Anaesthesia for vascular surgery on the extremities

Richard J Telford

Abstract

Peripheral arterial surgery is challenging, operations are frequently long and associated with insidious blood loss. Because of the high incidence of comorbidities these patients are a high-risk group with a high incidence of morbidity and mortality. They key to successful outcome is meticulous attention to detail by all those professions involved in their care.

Keywords Amputation; embolectomy; peripheral vascular disease

Royal College of Anaesthetists CPD Matrix: 3A05

Learning objectives

After reading this article, you should:

- understand that patients with peripheral arterial disease are a high-risk group
- be able to describe the management of patients presenting for surgical revascularization of the lower limb and amputation
- have an understanding of the management of patients with thoracic outlet syndrome

Pathophysiology

Peripheral arterial disease (PAD) is a common manifestation of generalized atherosclerosis. The pathogenic processes underlying the disease are the same as those affecting the coronary and cerebral circulations. Multiple factors are implicated including dyslipidaemia, endothelial dysfunction, inflammation, oxidative stress, hypercoagulability and chronic infection.

Atherosclerotic plaques form in the medium and large sized arteries – the lower limb being affected more frequently than the upper limb. These plaques may cause a chronic slowly progressing luminal reduction leading to exercise induced symptoms of tissue ischaemia (intermittent claudication – IC) or to an acute vessel occlusion, usually due to plaque rupture and thrombosis which causes acute ischaemia. Each year 500–1000 new cases of critical limb ischaemia are diagnosed per million population with an estimated annual cost to the NHS of more that £200 million. Critical limb ischaemia can lead to limb loss or death if not treated promptly.

Epidemiology

The prevalence of PAD increases with age, affecting 15–20% of people aged >70. Many of these individuals are asymptomatic – only one in three or four people with PAD develop IC. PAD confers a similar risk of cardiovascular death as a history of coronary or cerebrovascular disease. Patients with PAD have a six fold increase in the risk of death from cardiovascular disease than those without PAD. It is important that risk factors are managed as aggressively as possible.

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Risk factors

Sex: The male to female ratio is 2:1 rising to 3:1 in patients with critical ischaemia.

Smoking: Smokers are four times as likely to develop PAD as non-smokers. Those who continue to smoke are more likely to need intervention or amputation than those who give up smoking.

Diabetes: Diabetic patients are twice as likely to have PAD. Amputations are five to ten times more frequent in the diabetic population than non-diabetic population. Good glycaemic control is paramount; a metanalysis found that a 1% increase in glycosylated haemoglobin is associated with a 26% increase in the chance of developing PAD.

Dyslipidaemia: Those with high levels of cholesterol, low density lipoprotein (LDL) cholesterol and triglycerides and low levels of high density lipoprotein (HDL) cholesterol are associated with an increased likelihood of developing PAD.

Hypertension: A blood pressure of greater than 160/95 is associated with a 2.5 times risk of developing intermittent claudication in men and a fourfold risk on women.

Chronic kidney disease (CKD) is associated with accelerated atherosclerosis. CKD promotes dyslipidaemia and hypertension which are in themselves risk factors for the development of atherosclerosis. In addition inflammatory mediators are often elevated and the renin angiotensin system is frequently activated in patients with CKD, enhancing production of reactive oxygen species and further contributing to the accelerated atherosclerosis observed in CKD.

Black ethnicity: Doubles the likelihood of developing PAD.

Chronic limb ischaemia

Patients with PAD usually develop IC, the site of which is determined by the location of the disease. Disease in the superficial femoral artery tends to present with calf claudication. Disease in the iliac, common femoral or tibioperoneal arteries present with pain in the buttock, calf or foot, respectively. There is usually a gradual reduction in walking distance over months or years. Rapid exacerbation of symptoms or an acute onset of claudication are important warning signs as they may herald acute arterial occlusion secondary to plaque rupture or embolus.

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Causes of acute limb ischaemia

- **Thrombosis**: (60%) - which is usually secondary to pre-existing atherosclerosis compounded by acute plaque rupture or reduced flow secondary to cardiac failure or hypovolaemia.

  Predisposing factors include:

  1. Hypotension,
  2. Unusual prolonged posture
  3. Malignancy
  4. Dehydration
  5. Hypercoagulability syndromes
  6. Hyperviscosity syndromes
- **Embolus:** (30%) — The majority of emboli are cardiac in origin e.g. atrial fibrillation or acute myocardial infarction. Most emboli lodge at arterial bifurcations.
- **Aortic dissection**
- **Acute peripheral aneurysm thrombosis**- (~5%)
- **Trauma** — (~3%) Limb fractures and dislocations
- **Iatrogenic** — arterial cannulation, inadvertent intra-arterial drug administration, extended tourniquet use during surgery, major pelvic surgery

Box 1

Major amputation is rare in claudicants — only 1—3% of patients with IC require major amputation within 5 years.

Acute limb ischaemia (ALI)

Approximately 5000 patients present each year with acute limb ischaemia in England and Wales. Associated mortality at 1 year is significant (~20%) as is subsequent limb loss (~35%).

The main causes of acute limb ischaemia are outlined in Box 1.

The clinical features of the most common aetiologies differ. Those of *thrombosis* include a previous history of intermittent claudication, an onset over hours or days and reduced or absent peripheral pulses in the contralateral limb indicating chronic peripheral arterial atherosclerosis. An *embolus* is characterized by an acute onset of symptoms associated with an identified embolic source. Patients rarely have a history of previous IC.

Clinical classification

ALI severity dictates the initial treatment of the patient and is determined by the presence of pain, paraesthesia or paralysis. Severity is classified according to the recommendations of the Society for Vascular Surgery/International Society for Cardiovascular Surgery 1997 (Table 1).

Clinical management

- Category I patients need treatment with intravenous heparin and analgesia. There is usually adequate time for patients to be fully investigated both surgically and medically and optimized prior to any surgical intervention.
- Category II patients also require heparinization and analgesia. In contrast to category I, complete acute ischaemia (category II) is a medico-surgical emergency as irreversible tissue necrosis results if perfusion cannot be restored within 6 hours of the onset of symptoms. There is minimal time for investigation. Resuscitation and preoperative optimization should not overly delay the proposed urgent intervention.
  - *Treatment*: embolectomy is usually the first line surgical management of category II ALI. This may be followed by on-table arteriography with subsequent thrombolysis, angioplasty, stenting or arterial bypass. Compartment syndrome secondary to reperfusion injury within the calf muscle necessitates fasciotomy in about 5% of patients whose ALI is successfully treated. Direct pressure measurement of compartment pressures is important. A compartment pressure of 30 mmHg or a diastolic blood pressure <30 mmHg above the compartment pressure is an indication for fasciotomy. Patients should be closely monitored for acute rhabdomyolysis with serial creatine kinase measurements and close monitoring of renal performance. A brisk diuresis should be maintained to avoid renal failure.
- Category III patients presenting with irreversible ischaemia may require urgent amputation. This procedure should not be unduly delayed for medical optimization in order to minimize the life-threatening systemic effects of extensive muscle necrosis in the affected limb. Terminal care is sometimes the most appropriate option in patients with extensive tissue involvement and significant comorbidities.

Clinical classification of acute limb ischaemia

Category	Sensation	Paralysis	Suggested treatment
I (Viable)	No loss of sensation	None	Not immediately threatened. Time to investigate
IIa (Threatened)	Minimal loss (e.g. toe)	None	Urgent treatment needed for salvage
IIb (Threatened)	More than toes and associated with rest pain	Partial	Immediate treatment needed for salvage
III (Irreversible)	Profound, anaesthetic	Profound/Rigor	Irreversible - Primary amputation

From Rutherford et al. Recommended standards for reports dealing with lower extremity ischemia: revised version. *J Vasc Surg* 1997; **26**:517—538.

Table 1

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Anaesthetic considerations

Emergency embolectomy: this is most commonly performed under local anaesthesia. Monitored anaesthesia care is recommended; patients frequently have significant comorbidity and may be restless and in pain. General anaesthesia may be required if the patient is uncooperative or if more extensive bypass procedures or fasciotomies are required. Since the majority of patients have received systemic anticoagulation with heparin, regional anaesthesia is usually contraindicated.

Surgical revascularization: the number of surgical procedures to revascularize the ischaemic lower limb has reduced dramatically in recent years as many lesions are amenable to radiological intervention (angioplasty with or without stenting). Indications for surgery include rest pain, ischaemic or gangrenous tissue loss, severe claudication/rest pain secondary to distal disease and failed non surgical treatment. Surgical revascularization involves bypass of the diseased segment with autologous vein grafts or synthetic material. Procedures are lengthy and are associated with a risk of perioperative myocardial infarction or death of >5%, due to the prevalence of multiple comorbidities.

Central neuraxial blockade, balanced general anaesthesia or a combination are suitable techniques. Spinal or epidural blockade have theoretical advantages as they enhance peripheral blood flow and reduce post-operative cognitive dysfunction but do not affect overall outcome. Prior anticoagulation may preclude central neuraxial blockade. Close attention should be paid to maintaining normothermia, normovolaemia, normotension and providing good post operative analgesia. Surgery may be prolonged (several hours) and blood loss may be insidious.

Minimal monitoring includes five-lead ECG, temperature, urine output, invasive arterial pressure if the procedure is expected to last greater than 2 hours. Non-invasive cardiac output monitoring should be considered. If GA is used, balanced anaesthesia (oxygen, air or nitrous oxide and a volatile agent) using positive pressure ventilation is preferable. Neuromuscular blockade is usually used to facilitate intubation and IPPV. The airway can be managed using a tracheal tube or supraglottic airway device.

Heparin 5000 iU is usually administered before graft placement. Restoration of blood flow to an ischaemic limb during surgery may cause transient myocardial depression and hypotension. Since hypothermia induced vasoconstriction may impair postoperative graft perfusion and predispose to myocardial ischaemia, patient warming devices and warmed intravenous fluids are mandatory. PVR can be painful and postoperative pain can cause vasoconstriction. A combination of morphine 0.15 −0.25 $mg.kg^{-1}$ with local anaesthetic block or infiltration and NSAIDs (if not contraindicated) is usually satisfactory. Higher doses of opioids may be required in those patients who are taking large doses of opiates to control rest pain preoperatively.

Amputation: five thousand major amputations are performed annually in the UK. Amputations may be primary due to overwhelming sepsis or severe intractable pain when there is no viable arterial reconstructive option or secondary after failed arterial reconstruction. The incidence of amputation is eight times higher in the diabetic population compared to non-diabetics.

Lower limb amputation is associated with a high morbidity and mortality; the latest National Vascular Registry report (2017) found in-hospital mortality rates of 5.8% for below knee and 12.4% for above knee amputations. Patients are frequently elderly and frail. They have a high incidence of coronary artery disease (severe CAD in up to 90% of patients), but cardiovascular symptoms may be masked by an inability to exercise. Associated hypertension, diabetes, chronic kidney disease and smoking related lung disease is very common.

Patients requiring major amputation require careful assessment by a multidisciplinary team.and optimization of controllable risk factors. Operative interventions should be performed promptly by a senior anaesthetist and surgeon, on a routine operating list during normal working hours (Unpublished data suggests a three fold increase in mortality of patients operated on 'out of hours' when compared to 'in hours'). Routine antibiotic and DVT prophylaxis should be prescribed according to local policy. There should be formal pain management protocols in place and early access to a team of rehabilitation specialists.

Anaesthetic techniques should achieve cardiovascular stability, maintain normovolaemia and provide excellent postoperative analgesia. Catheter-based regional anaesthetic techniques are frequently used (epidural, ultrasound guided femoral and popliteal catheters) as they provide excellent post operative analgesia and improved postoperative respiratory function, and may help to reduce post-operative cognitive dysfunction. Aspirin is not a contraindication to central neuraxial blockade. The risk—benefit ratio of using central neuraxial blockade if the patient is taking clopidogrel needs careful documentation. Many vascular anaesthetists are perfectly happy to administer central neuraxial blockade in patients taking clopidogrel, however some patients may be systemically anticoagulated which contraindicates central neuraxial blockade.

If general anaesthesia is chosen it may be supplemented by surgically placed peripheral nerve catheters to enable a dilute solution of local anaesthetic to be infused around the sciatic and/or femoral nerve.

Phantom limb pain (PLP) is a significant complication of lower limb amputation with up to 70% of patient experiencing PLP at some stage. Peripheral nerve transection results in afferent nociceptive stimulus causing spinal cord hyperexcitability. Preemptive analgesia with epidural infusions, intrathecal and intravenous ketamine have not consistently reduced the incidence of PLP. Involvement of the acute pain team preoperatively to optimize pain control using multimodal analgesic techniques (including drugs for neuropathic pain — gabapentin and amitriptyline) may be useful.

Thoracic outlet syndrome (TOS)

TOS is characterized by symptoms relating to dynamic compression of the neurovascular bundle of the upper limb as it passes between the uppermost rib and the clavicle (Box 2). Clinical signs and symptoms may be neurological or vascular depending on which component of the neurovascular bundle is involved.

Neurogenic TOS accounts for the majority of cases; 90% of cases involve the C8 and T1 nerve roots. Patients complain of pain and

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Aetiology of TOS

Skeletal and bony abnormalities:
- Cervical rib, elongated C7 transverse process
- Exostosis or tumour of the first rib or clavicle
- Excess callus formation the first rib or clavicle following a fracture

Soft-tissue abnormalities:
- Fibrous bands
- Congenital variations in scalene muscle anatomy
  - Insertion variations
  - Supernumerary muscles

Acquired soft-tissue abnormalities:
- Post traumatic fibrous scarring
- Postoperative scarring

Poor posture and weak muscular support

Box 2

parasthesiae in the ulnar nerve distribution and weakness of the small muscles of the hand. The C5, C6 and C7 nerve roots are compressed less frequently causing pain referred to the neck, ear upper chest and outer arm.

Arterial TOS is caused by compression of the subclavian artery as it passes between a cervical rib or band and scalenus anterior causing subclavian arterial stenosis. Post-stenotic dilatation and aneurysm formation may be seen. There may be occlusion of the subclavian artery. Presenting symptoms are those of brachial ischaemia or a pulastile mass in the supraclavicular fossa.

Venous TOS (Paget-Schroetter syndrome) causes 'effort thrombosis' of the axillo-subclavian vein leading to swelling, aching and venous congestion of the arm, classically after strenuous upper body exertion. Approximately 10% of patients may develop pulmonary emboli. Individuals may notice the development of prominent veins over the chest and shoulder area on the symptomatic side. Repetitive compression of the subclavian vein causes venous stasis and intimal damage which further stimulates the coagulation system, eventually leading to thrombosis.

Treatment of thoracic outlet syndrome

Neurogenic: Vigorous attempts are made to treat neurogenic TOS conservatively. Physiotherapy is used to improve posture and relax the scalene muscles. Multimodal analgesic techniques are useful. Surgical exploration of the brachial plexus is indicated if conservative measures fail and if abnormal anatomical features are identified.

Arterial: Mild postural ischaemia is treated with physiotherapy. Initial treatment of complicated arterial TOS is focussed on revascularization to treat acute brachial ischaemia. Thrombolyis or thromboembolectomy may be indicated, and arterial reconstruction may be required to treat lesions that are often a combination of occlusive and aneurysmal in nature. Some patients are candidates for surgical decompression.

Venous: The treatment of venous TOS depends on the age and future aspirations of the patient. Younger sportsmen and women and those who are manual workers are offered a package of thrombolysis and surgery. Following successful lysis the patient is placed on heparin until transaxillary first rib resection can be performed on the next available list. Transaxillary rib resection is followed by venography and balloon venoplasty 2–3 weeks postoperatively to maintain vein patency.

Surgical decompression

Surgical procedures for TOS are varied. They vary from excision of bony and soft tissue abnormalities to complex vascular reconstructions. Some patients require excision of the first rib to relieve neurovascular compression. There are two surgical approaches to the first rib supraclavicular and transaxillary. Uncomplicated arterial and venous TOS cases are approached via the transaxillary route. It is possible to remove a cervical rib using a transaxillary route but the first rib must be removed initially to gain safe access. Complicated arterial cases (usually associated with aneurysm formation) and neurological TOS with a cervical rib/band merit a supraclavicular approach.

Anaesthetic considerations

Preoperative: The majority of patients are young and have minimal comorbidity. A group and save should be performed as there is potential for catastrophic blood loss.

Intraoperative: The patient is placed supine for a supraclavicular approach however if a transaxillary approach is planned the patient is positioned in the lateral position, operative side uppermost. Surgical access is more challenging with this approach as the arm has to be elevated upwards to open up enough space in the axilla to access the first rib. Large bore venous access is mandatory as surgical dissection is around the subclavian vessels and catastrophic haemorrhage has been reported. Air embolus is also a possibility. A high-dose opiate, low-dose hypnotic balanced general anaesthetic is required, and as a nerve stimulator may be used in complex neurological cases muscle relaxants should be avoided. Invasive monitoring is rarely required. Although the apical pleura is frequently breached with first rib resection perioperative hypoxia is rarely a problem; however avoidance of nitrous oxide is sensible.

Analgesia: Superficial cervical plexus block provides useful cutaneous analgesia if the supraclavicular approach is used. Patients frequently require additional increments of intravenous opioid analgesia in recovery. Patient controlled analgesia and simple regular analgesia with paracetamol and a non-steroidal anti-inflammatory agent should be prescribed.

Postoperative: An erect chest X-ray should be obtained before the patient leaves the recovery ward to exclude significant residual pneumothorax. Formal underwater seal drainage is rarely required. Although a drain is frequently employed, significant postoperative blood loss may not be apparent. A haemothorax may insidiously accumulate as shed blood tends to pass through the breach in the apical pleura rather than the drain. Occasionally, patients may have to return to theatre for video assisted removal of a large intrathoracic blood clot. Formal thoracotomy

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to control severe bleeding has been reported, usually when the subclavian artery has been damaged during surgical dissection. Most patients are able to go home 2–3 days postoperatively.

Vascular access surgery

Vascular access surgery for renal dialysis is amongst the most common operations carried out by vascular surgeons. In part this is because the number of patients on haemodialysis is steadily increasing in the UK. Successful haemodialysis mandates vascular access than can provide an extracorporeal blood flow rate of at least 300 ml/min via a conduit that can be accessed up to three times per week. Although an indwelling dialysis catheter can be used in the short term most long-term haemodialysis patients are dialysed using an arteriovenous (AV) fistula (a direct anastomosis between an artery and a vein) or an arteriovenous graft (when an artificial graft material is interposed between and artery and vein). Arteriovenous fistulae are used in the first instance if possible as they have greater long term patency rates (~5 years for a fistula, ~ 2–3 years for a graft) and a lower infection risk as no prosthetic material is involved.

Anaesthetic considerations

Fistulae can be created under local regional or general anaesthesia. Since renal patients frequently have multiple comorbidities many centres avoid general anaesthesia and prefer to use brachial plexus blockade. Ultrasound guided supraclavicular block 'the spinal of the arm', is most frequently used. Use of regional anaesthesia enhances forearm blood flow which may be associated with increased blood flow through the fistula/graft in the immediate postoperative period. There is no evidence that regional anaesthesia confers any long-term benefit in terms of fistula/graft survival. ◆

FURTHER READING

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