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Abdominal aortic aneurysms (AAAs) are the 13th leading cause of death in the United States¹ and approximately 40,000 patients undergo elective AAA repair each year.² With the population aging, this number is expected to increase. Although the use of endovascular AAA repair is becoming more common, open repair, first reported by Dubost et al. in 1951 remains the gold standard.² This chapter will review the etiology, risk factors, diagnosis, pathophysiology, operative technique, perioperative management, and postoperative complications of patients undergoing open AAA repair.

Definition

The International Society for Cardiovascular Surgery/Society for Vascular Surgery defines an abdominal aortic aneurysm as a focal dilation of the aorta leading to a diameter at least 50% larger than normal.³ Normal abdominal aortic diameter ranges from 17 to 24 mm and this depends on several factors such as age, gender, and body habitus. AAA is defined as an aorta with a diameter more than 30 mm.³

True abdominal aortic aneurysms affect all three layers of the aortic wall and are characterized by their shape and location. Fusiform aneurysms, the most common type, involve the entire circumference of the aorta while saccular aneurysms involve only part of the circumference of the aorta.¹ The most common location for these aneurysms is infrarenal, usually located several centimeters below the renal arteries, extending to the aortic bifurcation, and frequently involve the iliac arteries. When the aneurysm involves the renal ostia, it is defined as pararenal.

Pararenal AAAs are characterized by the absence of normal aortic tissue between the upper extent of the aneurysm and the renal arteries.⁴ Approximately 8–20% of AAAs are pararenal, which includes both juxtarenal and suprarenal AAAs. Juxtarenal AAAs are infrarenal aneurysms that involve the lower margin of the origin of the renal arteries. Suprarenal aneurysms are defined as an aneurysm that involved the aorta above the renal arteries and they require reconstruction of renal arteries.⁵

Etiology

The majority of AAAs are due to atherosclerotic disease, but there are other causes (Table 14.1). Inflammatory aneurysms deserve a special mention and are defined by a thickened aneurysmal wall, perianeurysmal and retroperitoneal fibrosis,

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Table 14.1 Etiology of abdominal aortic aneurysms

1. Atherosclerosis
2. Cystic medial necrosis
3. Connective Tissue Disorders
a. Marfan's syndrome
b. Ehler–Danlos syndrome type IVs
c. Turner's syndrome
d. Polycystic kidney disease
4. Arteritis
a. Giant cell arteritis
b. Takayasu's disease
c. Relapsing polychondritis
5. Infectious
a. Acute
i. Syphilis
ii. Salmonella
iii. Staphylococcus
iv. Brucellosis
b. Chronic
i. Tuberculosis
c. Fungal (mycotic)
6. Posttraumatic
7. Inflammatory

and dense adhesions to adjacent abdominal organs.¹ They represent between 3 and 10% of all AAAs. They are more common in males and patients with long history of tobacco use. They tend to present in the sixth decade of life and are associated with increased surgical risk.⁶ They have a genetic component and are seen in patients with a familial history of AAAs. Inflammatory aneurysms are thought to arise from an immune response to an antigen in the adventitial wall. This antigen is unknown but is thought to be viral, a lipid, or a product of lipid peroxidation from atherosclerosis.⁷ Herpes simplex virus and cytomegalovirus have been identified in the aortic wall in patients with inflammatory AAAs.⁶ Patients with inflammatory AAAs are very symptomatic (back, flank, and abdominal pain) and have an elevated erythrocyte sedimentation rate.

Risk Factors

Abdominal aortic aneurysms occur predominantly in the elderly, with the average age of 70 years, with its incidence increasing with advancing age. The prevalence of AAAs is

Table 14.2 Risk factors for the development of abdominal aortic aneurysms

• Age >60 years
• Male
• Family history
• Hyperlipidemia
• Hypertension
• Chronic obstructive pulmonary disease
• Smoking
• Diabetes
• Caucasian race
• Sedentary life style
• Coronary artery disease
• Peripheral vascular disease

estimated to be 2–5% in patients over 65 years. AAAs are uncommon in patients under 50 years of age unless the aneurysm is familial, posttraumatic, or mycotic. Additional risk factors for the development of AAAs are listed in Table 14.2. There is a strong association between smoking and AAA formation. Not only is the rate of AAA formation fourfold higher in smokers, aneurysm growth is more rapid. AAAs can be familial and first-degree relatives of patients with a history of AAAs are prone to develop aneurysms at a younger age with a more frequent rupture rate.

In the United States, approximately 150,000 patients die from ruptured AAAs each year. The most important risk factor for rupture is aneurysm diameter. Patients with aneurysm diameters ≥ 6 cm have a 10–20% chance of rupture per year.⁸ Other risk factors for rupture include female gender, uncontrolled diastolic hypertension, familial history, severe chronic obstructive pulmonary disease (COPD), current tobacco use, and rapid expansion of the aneurysm.

Pathogenesis

The two main determinants of the mechanical properties of the aorta are elastic fibers and fibrillar collagens. Elastin in the media is responsible for the viscoelastic properties of the aorta while collagen present in both media and adventitia provides tensile strength to help maintain

the structural integrity of the aortic wall. Early in aneurysm formation, there is fragmentation of elastic fibers, and, as the aneurysm develops, there is degradation of collagen, which is the ultimate cause of rupture. This process is mediated by proteolytic enzymes such as the matrix metalloproteins (MMPs) and plasmin-generated plasminogen activators.⁹ There is also a reduction in smooth muscle cells in the media, which participate in vascular wall remodeling and have a protective role against inflammation and proteolysis.¹⁰

AAAs may be associated with mural thrombus formation. Since the thrombus is continuously exposed to blood flow, there is constant remodeling of the thrombus. This leads to ongoing coagulation and fibrinolytic activity as measured by increased serum thrombin–antithrombin III complex and D-dimer levels.¹¹ Although the thrombus may reduce wall stress, its increasing thickness may lead to local hypoxia at the inner layer of the media, which can induce medial neovascularization and inflammation.

Most AAAs are infrarenal because of hemodynamic differences in the distal aorta. These include a higher peripheral vascular resistance, increased oscillatory wall shear stress, and reduced flow.⁸ There is also increased MMP-9 expression in the infrarenal aorta. These conditions can predispose the distal aorta to inflammation and aneurysm formation.

Clinical Presentation and Diagnosis

Unruptured AAAs

The majorities of unruptured AAAs are asymptomatic and are diagnosed as an incidental finding in patients with concurrent coronary, peripheral, or cerebrovascular disease or during population screening. Patients may experience chronic vague symptoms of back and abdominal pain, which result from direct pressure or distention of adjacent structures. The average rate of aneurysm expansion is approximately 3–5 mm per year. AAAs are prone for rupture and recent onset of severe lumbar pain may indicate impending rupture.

Rarely, unruptured AAAs can be diagnosed after complications such as distal embolization or acute thrombosis. Ureterohydronephrosis may occur with inflammatory aneurysms or aneurysms involving the iliac bifurcation.

AAAs can be diagnosed by palpating for a pulsatile mass in the supraumbilical area, but this is a poor screening tool with a sensitivity of approximately 68%. Sensitivity of abdominal palpation increases with the diameter of the lesion and decreases with abdominal girth.¹² Radiologic imaging techniques such as abdominal ultrasound, computed tomography (CT), or magnetic resonance imaging (MRI) are the preferred modalities to diagnose AAAs. An AAA may be diagnosed on a lateral abdominal x-ray if calcifications are present, which may allow visualization of the dilated aorta. Abdominal ultrasonography is the simplest and least expensive diagnostic modality. It can accurately measure aortic size and can be used for diagnosis, follow-up surveillance, and screening.¹³ If there is an aneurysm detected on screening, the next step is to perform an abdominal CT to determine the type of treatment.¹⁴ Information that can be elicited from a CT includes visualization of the proximal neck, extension to the iliac arteries, patency of the visceral arteries, and the presence and thickness of a mural thrombus. CT can also detect anatomic variants such as a left vena cava, posterior left renal vein, or horseshoe kidney that may interfere with the procedure, and can reveal blood within the mural thrombus called the crescent sign, which may be a predictive marker of imminent rupture.¹⁵ In patients with inflammatory aneurysms, CT can estimate the thickness of the aortic wall and determine the presence of para-aortic fibrosis. CT can also be used to determine the suitability for placement of endovascular devices. (Figs. 14.1 and 14.2).

MR angiography is useful in patients with suspected renal involvement because the gadolinium contrast material is less nephrotoxic than CT contrast. CT and MRI provide the same information, and the choice of study depends on the preference of the surgeon and radiologist, the availability of the study at the medical center, the patient's hemodynamic status, the ability of



Fig. 14.1 CT reconstruction of infrarenal abdominal aortic aneurysm



Fig. 14.2 CT angiography of infrarenal abdominal aorta



Fig. 14.3 Angiogram of abdominal aorta showing infrarenal AAA

the patient to lie still for an MRI examination, and the presence of renal dysfunction.

Angiography, the gold standard for the diagnosis of abdominal AAAs, is reserved for patients with inconclusive CT or MRI studies, in patients with more extensive aneurysms, such as those which involve the kidneys or viscera and require a more accurate determination of branch anatomy, in patients with peripheral occlusive disease, and in patients with renal abnormalities such as horseshoe or pelvic kidneys (Fig. 14.3).¹⁶

Ruptured AAA (RAAA)

A total of 50–60% of AAAs will rupture in 5 years. Larger aneurysms are more prone for rupture and 98% of the ruptured AAAs occur infrarenally. Rupture of the anterolateral wall of the aneurysm into the peritoneal wall carries mortality between 60 and 97% (Fig. 14.4 and 14.5). Most patients (88%) present with a rupture of the posterolateral wall into the retroperitoneum where the rupture can be sealed by clot formation, retroperitoneal hematoma, and abdominal muscle tone, and hemorrhage is limited by hypotension. This event is followed within hours by a larger rupture. The period between the two ruptures is called the intermediate period and is



Fig. 14.4 Intraoperative image of ruptured abdominal aneurysm



Fig. 14.5 Severe atherosclerotic ruptured abdominal aorta opened after cross-clamping

the time where the diagnosis and emergency repair can be accomplished.¹ The triad of symptoms of an RAAA include sudden onset of mid-abdominal or flank pain radiating to the scrotum, circulatory shock, and the presence of a pulsatile abdominal mass. The presence of flank ecchymosis (Grey Turner sign) represents retroperitoneal hemorrhage. Diagnosis is mostly clinical, but ultrasound, CT or MRI scanning, or aortography can also be helpful depending on hemodynamic stability. Contrast CT shows extravasation of contrast material, which is diagnostic of rupture. Early diagnosis reduces mortality by 50%. In rare cases, the first episode of rupture can be contained to become a chronic pulsatile extra-aortic hematoma or aneurysms can rupture into another structure, such as the duodenum, producing an aorto-duodenal fistula or the vena cava, which can lead to lower extremity edema.¹⁷

Surgical Management

Indications for Open Repair

Open AAA repair is recommended for aneurysms associated with an aortic neck >28 mm or significant angulation defined as $>45^\circ$ from midline, or those associated with laminated thrombus at the proposed site of stent-graft attachment. Open AAA repair is also recommended for the majority of pararenal aneurysms because endovascular repair with fenestrated grafts is a long complicated procedure, with the potential for failure, and is reserved for elderly or high-risk patients.¹⁸ Patients with atherosclerotic disease of the femoral, iliac, or renal arteries may be difficult to treat endovascularly.

Open Versus Endovascular AAA Repair

The recent endovascular aneurysm repair (EVAR-1) and Dutch Randomized Endovascular Aneurysm Management (DREAM) trials addressed management of AAAs larger than 5.5 cm in diameter. They randomized patients appropriate

for open repair between endovascular repair (EVAR) and open repair (OR). EVAR decreased perioperative complications, including myocardial events, and was associated with decreased recovery times. In patients with large AAAs who are fit for OR, EVAR offers an initial mortality advantage over OR, with a persistent reduction in AAA-related death at 4 years. However, EVAR offers no overall survival benefit, is more costly, and requires more interventions and indefinite surveillance with only a brief Quality of Life (QOL) benefit.¹⁹ Open repair is more durable and does not require the extensive surveillance unlike endovascular repairs. This may lead to a better quality of life. The DREAM trial patients received QOL questionnaires preoperatively, then at 3 weeks, 6 months, and at 1 year. Preoperative scores were similar. At 3 weeks, the patients undergoing endovascular repair had a better quality of life, but at 6 months and 1 year, the patients undergoing the open repair reported a better quality of life.²⁰

Techniques of Infrarenal AAA Repair

There are two surgical approaches for open AAA repair, midline transperitoneal approach, or a left retroperitoneal approach. Both can be performed either by laparotomy or by laparoscopic-assisted technique.

Transperitoneal Approach

With this approach, a midline incision is performed, the transverse colon and omentum are covered with moist towels and packed in the upper abdomen. The small bowel is reflected to the right side of the peritoneal cavity and a retractor is placed to maintain exposure (Fig. 14.6). The retroperitoneal lining is excised and the aorta is dissected until the renal artery is exposed proximally and the common iliac arteries are exposed distally. During the proximal dissection, bleeding can occur from injury to the left lumbar, gonadal, adrenal, or renal veins, especially when the retroperitoneum has inflammatory or fibrotic changes. Injury to the ureters or iliac veins can occur during distal dissection.

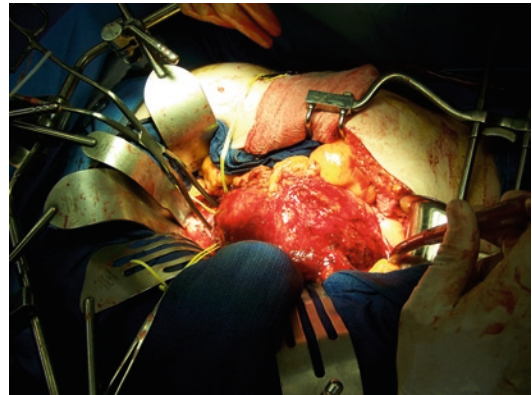


Fig. 14.6 Transperitoneal approach to the abdominal aortic aneurysm

The aneurysm is mobilized at the neck and the common iliac arteries are controlled at their bifurcations to prepare for clamping. Intravenous heparin is administered, vascular clamps are applied, and the aneurysmal sac is opened. Atherosclerotic debris and thrombi are removed, and back-bleeding from the lumbar and inferior mesenteric arteries is controlled with sutures within the sac. The aortotomy is extended to the aneurysmal neck and the aorta transected. A tube graft repair is performed, first with the proximal anastomosis to the aorta. After the proximal anastomosis is completed, a second clamp is placed on the graft to assess the strength of the anastomosis under pressure. The distal anastomosis is then performed either to the distal aorta or iliac arteries. The iliac artery anastomosis is preferred over the common femoral artery anastomosis because the femoral anastomosis is more prone to infection. The aneurysm sac is then closed over the graft, the retroperitoneum is approximated, and then the abdominal wall and skin are closed.

Retroperitoneal Approach

It is the technique of choice in patients with prior abdominal surgery, urinary or enteric stomas, previous abdominal or pelvic radiation, ascites, patient who are on peritoneal dialysis, morbid obesity, inflammatory aneurysms, aneurysms associated with horseshoe kidneys, and juxta- or suprarenal aneurysms. This approach

is indicated in the presence of accessory renal arteries and when removing an infected aortic graft. The retroperitoneal approach may be associated with lesser blood loss, fluid and blood transfusion, better pain control, improved pulmonary function, earlier return of bowel function, and reduced incidence of wound dehiscence.²¹ Disadvantages of this approach are that it may be difficult to access or control the right iliac artery and that the intraperitoneal contents cannot be inspected. Aortobifemoral grafts for aorto-occlusive disease also require transperitoneal approach.

Pararenal AAA Repair

Juxtarenal AAA repair requires suprarenal clamping, but the aortic reconstruction is performed infrarenally. Most juxtarenal AAAs can be approached by a retroperitoneal approach.⁵ For suprarenal AAAs and type IV thoracoabdominal aortic aneurysms, the patient is placed in the right lateral decubitus position with the patient's shoulders raised to 60° and the torso should be rotated so that the hips are as parallel to the table as possible. The retroperitoneal dissection is extended to the diaphragmatic hiatus, and the left crus is divided to expose the supraceliac aorta. The proximal clamp is then placed above the celiac axis or between the renal and superior mesenteric arteries. The inferior mesenteric artery is tied close to the aortic wall to keep it collateral to the superior mesenteric artery. In some cases, the inferior mesenteric artery is reimplanted into the aortic prosthesis to maintain blood flow to the sigmoid colon and rectum. Reimplantation is required if there is significant stenosis of the superior mesenteric artery and celiac trunk or if internal iliac flow cannot be restored after abdominal aortic surgery.²² If the left renal artery cannot be directly included into the anastomosis, it has to be reimplanted into the graft after primary reconstruction has been completed. The surgical risks of pararenal repair are greater because of renal and possibly mesenteric ischemia, increased alterations in cardiovascular physiology due to the higher aortic cross-clamp, and atheroembolization.

Laparoscopic Approach

Ferrari et al. described a laparoscopically assisted approach to AAA repair that is associated with less morbidity and mortality than a standard laparotomy approach.²³ This technique can be used to treat elective infrarenal and pararenal aneurysms. Contraindications for this approach include previous abdominal or aortic surgery, bilateral diffuse common iliac and/or internal iliac aneurysms, massive aortoiliac calcifications, inflammatory aneurysms, and severe underlying cardiac or pulmonary disease. The main advantage of this procedure is a decreased length of stay. Coggia et al.²⁴ described a total laparoscopic infrarenal aneurysm repair. This technique has the potential for faster wound healing and decreased discomfort.

Systemic Inflammatory Response Syndrome (SIRS)

Systemic inflammatory response is considered to be the precursor of morbidity and mortality after open repair.²⁵ The inflammatory response is higher in patients presenting for RAAA repair compared to patients presenting for elective AAA repair and the levels of inflammatory mediators correlate with the development of acute respiratory distress syndrome and multiorgan failure (MOF). Operative mortality following ruptured AAA repair may be divided into early and late deaths. The most frequent cause of early death is hemorrhagic shock while up to 90% of deaths after 24 h are caused by MOF. SIRS arises from the response to extensive surgical trauma, the release of vasoactive agents due to bowel manipulation and mesenteric traction, response to graft material, intestinal ischemia from intraoperative hypotension, aortic clamping and ligation of inferior mesenteric artery, ischemia-reperfusion injury, blood transfusions, which alter cytokine production, and cytokines released from the mural thrombus.

Endotoxin, which has been implicated as the trigger for the inflammatory response, can be released directly from bowel ischemia-reperfusion or indirectly through the release of inflammatory mediators after lower limb reperfusion.²⁶

The clinically significant cytokines that are released include TNF- α , IL-1 β , IL-6, and neutrophil CB 11b. IL-1 β is a locally acting cytokine, which acts as a pyrogen, induces hypotension, activates and promotes leukocyte adherence, induces acute phase protein synthesis, and stimulates the secretion of IL-6. Increased IL-6 is the most consistent response to aortic surgery. It enables the neutrophils to release TNF- α and neutrophil CD11b, which, along with IL-6, have been implicated as mediators of SIRS and MOF.²⁷ A study by Vasdekis et al.²⁵ demonstrated higher levels of endotoxin, IL-6, and neutrophil CD11b in patients who developed postoperative complications. Excessive manipulation in AAAs with intramural thrombi can lead to a clinical response similar to SIRS that is not seen in patients with small or no mural thrombi.

Risk Stratification and Perioperative Mortality

The operative mortality for elective AAA repair has been reported to range from 1.1% to 7%.² Risk assessment methods have become an essential part of preoperative evaluation and consent process for any surgical procedure. For unruptured AAAs, various risk-scoring systems have been tested to predict the risk of postoperative mortality and morbidity. Glasgow Aneurysm Scale (GAS), Vascular-Physiologic and Operative Severity Score for the enUmeration of Mortality and Morbidity (V-POSSUM), and Vascular Biochemical and Hematological Outcome Model (VBHOM) score are few of the risk-scoring methods (Table 14.3).

GAS is simple and easier to use compared to other scoring systems and has been validated numerous times and predicts mortality in elective and ruptured AAA. GAS does not reliably identify individual high-risk patients and does not predict morbidity. The V-POSSUM score is an equation based on 12 preoperative variables each scored and a total score obtained. This is a complex scale with numerous perioperative variables and several subjective-based parameters. The use of physiologic

Table 14.3 Risk stratification scores for abdominal aortic surgery

Glasgow Aneurysm Scale = Age + (17 for shock) + (7 for myocardial disease) + (10 for cerebrovascular disease) + (14 for renal disease)
V-POSSUM physiology-only score = $\ln(R/1 - R) = 6.0386 + 0.1539$ (physiology score)
Physiologic parameters
Age
Cardiac and pulmonary signs and symptoms
Systolic blood pressure
Heart rate
Serum sodium and potassium
Blood urea
Hemoglobin level
White cell count
Electrocardiography
Intraoperative variables
Grading of operation
Number of procedures
Total blood loss
Peritoneal soiling
Presence of malignancy
Timing of surgery
VBHOM = $\ln(R/1 - R) = -2.257 + 0.1511$ (gender)
+0.9940 (mode of admission) + 0.05923 (age on admission)
+0.001401 (urea) + 0.01303 (sodium)
+0.03585 (potassium) + 0.2278 (Hb) - 0.02059 (WBC).
Hardman Index
Preoperative Hb < 9 gm/dl
Serum creatinine > 1.9 mg/dl
ECG evidence of ischemia
In-hospital loss of consciousness
Age > 76 years

scores yielded inconsistent results when used to predict outcome after AAA repair. One reason for this outcome was that missing variables were scored as normal, which can overestimate the mortality in low-risk patients. The VBHOM score predicts mortality based on single biochemical and hematologic tests and is simple and suitable for collection in both emergency and elective situations. This model is yet to be validated.

Patterson et al. in a systematic review comparing different scoring systems concluded that GAS is the most useful and consistently validated scoring method for elective AAA repair.²⁸ A risk score that determined an individual patient's predicted risk in specific hospitals would be useful

in the consent process because the hospital volume of AAA cases and experience of the surgeons and staff influence the outcome significantly. Center-specific mortality has to be taken into consideration in addition to the patient and operative variables.

The intraoperative mortality for RAAAs ranges from 50% to 70% and various scoring systems have been evaluated to predict mortality after ruptured AAA. The Hardman system is the most well-known scoring system with five independent variables (Table 14.3). The mortality rate was 100% with three or more risk factors, 48% with two risk factors, 28% with one risk factor, and 18% with no risk factors.¹ POSSUM-RAAA score (combined operative and physiologic variables), the GAS, and Vancouver scoring systems are some other models studied in RAAA.

Irrespective of the scoring system used, the following parameters predict poor outcome: advanced age, cardiac arrest with cardiopulmonary resuscitation, hypotension (systolic BP < 80 mmHg on admission and < 100 mmHg at the end of surgery), loss of consciousness, low hematocrit, increased creatinine, ischemic heart disease, free intraperitoneal rupture, operating time > 4 h, the administration of more than 7 L of fluid, blood loss > 11 L, and blood transfusion of more than 17 units.²⁹ Tambyraja et al. in a review of published evidence revealed that none of the current scores has the ability to accurately predict outcomes during ruptured AAA repairs. The decision to operate or not in an individual patient is based on the subjective evaluation of the experienced surgeon until further evidence is available.³⁰

For patients who survived 48 h after RAAA surgery, Laukontaus et al. demonstrated that the best predictors of 30 day mortality include the preoperative Glasgow score, the need for supra-renal clamping, and the sequential organ failure assessment score (SOFA).³¹ The SOFA score uses the PaO₂/FiO₂ ratio, the platelet count, and presence of hypotension, serum bilirubin levels, the Glasgow Coma Scale, and the renal creatinine to predict the likelihood of developing MOF.

Preoperative Evaluation and Preparation

Clinical evidence of coronary artery disease (CAD) is reported in 25–69% of patients by various investigators. The rate of cardiac-related mortality ranges from 0 to 8% and cardiac complications account for 62% of all deaths after AAA repair.¹⁷ Hertzner et al.³² in their series from Cleveland Clinic revealed that in patients with AAAs undergoing coronary angiography, 6% had normal coronaries, 29% had mild to moderate coronary artery disease (CAD), 31% had correctable CAD, 12–15% had severe correctable CAD, and 5% had inoperable CAD. In their series, 44% with clinical indicators of CAD and 15% without clinical indicators had angiographic demonstration of severe CAD.

According to American Heart Association/American College of Cardiology (AHA/ACC) recommendations, patients with active cardiac conditions, such as unstable or severe angina, recent ST elevation or non-ST elevation MI, decompensated cardiac failure, or significant ischemic cardiac arrhythmias should undergo coronary angiography and revascularization before elective AAA repair. Coronary revascularization is also indicated in patients with stable angina with left main disease, stable angina with 3-vessel disease, and stable angina with 2-vessel disease and proximal left anterior descending (LAD) stenosis with low left ventricular ejection fraction (LVEF) less than 0.50 where the survival benefits are proven.³³

Preoperative treatment of patients with no active cardiac conditions but with significant clinical risk factors, such as a history of CAD, stable angina, treated stable or past history of heart failure, cerebrovascular disease, age more than 70 years, renal insufficiency, and diabetes mellitus is more complex. It is controversial whether these patients benefit from preoperative testing and subsequent revascularization. Few clinical trials addressed this issue.

Mcfalls et al. randomized their vascular surgical patients into revascularization group ($n=258$) and no-revascularization group ($n=225$) and

found a similar incidence of 30-day mortality, myocardial infarction (MI), and mortality after 2.7 years (Coronary Artery Revascularization Prophylaxis study-CARP). Though they excluded patients with unstable angina, left main coronary artery stenosis, and a low left ventricular ejection fraction (LVEF), 74% of their patients had 2 or more clinical risk factors or moderate or large reversible perfusion defects on nuclear stress testing.³⁴

The DECREASE-2 (Dutch Echocardiographic Cardiac Risk Evaluation Applying Stress Echo) clinical trial evaluated the value of preoperative cardiac stress testing in intermediate risk (1–2 risk factors) patients. Patients ($n=770$) were randomly assigned to testing ($n=386$) and no testing. All patients received beta-blockers. Tested and nontested patients had similar 30-day cardiovascular mortality and nonfatal myocardial infarction after vascular surgery. Limited ischemia was detected in 17% and extensive ischemia (>3 walls in stress perfusion scintigraphy or >5 segments in dobutamine stress echocardiography) in 8.8% of tested patients. Of 33 patients with extensive ischemia, only 12 were suitable for revascularization, which did not improve 30-day outcome. However, the cardiovascular death or MI was higher in patients with extensive ischemia (odds ratio, OR=107) and limited ischemia (OR=42), compared to patients with no ischemia.³⁵

The same group of investigators followed up with the DECREASE V clinical trial in which they randomized high-risk (> 2 clinical risk factors) patients with extensive ischemia during cardiac testing into revascularization ($n=49$) and no-revascularization groups ($n=51$). Revascularization did not improve 30-day or 1-year composite outcomes (cardiovascular death or nonfatal myocardial infarction) after the intended vascular surgery.³⁶

The results from these studies indicate that high or intermediate risk patients with stable CAD may undergo AAA repair without preoperative revascularization if medical therapy is expected to yield similar protection. The lack of benefit of revascularization is not fully understood, though plaque rupture induced by the perioperative stress response in nonsignificant

coronary lesions, which are not revascularized, may be the culprit.³⁷

Noninvasive stress testing has a low positive predictive value and is not routinely recommended in intermediate and low-risk groups. High risk (>2 risk factors) with poor functional status may be referred for stress testing for risk stratification, though the benefits of revascularization in patients with extensive ischemia are not well demonstrated.

The recommendation for coronary angiography (CAG) is similar to the nonoperative setting and it is the clinician's responsibility to take patient-specific factors into account before sending the patients for CAG before AAA surgery. Routine CAG is not advised before AAA repair because of its inherent risk, cost, and manpower.³⁷ Risk of CAG, mortality of revascularization procedure before AAA repair, hazards of AAA rupture during the waiting period, and risk of AAA surgery, all these put together do not justify routine CAG in all patients presenting for AAA repair.

Among the revascularization methods, percutaneous coronary intervention (PCI) was associated with increased risk of MI compared to coronary artery bypass grafting (CABG) in DECREASE V and CARP trials. Several other studies raised similar concerns. The increased risk could be related to stent thrombosis after PCI, especially if dual antiplatelet therapy (aspirin and clopidogrel) is discontinued perioperatively. The type of PCI planned will depend on the interval between PCI and planned noncardiac surgery. If AAA repair is planned within a month, bare metal stent implantation or balloon angioplasty are the most recommended options. With bare metal stents, after the month of dual antiplatelet therapy, the clopidogrel may be discontinued but aspirin should be continued. If discontinued, the clopidogrel should be restarted as soon as possible in the postoperative period. Drug-eluting stents require 12 months of uninterrupted dual antiplatelet therapy before discontinuing clopidogrel to prevent stent thrombosis.³³

In patients with unstable CAD and AAA, the options for treatment include a combined CABG/AAA repair or a staged approach with shorter

interval (2 weeks) between revascularization and AAA repair. The disadvantage of the staged procedure is postoperative rupture of the AAA, which can occur in up to 30% of patients.³⁸ The staged approach is therefore appropriate when aneurysm rupture is not imminent, such as with nontender aneurysms between 5 and 8 cm in diameter.³⁹ The combined approach has been adopted in patients when rupture of the aneurysm is imminent in the immediate postoperative period, which includes tender aneurysms, large aneurysms >8 cm, rapidly enlarging aneurysms, and aneurysms with recent contained leaks. The advantages of the combined procedure are a reduced total anesthesia time, convalescence, hospital stay, and total costs.^{40,41} The CABG can be done on or off cardiopulmonary bypass (CPB).^{42–47} In pump cases, both CABG and AAA repair were done while on CPB in some patients while in others, AAA resection was performed after weaning from CPB.⁴⁷ CABG on CPB is known to activate inflammatory mediators, produce coagulopathy and embolic phenomenon leading to end organ damage. Operative mortality is high in these patients and ranges from 6% to 33%.⁴⁸ Off-pump CABG is a safe and effective alternative to on-pump CABG in these patients. In selected patients, endovascular AAA repair with CABG is an option and can be done either as combined or staged procedure.⁴⁸

The anesthetic management of the combined procedure is similar to that for CABG alone. When the AAA is performed following the CABG, the initial heparinization required for CPB is reversed with protamine and then an additional smaller dose of heparin (5,000 U) is administered prior to aneurysm clamping. In patients who are hemodynamically unstable or who have large aneurysms, the surgeon may elect to perform the repair on CPB. This technique is associated with longer bypass times, which can be complicated by post-bypass coagulopathy, myocardial dysfunction due to long ischemic times, and hemodynamic instability.⁴⁹

In patients with diffuse small-vessel CAD and poor ventricular function, preoperative medical optimization with aspirin, statins, and beta-blockers and intensive perioperative monitoring may

reduce the risk of AAA repair. Other options for these patients include serial three-monthly observations with abdominal ultrasound or internal iliac ligation with axillobifemoral bypass.⁵⁰

Besides CAD, the preoperative evaluation should detect and determine the extent of other comorbid diseases including diabetes, hypertension with left ventricular diastolic dysfunction, COPD, chronic renal insufficiency, and cerebrovascular disease. Medications that are commonly prescribed to these patients include antihypertensive agents, oral hypoglycemic agents, insulin, diuretics, inhaled bronchodilators and steroids, antianginal agents, antiplatelet agents, and anticoagulants. Except for diuretics and possibly angiotensin-converting enzyme inhibitors, patients should continue their antihypertensive regimen up to the time of surgery.

Preoperative laboratory tests should include the baseline hematocrit, white cell with differential count to assess for occult infection, platelet and coagulation studies when epidural anesthesia is planned, electrolyte, renal, and hepatic panels, and urine analysis to rule out the presence of a urinary tract infection since a prosthetic graft is used. Blood should be typed and cross-matched. Fresh frozen plasma and platelets are obtained if the patient has a baseline coagulopathy or is taking antiplatelet and anticoagulant agents at the time of surgery. Since the majority of patients undergoing open AAA repair smoke or have COPD, pulmonary function tests and arterial blood gases may be useful in determining the extent of the patient's pulmonary dysfunction.

Monitoring

Standard ASA monitoring (ECG, noninvasive blood pressure, pulse oximetry, capnography, temperature), invasive arterial blood pressure, central venous pressure (CVP) or pulmonary artery catheter (PAC), urine output, and transesophageal echocardiogram (TEE) are used in these patients. Lead V₅ in ECG alone has the ability to identify approximately 75% of ischemic episodes and the combination of leads II, V₄, and V₅ have been demonstrated to have 96% sensitivity in detecting

myocardial ischemia.⁵¹ Arterial catheterization may be performed prior to the induction of anesthesia to monitor the hemodynamic changes during induction. In patients with peripheral vascular disease, difficult arterial cannulation should be anticipated. Arterial blood gases are monitored frequently and any acid base, electrolyte, blood glucose, and hematocrit disturbances are corrected accordingly. A blood gas is always sent before unclamping and any abnormalities corrected.

Central venous catheterization is performed after induction for venous access to administer fluids, blood and vasoactive medications, and to monitor filling pressures. In patients with normal ventricular function, CVP and pulmonary artery wedge pressures (PCWP) correlate and the use of PAC is not required. PACs may be indicated in patients with pre-existing left ventricular dysfunction, active ischemic heart disease, pulmonary disease with pulmonary hypertension, and renal insufficiency. PACs are especially useful in patients requiring a higher aortic cross-clamp and those with the potential for massive fluid losses. PAC can be used to detect ischemia but poor specificity makes it less useful than ECG and TEE. Ischemia manifests as increases in PCWP and ischemic mitral regurgitation produces large V waves in the pulmonary capillary waveform. PACs with capability to monitor mixed venous oxygen saturation and continuous cardiac output may be useful in patients with extensive SIRS, sepsis, and RAAA.

TEE is used in most of the open AAA repairs at our institution. TEE allows early recognition of evolving myocardial ischemia and facilitates immediate fluid and specific pharmacologic interventions to reduce perioperative cardiac complications. However, all RWMA are not ischemic and not all RWMA detected by TEE correlate with postoperative complications.⁵² TEE, the most sensitive monitor of myocardial ischemia, can also be used to evaluate volume status and valvular function. Cardiac output measured by TEE correlates very well with thermodilution cardiac output derived from PAC in the absence of significant mitral regurgitation.⁵³ Myocardial performance index (MPI), uses measurements taken during the cardiac cycle, from end-diastole to the

next end-diastole, is calculated using isovolumetric contraction time (IVCT), isovolumetric relaxation time (IVRT), and ejection time (ET). This index is believed to describe both systolic and diastolic performance of the myocardium. An elevated MPI has been shown to predict poor outcomes (congestive and ventilatory failure) in patients undergoing AAA repair.⁵⁴

Cardiac output can be measured from continuous analysis of pulse pressure waveform by the insertion of a catheter with manometer into axillary or brachial artery. These pulse contour cardiac output (PiCCO; Pulsion Medical Inc, East Brunswick, NJ, USA) devices can also measure transpulmonary thermodilution cardiac output, but require insertion of central venous catheter in addition to arterial catheter.⁵⁵ Echo-esophageal Doppler (Hemosonic 100; Arrow, Reading, PA), which measures cardiac output through mean blood velocity in the descending aorta and diameter of descending aorta, have been found to be useful in infrarenal abdominal aortic surgery.⁵⁶ These could be potential alternatives for PAC in these patients, although these devices need further evaluation and validation during different periods of aortic surgery such as clamping and unclamping.

Aortic Cross-Clamping

The effects of cross-clamping depend on the level of the clamp, the patient's baseline myocardial function and presence of CAD, the type of aortic disease, the degree of collateralization, volume status, and anesthetic technique.

While infrarenal clamping is well tolerated in most patients, suprarenal clamping produces more hemodynamic changes and is associated with an increased 30-day mortality after RAAA. This can be explained by the effect of the level of cross-clamp on blood volume redistribution and myocardial function as described in the chapter on anesthesia for open descending aortic surgery.^{57,58}

Left ventricular hypertrophy and diastolic dysfunction are not uncommon in AAA surgical patients. The impact of aortic cross-clamping on diastolic dysfunction and the resultant hemodynamic changes in patients with diastolic

dysfunction are not well studied. Infrarenal clamping is associated with a change in ventricular compliance and PCWP did not correlate with left ventricular end-diastolic area (LVEDA) during aortic cross-clamping.⁵⁹ Mahmood et al., using transmitral flow propagation velocity in 35 patients undergoing elective AAA surgery, reported an increase in diastolic dysfunction after aortic cross-clamping, which returned to baseline after unclamping. They did not report any hemodynamic parameters, so the impact of the diastolic dysfunction is not clear.⁶⁰ Meierhenrich et al. reported a 50% baseline incidence of diastolic dysfunction in patients undergoing AAA repair. Aortic cross-clamping was not associated with impairment of diastolic dysfunction and the hemodynamic changes were similar in patients with or without diastolic dysfunction.⁶¹ Shuetz et al. have shown that the treated hypertensive patients had significant increase in mean arterial pressure (MAP), PCWP, and mean peak A/peak E velocity after cross-clamping, compared to normotensive controls, but these disturbances normalized after unclamping and the intraoperative management did not differ in both groups.⁶² Fayed et al. reported acute diastolic dysfunction after high aortic clamping in six of nine patients with normal E/A ratios before clamping in thoracoabdominal surgery and three of those patients developed myocardial infarction.⁶³ The level of aortic cross clamp again has an influence on diastolic dysfunction and its effect on outcome.

Patients with CAD and a reduced LVEF experience more hemodynamic changes compared to patients without CAD and a normal ejection fraction.⁶⁴ Attita et al. reported a 30% incidence of myocardial ischemia after infrarenal cross-clamping in patients with known severe CAD.⁶⁵ Gooding et al. studied the effect of CAD on hemodynamic response to infrarenal aortic cross-clamping in 25 patients undergoing either aortofemoral bypass or abdominal aortic aneurysmectomy. Ten patients had evidence of CAD and 15 did not. They concluded that patients with preexisting CAD had significantly lower cardiac indices and increased PCWP, which may suggest that this subgroup is at greater risk for perioperative myocardial dysfunction.⁶⁶

Patients with aortic occlusive disease may experience less hemodynamic changes compared to AAA patients to clamping and unclamping. Johnston et al. compared hemodynamics in patients with aneurysmal and aorto-occlusive disease and found that patients in the aorto-occlusive group had an increased stroke volume and less of an increase in SVR during aortic cross-clamping compared to patients with aneurysmal disease.⁶⁷ These findings could be explained by persistent para-aortic collateral circulation to the lower extremities in chronic occlusive disease and minimal metabolite and lactate accumulation.^{68,69} This accounts for the stable hemodynamics that occurs during aortic cross-clamping in patients undergoing repair of aorto-occlusive disease.

The management of the hemodynamic changes associated with aortic cross-clamping center on decreasing afterload, normalizing preload, and maintaining coronary blood flow and contractility. Volume status should be monitored and optimized throughout the clamping period. TEE is very useful in discriminating between hypovolemia and myocardial dysfunction as the cause of hemodynamic compromise. Vasodilators and inotropes may be necessary in some patients. Blood pressure proximal to the clamp should be maintained at preoperative basal level so that the blood flow through collateral circulation to the viscera distal to the clamp is maintained.

Aortic Unclamping

The major hemodynamic response to aortic unclamping is hypotension. The etiology of this hypotension is multifactorial. During clamping of infrarenal aorta, the lower extremities undergo ischemic vasodilatation and vasomotor paralysis. Release of the clamp causes blood sequestration in the previously ischemic lower extremities, central hypovolemia, and decreased blood flow to the critical coronary, renal, and hepatic circulations (Fig. 14.7). The use of vasopressors to increase the blood pressure without restoring blood volume may further decrease blood flow to these critical organs. Adequate volume loading prior

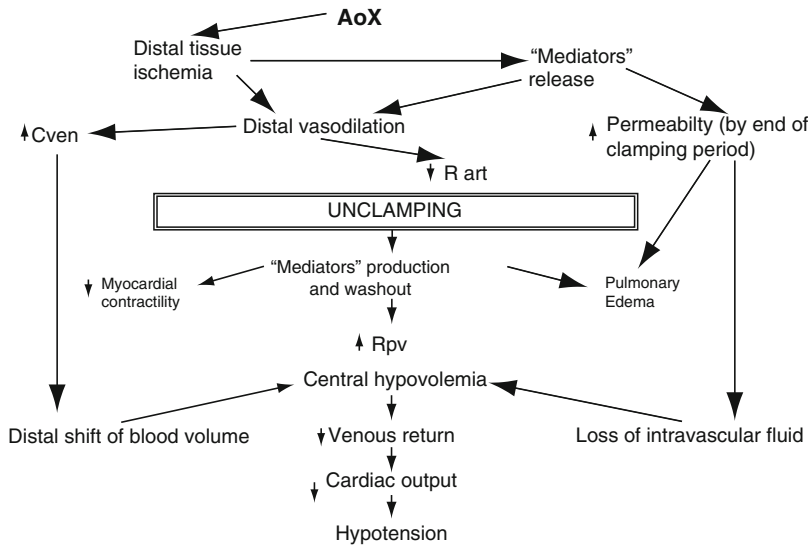


Fig. 14.7 Hemodynamic effects of aortic unclamping (Reprinted with permission from Gelman⁵⁸)

to release of the clamp to a higher PCWP than the baseline value is recommended.⁷⁰ Ueda et al.⁷¹ used volume loading with 10 ml/kg of albumin before unclamping during AAA repair and have shown that albumin did not prevent systemic hypotension, but significantly increased pulmonary artery pressure and right ventricular afterload causing RV dilatation. Cautious fluid administration is recommended in patients with cardiac dysfunction. Bitoh et al.⁷² reported left ventricular diastolic dysfunction, measured using the mitral annular velocity by tissue Doppler method, after unclamping in eight patients undergoing infrarenal AAA repair and the dysfunction persisted until the end of the surgery. Pulmonary edema occurred in two of their patients with the lowest tissue Doppler mitral annular velocity. Gradual release of the clamp is recommended to prevent adverse events after unclamping in such cases. Close communication with surgical team is essential.

Hypotension may also be the result of the accumulation and release of vasodilating and myocardial depressant metabolites. This response involves adenosine, inosine, hypoxanthine, lactate, oxygen free radicals, and prostaglandins, and the activation of neutrophils and the complement system (Fig. 14.7).⁷⁰ The correction of metabolic acidosis may be required but may not completely

correct arterial hypotension indicating persistent tissue acidosis. The degree and duration of hypotension depends on the level of the aortic clamp and duration of ischemia. Minimizing ischemic time is the goal to prevent severe hemodynamic disturbances. If hypotension is persistent or severe, the aorta may have to be reclamped.⁵⁰

Anesthesia for Ruptured Abdominal Aortic Aneurysms

The initial evaluation of these patients takes place in the operating room. Relevant history, comorbid conditions, details of resuscitation, and the results of laboratory tests and diagnostic imaging should be obtained from the patient, relatives, emergency medical service (EMS) providers, or primary care providers, such as the surgeon or the emergency medicine physician. A quick evaluation of the patient's hemodynamic status is desirable. This should occur quickly because the most important determinant of survival is time to aortic cross-clamping.

Usually, some intravenous access is already established in the emergency room or by EMS. If needed, additional large bore intravenous access should be obtained to allow for adequate fluid

resuscitation. Rapid infuser systems with warming capability should be set up and connected to large bore IVs for blood and fluid administration. Blood is also sent for cross-matching from the emergency room. Ten units of packed red cells, 10 units of fresh frozen plasma, and platelets are ordered and should be available by the time the patient reaches the operating room. Type-specific cross-matched blood is preferred. If cross-matching is not done, type-specific blood can be used. Upper extremity invasive arterial lines are placed while the patient is preoxygenated. In patients with hypovolemic shock, axillary or brachial arterial cannulation may be necessary. Ultrasound can be used to quickly cannulate the axillary artery. Fluid and blood administration should continue during induction to the target systolic blood pressure between 50 and 100 mmHg systolic in patients with severe hypovolemic shock.

Patients are induced only when the surgeon is prepared for incision because of the tamponade effect of tight abdominal muscles, which is lost with induction and can precipitate catastrophic hemorrhage. Typically, rapid sequence induction is done with etomidate and succinylcholine. All other medications, such as fentanyl, midazolam, and inhalational anesthetics, should be titrated very carefully in these patients. Scopolamine can be used to produce amnesia in hemodynamically unstable patients. Vasopressors and fluids should be administered cautiously before clamping, as aggressive resuscitation may unplug the hemostatic clot and cause bleeding. Several animal studies using an abdominal aortotomy model of ruptured AAA have demonstrated improved tissue perfusion, decreased blood loss, and improved survival associated with hypotensive resuscitation compared with aggressive resuscitation.⁷³ There are human studies advocating delayed rather than immediate resuscitation in trauma patients,^{74,75} but there are no prospective studies of hypotensive resuscitation in patients with ruptured AAAs. Minimal fluid resuscitation seems to be a logical option provided rapid surgical control of bleeding could be achieved. After induction, a PAC is inserted and TEE can be used along with the PAC to monitor volume status and contractility. Arterial blood gases

should be obtained to determine the presence of acidosis, electrolyte abnormalities, and hematocrit. A normal hematocrit may be indicative of hemoconcentration. If massive blood transfusion is required, a 1:1 mixture of packed red blood cells (PRBCs) and fresh frozen plasma (FFP) should be administered to prevent dilutional coagulopathy. Autotransfusion may also be useful. Platelets and cryoprecipitate should be available if multiple units of PRBCs and FFP are administered or if the patient is taking antiplatelet agents. Once the aneurysm is clamped, the goal is to maintain hemodynamics and to preserve cardiac function. Hypotension during maintenance is multifactorial and includes the loss of sympathetic tone, hypovolemia, and cardiac dysfunction. If the patient is hypotensive despite adequate fluid resuscitation, vasopressor agents, such as vasopressin or norepinephrine, may be required if the patient has normal cardiac function and inotropic agents, such as epinephrine or dopamine, may be required if the patient has decreased cardiac function. If massive transfusions have been administered, the patient may have citrate toxicity and calcium chloride should be administered. Unclamping shock can be profound and is related to the duration of clamping, degree of shock prior to clamping, the speed with which clamps are released, and the patient's volume status and cardiovascular reserve.

After the repair, the patient may be coagulopathic despite administration of FFP, cryoprecipitate, and platelets. Further transfusion should be guided by the use of thromboelastography (TEG) or coagulation studies such as prothrombin time, activated partial thromboplastin time, platelet count, and thrombin time. If the bleeding continues despite massive transfusion of blood products, activated Factor VII may be considered. Maintenance of normothermia is difficult and should be accomplished during and after the operation to prevent coagulopathy and arrhythmias.

Fluid and Transfusion Management

Open resection of AAA subjects the patient to a laparotomy incision, third space loss that follows

extensive tissue trauma, manipulation, exposure and retraction, systemic inflammatory response with increased microvascular permeability, major retroperitoneal edema, hemorrhage, ischemia-reperfusion of lower extremities, and hemodynamic effects of epidural anesthesia. Manipulation of the bowel can lead to the generation and release of vasoactive agents, including substance P, vasoactive intestinal peptide, prostacyclin, and nitric oxide, which can induce splanchnic ischemia and increased intestinal permeability. Fluid management should take all this into consideration. Typically, 2–4 L of intravenous crystalloids are infused for these procedures. Restrictive fluid strategy has been shown to reduce postoperative complications in abdominal surgery.^{76,77} McArdle et al.⁷⁷ in a retrospective study demonstrated that patients with positive fluid balance had an

increased incidence of adverse postoperative cardiac and respiratory complications and increased ICU and overall hospital stays after open AAA surgery (Fig. 14.8). Adesanya et al.⁷⁸ in another retrospective study on patients undergoing major vascular surgery have shown that fluid restriction less than 3 L decreased ICU stay and duration of mechanical ventilation and the benefits of fluid restriction were similar to those seen with major abdominal surgery.

A systematic review on the choice of fluid therapy indicates that there is no advantage of using any specific fluid over another in the management of aortic aneurysm surgery.⁷⁹ Lactated Ringer (LR) solution, normal saline, 5% dextrose, 5% dextrose in 0.45% saline, 5% dextrose in LR, mannitol, 1.8% saline, human albumin in LR, human albumin in water, 5% dextrose with human

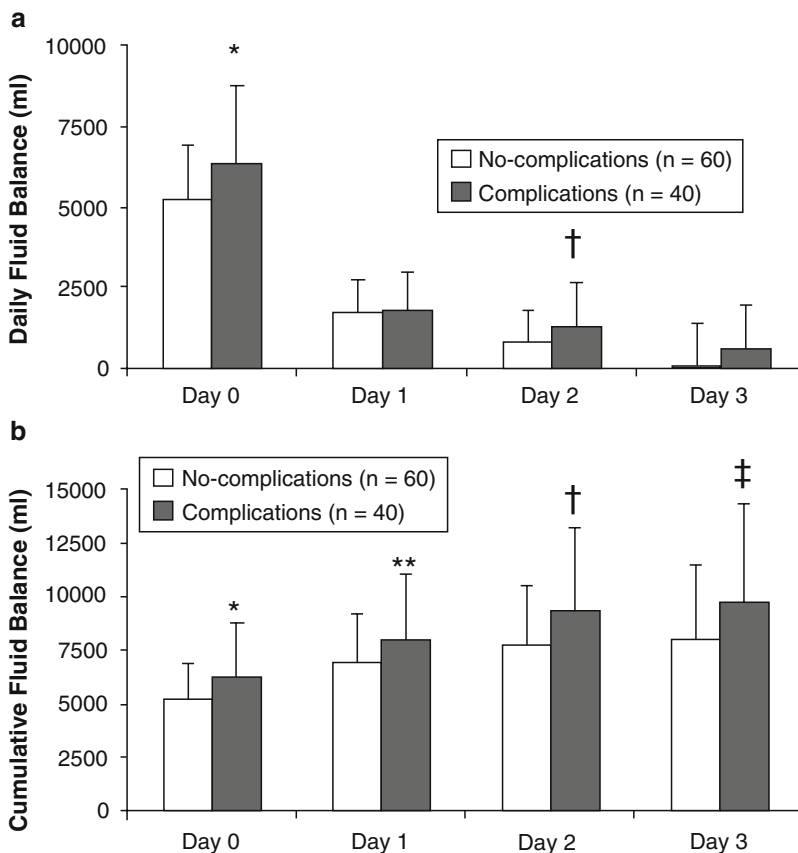


Fig. 14.8 Fluid therapy and outcome after abdominal aortic surgery (Reprinted with permission from McArdle⁷⁷)

albumin, Dextran, Isotonic hydroxyethyl starch (HES), and hypertonic HES (6% HES 7.2% saline) were some of the fluid types studied.

The blood loss during AAA surgery can occur from the lumbar arteries when aneurysm is opened or at the proximal and distal anastomotic site. Bleeding may be related to inadequate hemostasis, coagulopathy, or excess heparinization. Techniques to reduce homologous blood transfusion are employed to reduce the transfusion risks and prevent the adverse effects associated with extensive stored blood therapy. Preadmission autologous blood donation (PABD) in combination with intraoperative autotransfusion (IAT) has been shown to eliminate the need for homologous transfusion in two thirds of elective AAA patients.⁸⁰ Similar efficacy of PABD with IAT was demonstrated in aortic surgery when compared with IAT alone or with no conservation technique.^{81,82} Erythropoietin has also been used in patients undergoing preoperative blood donation.⁸³ The routine use of IAT in all elective AAA patients is controversial. A meta-analysis of four randomized controlled trials examining the efficacy of IAT reported 37% reduction in risk of allogenic blood transfusion.⁸⁴ Few other studies reported lack of efficacy and found this practice to be not cost-effective when applied to all AAA patients.^{85,86} While some surgeons use IAT routinely, few others consider it in selected operations where blood loss is likely to be high, such as RAAA, complex reconstructions, thoraco-abdominal aneurysms and suprarenal aneurysm surgery, or in patients with religious concerns.⁸⁷ The disadvantages of the washed red cells are that they do not contain platelets or coagulation factors. TEG is utilized intraoperatively to detect and treat coagulopathy. Acute normovolemic hemodilution (ANH) has been used in combination with intraoperative cell salvage (ICS) and found to be an effective strategy for AAA patients.⁸⁸ Wolowczyk et al.⁸⁹ in a randomized controlled clinical trial compared ANH with ICS to ICS alone in patients undergoing elective AAA repair and concluded that ANH did not reduce transfusion requirements and had no impact on systemic inflammatory response or overall outcome. Even without ANH, the large volumes of fluid

infused as part of standard fluid management in these patients produced hemodilution, which is the reason why additional ANH is ineffective in saving bank blood.⁹⁰

Pain Management

Intravenous opioid patient-controlled analgesia (PCA) with hydromorphone or morphine is the standard method of pain relief after abdominal aortic surgery to which other methods are compared. Compared to intravenous opioids, epidural analgesia provided superior pain control, reduced duration of tracheal intubation and mechanical ventilation by 20%, reduced the overall incidence of cardiovascular complications, acute respiratory failure, gastrointestinal complications, and renal insufficiency.⁹¹ Patients presenting for AAA resection have associated thromboembolic and cardiovascular disease and may take antiplatelet agents and anticoagulants. Medication and preoperative coagulation studies, such as the platelet count, bleeding time, prothrombin time, and partial thromboplastin time should be reviewed before insertion of epidural catheters. Lower thoracic epidural catheters are used in AAA surgery.

Combined epidural and general anesthesia may attenuate the increased systemic resistance with aortic cross-clamping and may produce stable cardiovascular dynamics following cross-clamp release if intravascular volume is maintained.⁹²⁻⁹⁴ The intraoperative use of epidural anesthesia in combination with general anesthesia is favored by some clinicians for these reasons while others elect not to use the epidural analgesia intraoperatively as they feel it causes more hypotension and necessitates increased infusion of intravenous fluids and vasopressors.⁹⁵ Intravenous fentanyl is used for intraoperative analgesia in such cases. Epidural analgesia with ropivacaine 0.1% or bupivacaine 0.125%, with hydromorphone 10 µg/cc is initiated before abdominal wound closure to facilitate extubation and pain relief in the immediate postoperative period. Initiation of epidural also controls the hypertensive response associated with extubation.

Typically, epidural analgesia is continued for 3–5 days. The superior analgesia by epidural is especially useful in patients with chronic obstructive pulmonary disease.⁹⁶ In these patients, epidural analgesia decreases the duration of mechanical ventilation and the incidence of pulmonary complications.⁹⁷ Epidural catheter insertion is not without risk. Epidural hematoma with paraparesis and iliopsoas hematoma with lumbosacral plexopathy have been reported with epidural analgesic use after AAA repair.^{98,99} Though these complications are rare when appropriate precautions are taken, the prognosis of these neurological complications can be serious. Postoperative monitoring of neurological dysfunction is an essential part of epidural analgesia. Anticoagulants should be discontinued and normal coagulation studies should be documented before the removal of epidural catheters.

Bilateral paravertebral blocks (PVB) offer an alternate effective method of pain relief with infrequent neurologic and hemodynamic effects. Paravertebral catheters are placed outside the central neuraxis making the possibility of central neuraxial hematoma unlikely. Compared to epidural analgesia, PVB was also associated with better pulmonary function, less hypotension, urinary retention, and postoperative nausea and vomiting (PONV) after thoracic surgical procedures.¹⁰⁰ There are no clinical trials comparing PVB and epidural analgesia in abdominal vascular surgery. Richardson et al.¹⁰¹ reported their experience of bilateral PVB as a part of balanced analgesia, with intravenous morphine and diclofenac, in eight patients undergoing abdominal vascular surgery. They noted satisfactory intraoperative hemodynamic stability and good quality of postoperative analgesia without any significant pain-related complications. Randomized trials comparing epidural and paravertebral analgesia are needed in abdominal aortic surgery.

Intrathecal opioids can also be utilized to provide effective postoperative analgesia. Low-dose (0.2 mg) intrathecal morphine decreased pain scores and IV morphine consumption compared to controls in patients undergoing abdominal aortic surgery.¹⁰² In another study using intrathecal sufentanil (1 µg/kg) with intrathecal morphine

(8 µg/kg), intense analgesia was produced in the first 24 h with intrathecal regimen compared to control group.¹⁰³ Improvement in analgesia was not associated with decrease in cardiovascular, respiratory, and renal complications. Intrathecal opioids can be used as an alternate method in selected patients in whom continuous epidural or PVB catheters cannot be utilized.

Wound infiltration of local anesthetics has not been shown to be effective in improving pulmonary function or decreasing opioid consumption after aortic surgery.¹⁰⁴ Other analgesic adjuvants that can be used include ketorolac, acetaminophen, and ketamine, especially in opioid-dependent patients.

Optimal pain management is important when the patient is considered a candidate for fast track AAA repair. Mukherjee et al. described a multidisciplinary technique involving anesthesiologists, surgeons, and nurses, which allows patients to be ambulatory and to eat by postoperative day 1, and to be discharged, on average, by postoperative day 3.6. “Fast-tracking” involves the use of a limited retroperitoneal incision, with minimal bowel manipulation, an anesthetic that allows extubation in the operating room and postoperative pain control with epidural anesthesia, and metoclopramide to facilitate gastric emptying.¹⁰⁵ By postoperative day 1, patients are encouraged to ambulate and are offered a clear liquid diet, which is advanced as tolerated. By the third postoperative day, all intravenous catheters are removed and oral analgesics are started. If tolerated, the patient is discharged by postoperative day 3 or 4. In a limited series of 30 patients, there was one death and one ureteral injury, which had similar morbidity and mortality to the standard procedure.

Blunt Aortic Trauma

Blunt injury of the abdominal aorta is rare and results from direct mechanical forces that compress the aorta against the lumbar spine. The injuries that result include contusions, intimal disruption, intramural hematoma, false aneurysm, and rupture.¹⁰⁶ Since aortic injuries tend to

progress from the intima to the adventitia, the most common injury from blunt aortic trauma is intimal tearing. This injury occurs predominantly in males and the majority are caused by automobile accidents including steering wheel and seat belt injuries. Early clinical signs include acute arterial insufficiency, acute abdomen, and neurologic manifestations, such as paraplegia and sensory loss and late clinical signs include claudication, an abdominal mass or bruit, and persistent abdominal pain. The diagnostic modality of choice is a helical CT with aortography for inconclusive CTs¹⁰⁷ and treatment depends on the injury and includes open or endovascular repair, flap suture of intimal tears, and thromboembolectomy.¹⁰⁸

Postoperative Management

The postoperative care of these patients is usually in the intensive care unit where the patient can be closely monitored. The management of these patients includes monitoring for and treating myocardial ischemia and ventricular dysfunction, treating hypertension by continuing preoperative antihypertensive regimens, including beta-blocking agents, optimizing pulmonary function, through the use of inhaled bronchodilators, incentive spirometry, and early extubation, providing analgesia either with regional or parenteral analgesia, and monitoring for bleeding, bowel, and renal dysfunction. Renal dysfunction is treated by avoiding nephrotoxins and maintaining adequate renal perfusion through the use of fluids and inotropic agents.

Postoperative Complications

Cardiac Complications

Postoperative myocardial infarction (MI) is the most common cause of death following AAA repair. Predisposing factors for myocardial ischemia include pre-existing coronary artery disease, pain, mobilization of third-space fluid into central circulation, stress and hypermetabolism, sympathetic overactivity, anemia, and hypothermia. Added to

the hemodynamic stress is hypofibrinolysis and thrombin activation leading to prothrombotic state and myocardial injury.^{109,110}

Identifying perioperative MI is difficult because chest pain is infrequent (fewer than 50% of ischemic episodes are symptomatic) and disguised by analgesics. ECG is the mainstay of the diagnosis, but the changes may be subtle and transient. Most of the postoperative MIs are of the non-Q wave variety. Moderate elevations of creatinine kinase myocardial band fraction can occur in aortic surgical patients. Cardiac troponin (TnI) elevation may be more specific. Elevated TnI has been demonstrated in 46% of the patients who survived RAAA repair and was associated with increased cardiac dysfunction and death.¹¹¹ Ali et al.¹¹² studied 43 patients undergoing elective AAA repair for evidence of myocardial injury. Elevated TnI was seen in 20 (47%) patients. Clinical evidence MI and myocardial events occurred in 11 patients. Survival free of cardiac events was seen in 55% of patients who had elevated CnI compared to 81% who did not have elevated CnI at 1.5 years after discharge. In doubtful cases of perioperative MI, echocardiography and nuclear scans may help in diagnosis.

Therapy of non-ST elevation MI includes oxygen, analgesics, nitrates, beta-blockers, angiotensin-converting enzyme inhibitors, and anticoagulation with aspirin and heparin, when approved by the surgeon. An increased bleeding risk precludes the use of thrombolytics in the immediate postoperative period. Cardiology consultation is initiated immediately for percutaneous interventions, such as cardiac catheterization, angioplasty, stenting, or surgery in patients who still have ischemia despite maximal medical therapy. Intervention is required in all cases of ST elevation MI. Treatment of cardiogenic shock require invasive monitoring, such as a PAC with continuous cardiac output and mixed venous saturation monitoring and inotropic support.

Pulmonary Complications

Various perioperative factors influence the development of respiratory failure after AAA repair.

Preoperative factors: Advanced age, cigarette smoking, history of COPD, morbid obesity, and

ASA IV status are risk factors for postoperative pulmonary complications. Abnormal pulmonary function tests (FEV1 or FVC < 70% predicted, Expiratory flow rate less than 200 ml/min) and arterial blood gas (PCO₂ > 45 mmHg on room air) also increase the incidence of postoperative pulmonary complications.

Intraoperative factors: Prolonged surgery, large volume infusion, large midline vertical incision, and transperitoneal approach are cited as risk factors for pulmonary complications.

Postoperative factors: Abdominal distension from ileus, inadequate analgesia, large doses of parenteral narcotic administration, hypothermia, bed rest in supine position all prolong weaning and early extubation

Respiratory failure is caused by a restrictive defect with decreased functional residual capacity, altered secretions, impaired cough and mucociliary clearance, bronchospasm, atelectasis, exacerbation of chronic lung disease, and pneumonia. Respiratory failure prolongs the hospital stay. Pneumonia is usually caused by *Pseudomonas aeruginosa* and *Staphylococcus aureus* and associated with mortality of 21%.¹¹³

Pulmonary complications can be minimized by tobacco cessation several weeks before the procedure, aggressive perioperative pulmonary toilet, lung expansion maneuvers, such as deep breathing exercises and incentive spirometry, antibiotics for pneumonia, and epidural or paravertebral analgesia, which allow for early extubation and mobilization.¹⁷

Renal Insufficiency

Pre-existing renal dysfunction with creatinine levels higher than 2 mg/dl is associated with operative mortality of 19% versus 4.2% mortality if the creatinine was less than 2 mg/dl.¹¹⁴ Patients with creatinine levels more than 4 mg/dl require preoperative initiation of hemodialysis.¹¹⁵ AAA may be associated with renal artery stenosis or ureteric obstruction, which should be evaluated preoperatively by renal ultrasound and excretory urography, respectively.¹¹⁶

The incidence of new onset of renal failure after infrarenal clamping is 3% and the rates for

suprarenal occlusion are five times greater. Transient renal insufficiency is more common and patients who develop renal insufficiency have longer periods of intubation, ICU, and hospital length of stay. Aortic cross-clamping increases renal vascular resistance and decreases renal cortical blood flow and increases renin-angiotensin secretion and decreases glomerular filtration rate (GFR). Suprarenal clamping decreases blood flow by 80% and infrarenal clamping decreases by 38%.^{117,118}

The incidence of renal failure following ruptured AAA repair ranges from 8 to 46% and is associated with a mortality of 57–97%.¹ Additional perioperative factors that contribute to the development of renal insufficiency include pre-existing cardiac disease, advanced age, contrast studies without proper hydration, suprarenal clamping, ischemic time > 30 min, hypovolemia (due to fasting, bowel preparation, and blood loss), hypotension, large volume transfusion, rhabdomyolysis, early reoperation, and atheromatous plaque embolization.

Urine output does not correlate with GFR and oliguria does not predict postoperative renal insufficiency.¹¹⁹ The incidence of postoperative renal insufficiency may be decreased by preventing hypovolemia, maintaining cardiac output, and by diagnosing and treating oliguria early.

Neurologic Complications

The two major neurologic complications include spinal cord ischemia and postoperative cognitive dysfunction. Benoit et al. studied the incidence of postoperative delirium following AAA repair and found that delirium occurs in up to one third of patients within the first 6 postoperative days. Risk factors for postoperative delirium include advanced age, being unmarried and living alone, preoperative depression and psychoactive medication use, a lower education, and a history of prolonged tobacco abuse.¹²⁰

Spinal cord ischemia (SCI) is a rare (0.3%) but potentially devastating complication after abdominal aortic surgery.¹²¹ SCI has been reported to occur in both elective repairs and ruptured AAAs.

Ruptured aneurysms produce significant hypotension, require supraceliac cross-clamping, and usually heparin is not used; all these factors predispose to SCI. SCI can occur with infrarenal and suprarenal aortic cross-clamps and with both surgery for AAA and aorto-iliac occlusive disease.¹²²

The low incidence of paraplegia after elective AAA surgery may be explained by the low level of clamping relative to the origin of arteria radicularis magna and the presence of adequate pelvic collaterals. In general, when the greater radicular artery is open and of normal size, the pelvic blood supply is of minor importance. When greater radicular artery is compromised, the pelvic blood supply becomes critically important. Interference of pelvic blood supply has to be avoided preventing significant hypotension, revascularization of internal iliac artery with a separate graft, and gentle surgical techniques to avoid embolization.²¹ SCI after infrarenal AAA and iliac surgery has a poor prognosis and may be reversed by spinal fluid drainage.

Gastrointestinal Complications

The most common complication is paralytic ileus, which is due to bowel manipulation and fluid sequestration. The return of GI function depends on the technique employed and the level of the clamp. GI function returns more rapidly with the retroperitoneal technique and infrarenal cross-clamping. Infrarenal cross-clamping produces little effect on splanchnic blood flow compared to suprarenal or supraceliac cross-clamping.

A rare (0.6–1%) but more devastating complication is ischemic colitis, which is associated with a higher mortality rate (50–75%). RAAA repair, ligation of a patent inferior mesenteric artery, marginal collateral circulation, and perioperative low flow state are risk factors. Inferior mesenteric stump pressure less than 40 mmHg and loss of mesenteric Doppler signals are predictors of the development of colonic ischemia. The risk is reduced by IMA revascularization and ensuring the circulation is preserved through at least one internal iliac artery.²¹

Signs and symptoms of ischemic colitis include excessive fluid requirements, abdominal

pain, fever, leukocytosis, diarrhea, and bloody stools. Nonsurgical treatment includes bowel rest, aggressive fluid resuscitation, and antibiotics. Surgical treatment is indicated for full thickness bowel necrosis.¹⁷

Hemorrhage

Hemorrhage is the second most common postoperative complication and is almost always the result of technical error. Continued hemorrhage increases transfusion requirements and mortality. Surgical causes include persistent retroperitoneal bleeding, anastomotic disruption, and injury to adjacent venous structures while nonsurgical causes include dilutional coagulopathy, disseminated intravenous coagulation, and preoperative anticoagulants, such as antiplatelet agents. If the administration of platelets and clotting factors fail to improve the bleeding or if the TEG and coagulation studies are normal, the patient may require a reoperation to search for a surgical bleeding. Patients undergoing repair of a ruptured AAA have a higher incidence of postoperative bleeding and hence re-explorations.

Disseminated Intravascular Coagulation (DIC)

Activation of fibrinolysis and consumptive coagulopathy can present in AAA patients preoperatively or develop during and after operation.^{123,124} Though DIC can occur in both ruptured and unruptured cases, clinically manifesting DIC is more common in RAAA.¹²⁵ Clinical presentation can range from bleeding to thrombotic complications and MOF. Heparin therapy may be needed preoperatively and if this is not effective, immediate surgical treatment is curative.^{126,127}

Limb Ischemia

Limb ischemia is usually associated with anastomotic complications or distal thromboembolism. Thromboembolism can be micro or macroembolization.¹²⁸ Placing the iliac clamp before aortic

cross-clamp minimizes distal embolization. Peripheral pulses are evaluated before leaving the operating room. Management includes thrombolytic, anticoagulant, and antiplatelet therapy. Macroembolism can be treated either surgically or by thrombolysis or amputation.¹²⁹

Key Notes

1. An abdominal aortic aneurysm is a focal dilation of the aorta, is defined as an aortic diameter of at least 50% larger than normal or a diameter of greater than 30 mm.
2. The majority of AAAs are infrarenal and are located several centimeters below the renal arteries. Pararenal AAAs are aneurysms that involve the renal ostia and include juxtarenal AAAs, which involve the renal artery origin and suprarenal AAAs, which involve the aorta above the renal arteries.
3. AAAs associated with mural thrombi have increased coagulation and fibrinolytic activity, produce local hypoxia in the media, which can lead to neovascularization and inflammation.
4. The majority of AAAs are asymptomatic and are discovered during routine screening. Ultrasound is the recommended screening modality CT or MRI are used to delineate the anatomy of the aneurysm, and aortography, the gold standard, is reserved for patients with inconclusive studies.
5. For ruptured AAAs, the triad of symptoms include sudden onset of mid-abdominal or flank pain radiating to the scrotum, circulatory shock, and a pulsatile abdominal mass.
6. Open AAA repair is recommended for the majority of pararenal aneurysms, aneurysms with an enlarged neck or significant angulation, and those with laminated thrombus at the stent-graft attachment. Open AAA repair does not require yearly surveillance as with endovascular techniques and is associated with improved patient quality of life as compared with endovascular repair.
7. The surgical approaches to open AAA include the transperitoneal and retroperitoneal approach, including total and assisted laparoscopic techniques. The retroperitoneal approach is associated with decreased pain and perioperative complications, and is the approach of choice for pararenal aneurysms.
8. SIRS, more common in patients with RAAAs, is associated with the release of endotoxin, which causes the release of inflammatory cytokines, such as IL-6, TNF α , and IL-6 β , leading to the development of ARDS and MOF.
9. Advanced age, cardiac arrest with cardiopulmonary resuscitation, hypotension, loss of consciousness, low hematocrit, and elevated creatinine are associated with a poor outcome.
10. Since up to half of patients with AAAs have underlying coronary artery disease and perioperative cardiac complications are the major cause of morbidity and mortality, these patients should be evaluated according to the 2007 ACC/AHA guidelines.
11. Monitoring for patients with uncomplicated AAAs includes standard ASA monitors, along with arterial blood pressure, central venous pressure, and urine output. For high-risk patients, additional monitoring may include pulmonary artery catheterization and transesophageal echocardiography.
12. The effects of aortic cross-clamping depends on the level of the clamp, the baseline myocardial function and presence of CAD, the type of aortic disease and degree of collateralization, volume status, and anesthetic technique.
13. Aortic unclamping produces hypotension and acidosis, which is due to the release of vasodilating and myocardial depressant metabolites from ischemia and reperfusion. This can be treated with vasopressors, fluid administration, and re-clamping, if necessary.
14. The most important determinant of survival in patients with RAAA is time to aortic cross-clamp. The goals of anesthetic management in these include performing a rapid evaluation, including hemodynamic status, obtaining large bore intravenous access, obtaining blood

and blood products for transfusion, and maintaining hemodynamic stability, with vasoactive agents

15. Fluid and transfusion management for these patients should include fluid restriction and blood conservation techniques, including cell salvage. Patients with coagulopathy during AAA repair should be monitored with TEG and treated with blood products and factor VIIa, if necessary.
16. Postoperative pain management in these patients is diverse and includes neuraxial analgesia with spinal opioids or epidural anesthesia, paravertebral analgesia, and intravenous patient-controlled analgesia.
17. Although the most common perioperative complication is cardiovascular, other complications during AAA repair include renal, pulmonary, neurological, gastrointestinal, hemorrhage, DIC, and limb ischemia.

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