnosis of ruptured AAA

DECISION NOT TO OPERATE

DECISION TO OPERATE

SUITABLE FOR OPEN REPAIR

SUITABLE FOR EVAR

Consider LA vs GA
discussion with patient,
anaesthetist, surgeon, radiologist

**MOVE TO PREPARED
OPERATING THEATRE**

**MOVE TO PREPARED
HYBRID THEATRE**

➡ GA induction with surgeons scrubbed
➡ Invasive monitoring
➡ Ongoing communication including with haematology

➡ GA or LA (be ready to convert to GA)
➡ Invasive monitoring
➡ Ongoing communication including with haematology

TRANSFER TO ICU

EMERGENCY DEPARTMENT

THEATRE / HYBRID

POST-OP

```
                    ┌─────────────────────────┐
                    │  DIAGNOSIS OF RUPTURED   │
                    │           AAA            │
                    │  (clinical or radiological) │
                    └─────────────────────────┘
```

➡ Large bore IV access
➡ Aim to maintain SBP >70mmHg
➡ Quickly assess clinical condition,
   co-morbidities & functional capacity

```
┌──────────────────┐  ┌──────────────────┐  ┌──────────────────┐
│ ➡ Cardiac arrest │  │ ➡ Age > 85       │  │ ➡ Age < 85       │
│ ➡ Intubated      │  │ ➡ Severe systemic│  │ ➡ No / mild / moderate │
│                  │  │    disease       │  │    systemic disease    │
└──────────────────┘  └──────────────────┘  └──────────────────┘
```

Transfer
contra-indicated ← Consultant discussion

```
┌─────────────────────────┐
│  REFER WITHOUT DELAY     │
│  TO VASCULAR CENTRE      │
└─────────────────────────┘
```

➡ NO OTHER INVESTIGATIONS ESSENTIAL:
➡ ABG & US useful if they don't delay
➡ CT confirmation not needed
➡ Analgesia & IV fluids as required

```
┌─────────────────────────┐
│        TRANSFER          │
│  (within 30 mins of      │
│      diagnosis)          │
└─────────────────────────┘
```

➡ Time-critical transfer - in 999 ambulance with
   paramedic crew preferable but not essential
➡ Medical escort needed if patient on inotrope support
➡ Unless transfusion already commenced, do not travel
   with blood products
➡ Analgesia & IV fluids as required

```
┌─────────────────────────┐
│  ON ARRIVAL TO VASCULAR  │
│        CENTRE            │
└─────────────────────────┘
```

```
┌──────────────────┐                    ┌──────────────────┐
│ Cardiovascularly │                    │ Cardiovascularly │
│     stable       │                    │    unstable      │
└──────────────────┘                    └──────────────────┘

┌──────────────────┐                    ┌──────────────────┐
│ Emergency Dept   │ ────────────────►  │  To emergency    │
│       CT         │                    │ operating theatre│
└──────────────────┘                    └──────────────────┘
```