

Anaesthesia for Surgery of the Thoracoabdominal Aorta



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Aneurysms involving the descending thoracic and abdominal aorta are referred to as thoracoabdominal aneurysms (TAAAs). The widely accepted definition of TAAAs is a dilatation of at least 50% the expected size of the aorta at the diaphragmatic hiatus. TAAAs are relatively rare and account for only 3% of all aortic aneurysms. The natural history of TAAAs left untreated is continuous expansion with high probability of rupture and death. Patients managed conservatively have a 5-year survival rate of less than 20%.

Aneurysmal dilatation of the aorta occurs commonly as a result of degeneration of the medial layer due to loss of smooth muscle cells and elastin fibres. Dilatation can also develop following an aortic dissection when the intima tears and the aortic wall weakens.

Congenital collagen vascular diseases such as Marfan's and Loeys-Dietz Syndromes are commonly associated with TAAAs. Acquired degeneration of the wall of the aorta occurs as a result of hypertension and atherosclerosis.

The Crawford classification scheme provides a standard framework for surgical planning in TAAAs procedures and facilitates standardized reporting of outcomes. The Crawford classification divides TAAAs aneurysm repairs according to the extent of aorta that needs replacing (Fig. 1):

- Extent I: From origin of left subclavian artery to above the renal arteries origin
- Extent II: From origin of left subclavian artery to aortic bifurcation distally
- Extent III: Replacement of distal half of the descending aorta. From mid descending aorta to aortic bifurcation
- Extent IV: From the diaphragmatic hiatus involving the abdominal aorta.

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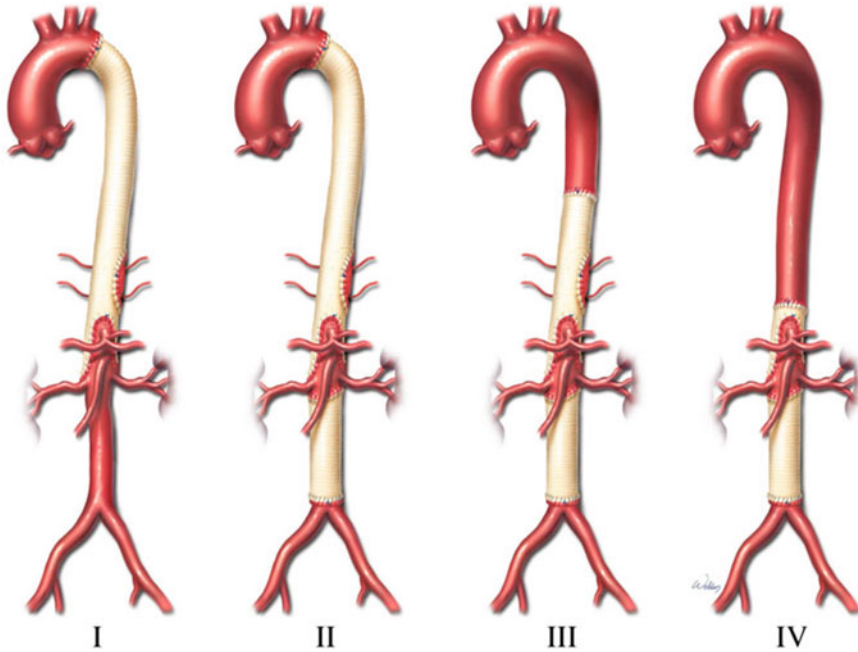


Fig. 1 The Crawford classification scheme for thoraco-abdominal aortic aneurysm repairs

Aortic dissections are classified using the classic Stanford or DeBakey classifications covered elsewhere in this book.

Aneurysms of the descending thoracic aorta tend to display exponential growth with a rate of 0.12 cm per year when they exceed a diameter of >5.2 cm. A diameter of 7.0 cm is regarded a critical for risk of rupture in descending aorta aneurysm. When managed conservatively, the survival rate is only 10–20% at 5 years. Indications for surgery include rupture, acute dissection, pain arising from the aneurysm or symptoms related to compression of adjacent structures, enlargement of ≥ 1 cm per year, or absolute size >6.5 cm or >6 cm in patients with connective tissue disorders.

Open surgical repair of TAAAs are lifesaving procedures but are associated with significant perioperative morbidity and mortality. Morbidity such as spinal cord injury, stroke and renal failure requiring dialysis can have a significant impact on long term survival and quality of life for patients. Initial high rates of morbidity and mortality have been significantly reduced by highly specialised multidisciplinary perioperative teams and management strategies that have evolved over the years.

Preoperative Assessment

The pre-anaesthetic assessment evaluates patient specific and surgical procedure related factors that influence the perioperative plan and the conduct of anaesthesia.

Patients presenting for TAAA aneurysm repair often have multiple co-morbidities common to patients with cardiovascular disease that can affect their tolerance to withstanding major aortovascular surgery.

Assessment of cardiovascular fitness includes screening with coronary artery disease with CT coronary angiogram or traditional angiography in patients with risk factors for coronary artery disease. Echocardiography evaluates the function of both ventricles and screens for valvular abnormalities that may affect the conduct of left heart bypass such as aortic insufficiency.

Lung function tests such as dynamic spirometry and transfer factor evaluate tolerance for prolonged one lung ventilation. We believe that functional tests such as the six-minute walk test provide a better indication of cardiovascular reserve and tolerance. Patients with borderline respiratory function are counselled about the possibility of postoperative pulmonary complications and the risk prolonged respiratory wean in the ICU.

Lung isolation with double lumen tubes (DLT) can be challenging as large aneurysms can compress the lower airway with significant deviation of the trachea. This issue can be identified and planned for with careful evaluation of the preoperative CT scan of the Thorax.

Pre-existing renal dysfunction with reduced eGFR is a risk factor for requiring postoperative dialysis in patients undergoing major aortovascular surgery. Preoperative hydration is crucial, and patients are encouraged to drink clear fluids up to 2 hours before surgery.

Anaemia is associated with increased bleeding and transfusion requirements. Whenever possible patients with anaemia (Hb <130 g/l) are screened for true or functional iron deficiency anaemia and optimised with oral or intravenous iron pre-operatively.

The surgical approach or timing may need to be modified according to the patient functional status and comorbidities. For instance, a patient with very limited respiratory reserve may be assessed for suitability of an endovascular approach rather than an open repair.

Surgical Approach and Positioning

Surgical access for TAAA aneurysm repair is usually obtained through a left thoraco-phreno-laparotomy. The patient is positioned in a left semi lateral position with the use of a deflatable vacuum bag. The right side of the torso is placed in a 60° lateral position with the hip and legs in a semi supine position at 30°.

The thoracotomy incision starts below the scapulae laterally and extends medially in to the abdomen often below the umbilicus.

Aortic clamping sites and sequence will depend on the anatomy and extent of the aneurysm. Partial left heart bypass (PLHB) is desirable in terms of bleeding, coagulopathy and inflammatory response when compared with full cardiopulmonary bypass (CPB). However, PLHB requires a safe clamping area to be present at or beyond the level of the left subclavian artery otherwise full CPB is required. Aneurysms involving the distal aortic arch often require a two-stage approach consisting of an elephant trunk procedure first followed by a replacement of the thoracic descending aorta at a different time.

Physiology of Cross Clamping and Left Heart Bypass

The descending thoracic aorta is cross clamped at different stages of the procedure. Sequential cross-clamping helps to protect the spinal cord. Cross-clamping causes marked physiological changes involving different organ and body systems. It is essential for the anaesthesiologist to recognise and pre-empt the effects of cross clamping. The physiological effects of cross clamping are summarized in Table 1.

Left heart bypass is used to ameliorate the changes in cardiac afterload caused by proximal aortic cross-clamping. Left heart bypass also provides distal perfusion, reduces lower body ischaemia–reperfusion injury and decreases the occurrence of spinal cord injury. A common site of cannulation is the left inferior pulmonary vein (LIPV) that drains oxygenated blood from the left atrium. Blood is then reinfused

Table 1 Physiological effects of aortic cross-clamping. MAP, Mean Arterial Pressure; MPAP, Mean Pulmonary Artery Pressure; PVR, Pulmonary Vascular Resistance; CSF, Cerebrospinal fluid; SCPP, Spinal Cord Perfusion Pressure

System	Effects
Cardiovascular	<ul style="list-style-type: none"> • Increased afterload and MAP • Decrease in Cardiac Output • Increased myocardial oxygen consumption • Supraceliac clamping increases preload proximal to clamp • Infracoeliac clamping causes changes in preload depending on the splanchnic vascular tone
Respiratory	<ul style="list-style-type: none"> • Increase in MPAP and PVR • Increase in pulmonary blood flow with supraceliac clamping
Gastrointestinal	<ul style="list-style-type: none"> • Ischaemia–reperfusion insult • Bacterial translocation • Release of pro-inflammatory cytokines TN, IL-6, IL-8 and IL-10
Renal	<ul style="list-style-type: none"> • Decrease in renal blood flow. Suprarenal > infrarenal cross clamp • Increase in renal vascular resistance
Spinal Cord	<ul style="list-style-type: none"> • Increase in CSF pressure proximal to clamp • Decrease in SCPP distal to clamp

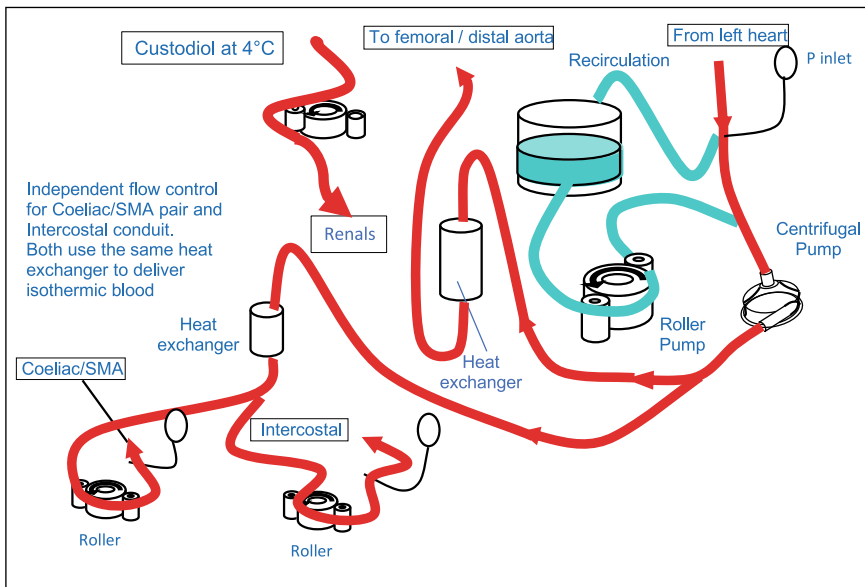


Fig. 2 Left Heart bypass circuit diagram. Drainage from left inferior pulmonary vein and reinfusion by centrifugal pump into the distal aorta/left femoral artery. Balloon-tipped perfusion catheters (10 and 13F) are used for selective perfusion of visceral vessels and the kidneys. A recirculation reservoir (Blue) is used in the rapid transfusion device. SMA, Superior mesenteric artery. (Diagram courtesy of Professor Aung Oo and the Aortovascular Team at St Bartholomew’s hospital, London)

using a centrifugal pump into the distal aorta or left femoral artery. LHB circuits typically do not require an oxygenator, therefore only moderate heparinization is required (1 mg/kg) with an ACT target of 250–300 s. Temperature is allowed to drift to 32–34 °C. Mean distal perfusion pressure (MAP) is maintained at 60 mmHg or above with flows usually oscillating between 1.5–2.5 l/min. MAP above the proximal aortic cross clamp needs to be carefully controlled and maintained at 80–90 mmHg. Excessive proximal hypertension increases cardiac afterload and may also result in proximal aortic dissection. On the other hand, excessively low proximal aortic pressures can impair coronary perfusion, lead to spinal cord injury and other organ injury. Balloon-tipped perfusion catheters are used for selective perfusion of visceral vessels using roller pumps from the bypass machine. Isothermic blood is used for visceral vessels and intercostal conduits and Custodiol solution at 4 °C is used for renal perfusion Fig. 2.

Initiation of LHB requires careful coordination and communication between the surgeon, anaesthetist and perfusionist. Proximal hypertension caused by the initial proximal clamp can be mitigated by prompt initiation of LHB. As blood is drained from the LIPV, left ventricle preload is reduced, and proximal aortic pressure reduced. An increase in distal aortic pressure and flow is associated with a decrease in left ventricle preload and proximal aortic pressure.

Once the aorta is opened there can be significant retrograde bleeding from aortic branches. Cell salvage and a rapid infusion devices (Belmont Medical Technologies) capable of fast rates of infusion (up 750–1000 ml/min) are required to return circulation volume to the patient and maintain adequate preload for both the proximal aorta supplied by the left ventricle and the distal aorta supplied by the pump of the LHB machine. Very fast blood loss is returned directly to the Belmont device with slower blood loss being processed through the cell saver devices. Surges in proximal blood pressure may require the use of short acting vasodilatory agents such as glyceryl trinitrate.

Cross clamps need to be repositioned at different stages of the operation which again requires close communication between surgeon, anaesthetists and perfusionists. Removal of the cross clamp can cause precipitous drops in proximal aortic pressure due to the sudden loss of afterload. The fall in pressure can be reduced by gradual release of the cross clamp and use of short acting vasopressor agents may be required i.e. metaraminol or phenylephrine.

Protection of Right Ventricular function is essential during left heart bypass to ensure adequate LV preload. Therefore, situations that cause elevation of pulmonary vascular resistance such as hypoxia or hypercarbia should be avoided. Bradycardia can be problematic as it can interfere with the performance of the right ventricle when faced with rapid infusion of fluids. We use a low dose adrenaline infusion (0.01–0.05 mcg/kg/min) to aid right ventricular performance.

A citrate-dextrose solution (ACD) is used by the cell saver as an anticoagulant exerting its effect by chelating calcium. Therefore, ionised calcium needs to be frequently monitored in arterial blood gas analysis and supplemented during left heart bypass otherwise life-threatening hypocalcaemia may ensue.

LHB is only possible if a safe proximal clamping site distal to the left subclavian artery is identified. Aneurysm involving the aortic arch can preclude safe proximal aortic clamping and surgery needs to be performed using full CPB and Deep Hypothermic Arrest (DHCA) which is associated with significantly higher blood loss and coagulopathy.

Spinal Cord Protection and Neuromonitoring

The spinal cord blood supply depends on an anterior spinal artery that irrigates the motor areas of the spinal cord and originates in the vertebral arteries, and two posterior spinal arteries that originate from the subclavian artery and supply sensory areas of the spinal cord. The anterior spinal artery is crucially supplemented by segmental arteries in the thoracic and lumbar aorta. The artery of Adamkiewicz is a large intercostal artery in the lower thoracic aorta and is a major tributary of the anterior spinal artery. However, recent understanding of the anatomy and physiology of spinal cord supply points to a complex network of segmental intercostal and lumbar arteries rather than a single artery, as responsible for maintaining spinal cord perfusion. These intercostal and lumbar arteries are interconnected both

longitudinally and transversely with arterial networks consisting of the anterior spinal artery, epidural arcades, paraspinous muscles and tissues.

Spinal cord deficit (SCD) is a feared complication of open TAAA repairs. Extent II repairs carry the highest risk of SCD with large case series reporting an incidence of 13.6% with 7.7% of the cases being permanent paraplegia.

Spinal cord perfusion is the product of the mean arterial blood pressure (MAP) minus the cerebrospinal fluid pressure (CSFP).

$$SCPP = MAP - CSFP$$

Aortic cross clamping results in reduced arterial blood flow and marked increase in central venous pressure that in turn causes spinal cord oedema and increase in CSFP. If the drop in SCPP is prolonged enough, irreversible damage to neuronal bodies and spinal nerve tracts will occur. Postoperatively, Spinal Cord Injury occurs due to a permanent reduction of blood supply when critical segmental arteries are interrupted by the surgical repair.

Risk factors for spinal cord injury include total extent of the repair with Extent II carrying the highest risk, previous infrarenal aortic repair, compromised left sub-clavian or internal iliac patency, aortic dissection with visceral malperfusion, emergency repair, prolonged duration of aortic cross clamping, renal disease, type II DM and age > 60 years.

Strategies to prevent Spinal Cord Injury can be divided into pre-operative, Intraoperative and Postoperative Table 2.

Table 2 Perioperative strategies for spinal cord protection in open TAAA repair. CSF, cerebrospinal fluid; MAP, Mean Arterial Pressure; MEP, Motor Evoked Potentials; NIRS, Near Infrared Spectroscopy

Preoperative	<ul style="list-style-type: none"> • Staged approach to repair if possible, combined endovascular repairs • Segmental artery staged coil embolization
Intraoperative	<ul style="list-style-type: none"> • CSF drainage pressure <10 mmHg. Max 20 ml/h • MAP >85/90 mmHg SBP >130 mmHg • Hb >100 g/dl • Hypothermia 32–34C • MEP monitoring • Paraspinal NIRS
Postoperative	<ul style="list-style-type: none"> • CSF pressure <10 mmHg. Max 20 ml/h • MAP >85/90 mmHg SBP >130 mmHg • Early sedation hold and hourly neurological assessment • Hb >100 g/dl

CSF Drainage

CSF drainage and maintenance of CSFP significantly reduces the incidence of paraplegia, especially in the higher risk procedures such as Extent I, II and III repairs. We do not routinely insert CSF drainage for Extent IV repairs. In our institution the drain is inserted following induction of anaesthesia using a midline approach. The patient is placed in a lateral decubitus position with the legs flexed towards the head. The L3–L4 and L4–L5 spaces are the most commonly used to avoid the risk of direct spinal cord injury with higher insertion levels. We use an Integra LifeSciences (Princeton, NJ) lumbar drainage kit containing a Tuohy needle and an 80 cm 5Fr catheter with guidewire. The guidewire is lubricated with saline and pre-loaded into the catheter before insertion. The Tuohy needle is inserted with the bevel facing cephalad and with a 20 degree cephalad angulation. Once free flow of CSF is identified the catheter is inserted to a depth of 8–10 cm past the tip of the Touhy needle. A bloody tap, that does not clear, may lead to postponement of the procedure as there is a risk of intrathecal bleeding or haematoma with heparinisation. Following insertion, the spinal drain is attached to an automated pressure monitoring and drainage system (LiquoGuard; Möller Medical, Fulda, Germany). The LiquoGuard system is capable of volume- or pressure-controlled drainage. We set the system to drain if pressure exceeds 10 mmHg with a maximum drainage of 20 ml/h as excessive drainage may lead to a subdural bleed. Complications of spinal drainage include epidural/subdural haematoma, persistent CSF leak and low CSF pressure headache, subarachnoid haemorrhage, meningitis and puncture site bleeding Fig. 3.

Neurological Monitoring

The integrity of the corticospinal motor pathways can be monitored using motor evoked potentials (MEPs). Measurement of MEPs provides a reliable measure of spinal cord function and can guide interventions to improve spinal cord perfusion such as reimplantation of segmental arteries or augmentation of SCPP. MEP monitoring requires expert staff trained in neurophysiological monitoring and institutional experience. The anaesthesia technique needs to be modified as neuromuscular blockade and inhalational agents affect the amplitude of the MEP signals.

The blood supply to the paraspinous muscles correlates closely with that of the corresponding level of the spinal cord as they are part of a collateral network. This association is utilized by NIRS monitoring of the oxygen saturation in the paraspinous muscles that serve as real-time non-invasive monitoring of spinal cord perfusion. Evidence from small pilot studies suggest that changes in paraspinous NIRS correlates well with MEPs in detecting spinal cord ischaemia.

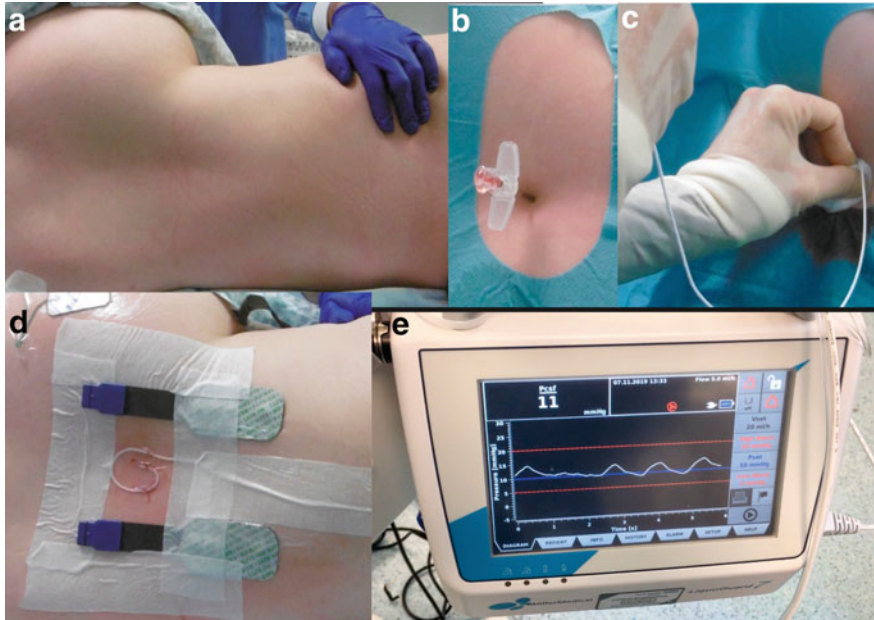


Fig. 3 Insertion of spinal drain catheter for (CSF) drainage. A, Patient is positioned in the right lateral decubitus position with hips and knees flexed. B, L3–L4 or L4–L5 space punctured with a 14 g Tuohy needle and free flowing CSF obtained. C, A 5f silicone lumbar catheter is inserted into the subarachnoid space. D, Catheter is secured with sutures and adhesive dressings. Near Infrared Spectroscopy (NIRS) sensors are placed at the paraspinal level. E, Drain is connected to automated drainage device (LiquoGuard; Möller Medical, Fulda, Germany) and the system set to drain when pressure exceeds 10 mmHg with a maximum drainage of 20 ml/h. Courtesy of Professor Aung Oo and the Aortovascular Team, St Bartholomew’s Hospital, London

Conduct of Anaesthesia

The anaesthesia technique for open TAAA repairs includes very particular considerations that are determined by the anatomical and physiological consequences of the TAAA, complex surgical aspects and the use of specialist neuromonitoring adjuncts. The use of MEP requires significant modification of the anaesthetic plan with avoidance of neuromuscular blockade and use of intravenous agents for maintenance of anaesthesia. The anaesthetist and neurophysiologist need to communicate closely to ensure that anaesthetic agents don’t affect the MEP signals. Depth of anaesthesia monitoring is required to avoid deep planes of anaesthesia that may dampen MEP signals.

We administer methylprednisolone 10–15 mg/kg intravenously following induction of anaesthesia and prior to the initiation of left heart bypass. Evidence suggests that steroids lower risk of respiratory failure and reduce length of ICU and hospital stay in patients undergoing complex cardiac surgery.

Particulars of the anaesthesia techniques for TAAA are outlined in Table 3.

Table 3 Considerations for anaesthetic management of open thoracoabdominal aneurysm repair (TAAA). DLT, Double lumen tube; TIVA, Total intravenous anaesthesia; MEP, Motor Evoked Potentials; PAC; Pulmonary artery catheter; CSF, Cerebrospinal fluid; NIRS, Near Infrared Spectroscopy; TEE, Transesophageal echocardiogram; PEEP; Positive End Expiratory Pressure

Airway	<ul style="list-style-type: none"> • Lung isolation with DLT. Isolation can be challenging when the TAAA causes tracheal deviation or bronchial compression • Bronchoscopic guidance is essential to ensure adequacy of isolation • Exchange to single lumen tube at the end of procedure if safe to do so
Induction	<ul style="list-style-type: none"> • Avoid surges in blood pressure • Use intermediate acting Neuromuscular blockers i.e. Vecuronium, Rocuronium
Maintenance	<ul style="list-style-type: none"> • Avoid Neuromuscular blockers • TIVA based technique with Propofol 3–6 mg/kg/h or TCI 3–5 mcg/ml • Use of depth of anaesthesia monitor • Fentanyl 10–15 mcg/kg • Inhaled agents with a MAC not higher than 0.5 as it affect MEPs
Invasive lines	<ul style="list-style-type: none"> • Right radial artery catheter to monitor proximal aortic ‘brain’ perfusion pressure • Right Femoral artery catheter to monitor distal aortic pressure • Ultrasound guided insertion of lines as aneurysm can compress mediastinal structures affecting calibre of great veins • Left internal jugular 5 lm central line and 7 French PAC introducer sheath • Right Femoral Vein dialysis catheter (Vascath) attached to rapid infuser
Monitoring	<ul style="list-style-type: none"> • Nasopharyngeal and bladder temperature monitors • Cerebral and Paraspinal NIRS • TEE • PA catheter If > mild RV or LV dysfunction • MEP • CSF pressure monitoring. Maintain CSF <10 mmHg
Respiratory	<ul style="list-style-type: none"> • Lung protective ventilation VT <6 ml/kg, patient-specific PEEP • Frequent suctioning and protection of dependant lung (right) from soiling with blood from left lung • CPAP on non-ventilated lung may be required
Circulation	<ul style="list-style-type: none"> • Prepare infusion and bolus of vasodilator i.e. Glyceryl Trinitrate to lessen hypertensive response to cross clamping • Noradrenaline infusion may be necessary to augment MAP and SCPP • Low dose adrenaline infusion (0.01–0.05 mcg/kg/min) to support RV function and heart rate during LHB • Ionised calcium monitoring and replacement • Rapid infuser (Belmont) capable of high infusion rates (750 ml–1000/min)

Transesophageal Echocardiography

Transoesophageal echocardiography (TEE) is a crucial diagnostic and monitoring tool during open TAAA repairs. A comprehensive and systematic assessment of the thoracic aorta from the aortic root to the distal descending aorta is required using standard imaging planes. The dimensions of the aortic root (i.e. aortic valve

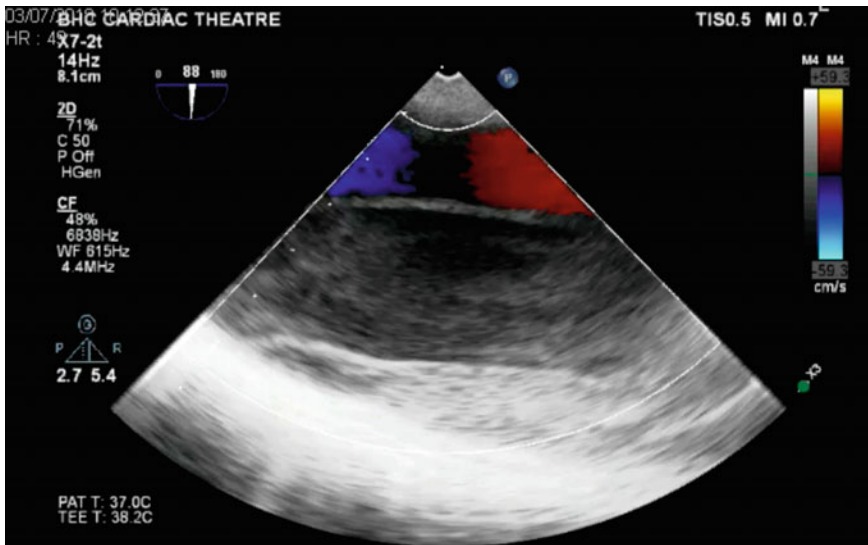


Fig. 4 Descending Thoracic Aorta TOE long axis view demonstrating an aortic aneurysm with chronic type B dissection. Colour doppler examination reveals flow in the smaller true lumen with a larger false lumen with 'smoke' due to stagnant flow

annulus, sinuses of Valsalva, sinotubular junction), ascending aorta, aortic arch and descending aorta should be recorded and taken in diastole from leading edge to leading edge. The maximum diameter and extension of aneurysmal dilatation should also be noted. In the case of dissections, efforts should be made to identify the origin and extension of dissection flap (Fig. 4). Identification of the true lumen is especially important and helps the surgeon to select a safe place for distal cannulation in the descending aorta or femoral artery for LHB. Intraoperative epi-aortic ultrasound can also be used to guide instrumentation of the descending aorta.

Aortic insufficiency should be ruled out and if present, quantified using international guidelines parameters. Anything other than mild aortic insufficiency may lead to left ventricular distension during aortic cross clamping. Ventricular distension can be prevented by initiation of left heart bypass prior to cross clamping and careful management of afterload with intravenous vasodilators. Key aspects of TEE evaluation of TAAA are outlined in Table 4.

Blood Conservation, Coagulation and Haemostasis

Open repair of TAAA causes significant blood loss, coagulopathy and transfusion requirements. Left heart bypass is preferable in terms of coagulopathy and blood loss as lower doses of heparin are required when compared with full cardiopulmonary bypass. However, extensive procedures in LHB can still lead to significant

Table 4 Key points for TEE imaging during open TAAA aneurysm repair. TAPSE, Tricuspid Annular Plane Systolic Excursion; FAC, Fractional Area Change; DTA, Descending Thoracic Aorta

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- Right ventricular function: Quantification of Longitudinal (TAPSE) and radial (FAC) function
 - Quantification of Left Ventricular function (EF)
 - Assessment of volume status
 - Aortic valve morphology and presence of regurgitation
 - >mild aortic regurgitation
 - Size of aortic root, ascending aorta, arch and DTA
 - Dissection flaps and differentiation of true versus false lumen
-

derangements in coagulation homeostasis. The prolonged contact of blood with the extracorporeal circuit leads to activation of coagulation, excessive fibrinolysis, consumption of clotting factors and platelets. Depletion of fibrinogen and platelets can be especially problematic.

We use multiple blood conservation strategies and point of care monitoring to guide management of coagulopathy. The use of cell salvage is routine and essential for open TAAA repairs. We use at least two cell salvage devices.

The use of autologous platelet-rich plasma (PRP) is associated with reduced allogenic blood product utilization and decreased postoperative morbidity in major aortovascular surgery. Our practice is to collect approximately 15 ml/kg of whole blood in a normovolaemic manner following induction of anaesthesia by withdrawing blood from large bore IV access lines inserted for the procedure. The blood is then processed by an autologous transfusion system yielding PRP and red cells. The PRP is given to the patient following administration of Protamine.

Coagulation status is monitored using point of care thromboelastography (TEG 6S global coagulation cartridge, Haemonetics) allowing assessment of all determinants of coagulation. Excessive bleeding associated with coagulation defects may be treated with blood products such as FFP, Cryoprecipitates, platelets or factor concentrates. An algorithm is used to facilitate interpretation and guide therapy according to thromboelastography parameters. Factor concentrates such as prothrombin complex concentrate and fibrinogen may have some advantages when compared with blood products e.g. lower volume of administration, no requirement for thawing and more rapid correction of coagulation abnormalities.

Antifibrinolytics are routinely used with Aprotinin being commonly used in our centre. Tranexamic Acid and aminocaproic acid are also effective at reducing blood loss. The use of cell salvage is essential for blood conservation.

Pain Management

Patients encounter significant acute and chronic pain issues due to the extensive nature of the thoracolaparotomy incision. Gabapentinoids are administered as premedication on the morning of surgery (Pregabalin 150 mg) and continued

postoperatively (Pregabalin 75 mg bd) until patients are reviewed in a clinic following discharge. Gabapentinoids have opiate sparing effects and reduce acute and chronic pain after cardiac surgery.

The ideal pain management strategy would facilitate adequate respiratory effort, good cough and secretion clearance with minimal sedation. Acute postoperative pain requires a multimodal analgesia approach with intrathecal diamorphine supplemented by fentanyl administered using a patient-controlled analgesia (PCA) device, dexmedetomidine infusion and intravenous paracetamol. Epidural analgesia may cause significant motor or sensory blocks that will cloud neurological monitoring of the spinal cord. Therefore, we do not use it in our centre. Emerging pain management strategies include novel regional anaesthesia techniques such as the Erector Spine Plane block (ESP) and the use of cryoablation of intercostal nerves.

Early input from a specialist pain team is essential not only for early acute pain management but for follow up of chronic pain issues that are very frequent in this patient population.

Post-operative Management

The composite reported incidence of common complications following open TAAA repairs including paraplegia, paraparesis and renal failure requiring dialysis is 14.4% with Extent 2 TAAA repairs carrying the highest rate of complications. Perioperative mortality ranges between 8–10%. The last two decades have seen major advances in the prevention and treatment of complications arising from open thoracoabdominal aneurysm repair. Crucial to reduction of complications is the seamless continuation of multidisciplinary team treatment and monitoring from the operating theatre into the critical care unit.

Permanent paraplegia is a devastating consequence hence prevention of spinal cord injury is a critical aspect of postoperative care. Therapy is tailored to ensure adequate perfusion and oxygen delivery to the spinal cord. Frequent neurological assessment (hourly whilst CSF drain is in situ) of power and sensation in lower limbs is essential and requires early weaning of sedation postoperatively. If sedation weaning is not possible, monitoring of MEPs is continued in the ICU. Spinal cord deficit can occur intraoperatively, in the immediate postoperative period or be delayed for several days after the procedure. Delayed spinal cord injury has a better prognosis but depends on early detection and intervention.

Detection of neurological impairment triggers the initiation of the COPS protocol. Elements of the COPS protocol include:

- **C: cerebrospinal fluid management:** ensure patency of drain or insert one if not in situ, set CSF pressure ≤ 10 mmHg, drain in place for at least 7 days
- **O: adequate oxygen delivery:** O_2 saturations $>92\%$, Haemoglobin >100 g/l, ensure adequate cardiac index >2.5 l/min/body surface area

- **P: pressure management for perfusion of spinal cord:** MAP >90 mmHg, SCPP >80 mmHg, adequate right sided pressures
- **S: patient monitoring status:** half hourly neurological observations, CT head and spine, MRI spine if feasible.

The extensive nature of the open TAAA repair impacts the homeostasis of practically all of the body’s organ systems. Postoperative pulmonary complications are very common following major aortovascular surgery. Mechanisms for lung injury include direct trauma to the lung due to surgical retraction, ischaemia/reperfusion injury, circulatory overload and massive transfusion. Other body systems, potential postoperative issues and suggested interventions are summarised in Table 5.

Table 5 Potential postoperative issues and complications following TAAA repair classified according to body systems, targets and interventions. DLT, Double Lumen Tube; RASS, Richmond Agitation-Sedation scale; CAM-ICU, Confusion Assessment Method ICU; MAP, Mean Arterial Pressure; CSF, Cerebrospinal Fluid; ARDS, Adult Respiratory Distress Syndrome; POC, Point of Care

System	Potential issues	Targets and interventions
Airway	<ul style="list-style-type: none"> • Airway oedema precluding exchange of DLT • Vocal cord palsy due to recurrent laryngeal nerve injury 	<ul style="list-style-type: none"> • Head elevation to reduce oedema • Speech and Language therapy screening and follow up
CNS	<ul style="list-style-type: none"> • Stroke (incidence 2%) • Delirium • Subdural Haematoma 	<ul style="list-style-type: none"> • Early sedation holds postoperatively • Target RASS score 0 to -1. We use dexmedetomidine 0.4–1.4 µg/kg/h • CAM ICU screening
Spinal cord	<ul style="list-style-type: none"> • Prevention of spinal cord injury 	<ul style="list-style-type: none"> • Target MAP >85/90 mmHg. Volume resuscitation and vasopressors i.e. Noradrenaline ± Vasopressin usually required • CSF pressure ≤ 10 mmHg • CSF drainage for 72 hours • Hourly neurological observations
Cardiovascular	<ul style="list-style-type: none"> • Haemodynamic instability • Right Ventricular dysfunction • Atrial fibrillation 	<ul style="list-style-type: none"> • Adequate filling pressures CVP 8–12 mmHg requiring fluid resuscitation • Positive fluid balance often >5 L in the first 48 hours • Cardiac Index ≥ 2.2 L/min. Inotropes (Adrenaline or Milrinone) may be required. Caution with excessive vasodilatation with Milrinone • Restoration of sinus rhythm (Amiodarone or cardioversion)

(continued)

Table 5 (continued)

System	Potential issues	Targets and interventions
Respiratory	<ul style="list-style-type: none"> • ARDS • Lung contusion • Pulmonary Haemorrhage • Protracted Weaning 	<ul style="list-style-type: none"> • Lung Protective Ventilation • Ventilator care bundle • Early Respiratory Therapy involvement • Non-Invasive ventilation/High flow oxygen therapy post-extubation • Tracheostomy
Renal	<ul style="list-style-type: none"> • Acute Kidney Injury • Requirement for Renal replacement therapy 	<ul style="list-style-type: none"> • Monitor urinary output, fluid and acid base balance • Avoid nephrotoxic medications • Renal replacement therapy for hyperkalaemia, severe metabolic acidosis, uraemia or overt fluid overload
Gastrointestinal/ Nutritional	<ul style="list-style-type: none"> • Postoperative Ileus • Increased catabolism 	<ul style="list-style-type: none"> • Cautious initiation of oral intake • Enteral feeding • Laxatives • Early dietitian input
Haematological	<ul style="list-style-type: none"> • Postoperative coagulopathy • Thrombocytopenia • Excessive chest drain output • Prevention of thromboembolism 	<ul style="list-style-type: none"> • Haemoglobin >100 g/l • POC thromboelastography guided management of coagulopathy • Blood products ± factor concentrates • Heparin prophylaxis 5000 units subcutaneous tds

Successful recovery and discharge from hospital requires the support of a highly specialised rehabilitation team comprised of physiotherapist, occupational therapists and specialist nurses.

Recommended Readings

1. Lopez-Marco A, Adams B, Oo AY. Thoracoabdominal aneurysmectomy: operative steps for Crawford extent II repair. *JTCVS Techniques*. 2020.
2. Coselli JS, Green SY, Price MD, et al. Spinal cord deficit after 1114 extent II open thoracoabdominal aortic aneurysm repairs. *J Thorac Cardiovasc Surg*. 2019.
3. Parotto M, Ouzounian M, Djaiani G. Spinal cord protection in elective thoracoabdominal aortic procedures. *J Cardiothorac Vasc Anesth*. 2019;33(1):200–8.
4. Rong LQ, Kamel MK, Rahouma M, et al. Cerebrospinal-fluid drain-related complications in patients undergoing open and endovascular repairs of thoracic and thoraco-abdominal aortic pathologies: a systematic review and meta-analysis. *Br J Anaesth*. 2018;120(5):904–13.
5. Luehr M, Salameh A, Haunschild J, et al. Minimally invasive segmental artery coil embolization for preconditioning of the spinal cord collateral network before one-stage descending and thoracoabdominal aneurysm repair. *Innovations (Phila)*. 2014;9(1):60–5.
6. Whitlock RP, Dieleman JM, Belley-Cote E, et al. The effect of steroids in patients undergoing cardiopulmonary bypass: an individual patient meta-analysis of two randomized trials. *J Cardiothorac Vasc Anesth*. 2020;34(1):99–105.