Arterial disease and its management

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Introduction

In this chapter, an overview of the arterial pathologies that present commonly to the vascular surgeon will be given. Included will be a summary of pathophysiology and epidemiology of these conditions as well as their clinical assessment and the imaging modalities used in contemporary practice. Finally, available treatment options, both open surgery and endovascular, will be summarised.

Atherosclerosis

Atherosclerosis is a disease of large and medium-sized arteries and results in the build-up of cholesterol, lipid, calcium, smooth muscle cells and other cell debris in the tunica intima of the involved vessel. The cause of this accumulation is complex and multifactorial, but the common theme is an injury to the vascular endothelium that sets off a cascade of events leading to plaque formation. There are different types of injury at work simultaneously including chemical (e.g. noxious agents present in cigarette smoke, hyperglycaemia, hyperlipidaemia) and physical issues (e.g. the presence of untreated hypertension, a predisposition for plaque development at points of high shear stress in the arterial tree such as at the carotid bifurcation). A section of carotid endothelium, excised during a carotid endarterectomy, is shown in Figure 1.1. The specimen contains an ulcerated atheromatous plaque. The earliest manifestation of atherosclerosis is the ‘fatty streak’ and as the name suggests this is the visible accumulation of lipids and inflammatory cells in the tunica intima below the endothelium. In the Western world, most people in their 20s will have evidence of fatty streak formation. With lesion progression, lipid and smooth muscle cell proliferation results in a fibrous plaque, which encroaches on the vessel lumen. On the luminal aspect the endothelium becomes increasingly attenuated as a so-called fibrous cap. Eventually this cap gives way and plaque rupture occurs. It is at this juncture that acute vessel occlusion can occur or distal atheroembolisation and these events correlate with significant clinical sequelae (for example, acute myocardial infarction, limb ischaemia, carotid territory transient ischaemic attack or stroke).

Although it is clearly a systemic disorder, vascular surgeons encounter atherosclerotic occlusive disease in certain specific locations. Most common is lower limb disease giving rise to intermittent claudication and if more severe, limb-threatening critical ischaemia. Carotid artery disease is a common cause of stroke and is a frequent manifestation of the disease.
Peripheral arterial disease

Epidemiology

The Edinburgh Artery Study demonstrated that symptomatic peripheral arterial disease (intermittent claudication) is present in 2–7% of 50–74-year-olds [1]. Asymptomatic disease, detected by ankle–brachial pressure measurements is commoner, affecting about a quarter of the population over 50 years of age. The major risk factors for the development of peripheral arterial disease (PAD) include cigarette smoking, hypertension, diabetes mellitus and hypercholesterolaemia.

The recently published REACH registry data confirm the serious prognosis for patients with a diagnosis of lower limb arterial disease [2]. Over the first 12 months of the study, 21% of patients with PAD either died from cardiovascular disease, suffered a non-fatal myocardial infarction or stroke or were admitted to hospital because of another vascular event. Patients who in addition to PAD also had symptoms of coronary artery disease and/or cerebrovascular disease had a substantially higher incidence of these events.

Clinical presentation

Clearly the mode of presentation will be dictated by the vascular bed involved.

Lower limb ischaemia presents as either non-limb-threatening intermittent claudication or critical ischaemia with the presence of opiate-dependent rest pain and/or tissue loss or gangrene. Clinically, the level of occlusion(s) can be estimated by the presence or absence of pulses at groin, popliteal or ankle level. The measurement of ankle–brachial pressure indices is helpful but beware patients with heavily calcified vessels (e.g. diabetics) in whom the ankle vessels are incompressible and pressure measurements not particularly helpful.

Cerebrovascular events (transient ischaemic attack or stroke) can frequently be caused by embolisation from active plaques affecting the proximal internal carotid arteries. The decision about management is made based upon the degree of carotid stenosis present and the time from event to presentation. Drop attacks due to subclavian steal typically arise when patients have been using the upper limb above head level. Absent ipsilateral upper limb pulses will be the clinical clue to the diagnosis.

Mesenteric ischaemia is often a diagnosis made too late when irreversible bowel ischaemia has developed. When chronic, the symptoms of weight loss, post-prandial abdominal pain and diarrhoea and fear of food should point to the diagnosis. Co-existing atherosclerotic disease (lower limb ischaemia, ischaemic heart disease) should heighten clinical suspicion.

Aggressive surgical or endovascular intervention for renovascular disease presenting as either deteriorating renal function or drug-resistant hypertension has become far less common than was previously the case. Evidence gathered over the years indicates that most interventions do not confer any advantage over drug therapy, the ASTRAL trial being the most recent evidence base for this approach [3].

Investigation of arterial disease

Nowadays, the vast majority of patients can be investigated non-invasively. Duplex ultrasound gives very clear images of much of the arterial tree. As well as a grey-scale image, colour flow and spectral waveform analysis allow accurate delineation of the extent and degree of arterial stenosis and occlusion. Figure 1.2 is a duplex ultrasound of a carotid bifurcation with colour flow imaging which shows narrowing and turbulent flow in the internal carotid artery. In some situations, such as aorto-iliac disease not easy to visualise with duplex or mesenteric arterial disease, alternative imaging modalities are necessary. Magnetic resonance angiography (MRA) has become a very valuable tool in this setting. There is no need for ionising radiation or iodinated contrast and often it complements information obtained from duplex ultrasound. One has to remember that MRA is not
safe for patients with significantly deranged renal function as there is a well-documented potential risk of disseminated systemic fibrosis [4]. Computerised tomographic angiography (CTA) can be helpful in such patients, but heavy calcification can compromise the quality of images obtained.

It is relatively rare in contemporary practice to use intra-arterial angiography as a tool to investigate occlusive disease. Rather this technique comes to the fore during (endovascular) intervention.

**Treatment**

**Lower limb ischaemia**

At initial presentation, patients with intermittent claudication will be carefully assessed clinically. All risk factors should be corrected, especially smoking. It is not uncommon to diagnose diabetes mellitus in this patient group and this should be managed appropriately. Hypercholesterolaemia and hyperlipidaemia should be identified and appropriate treatment with statin therapy commenced. There is strong evidence for administering statins to these patients irrespective of their lipid profile [5].

Depending on the social circumstances (e.g. symptoms threatening employment) most surgeons manage newly diagnosed claudicants conservatively in the first instance with risk factor control and exercise. There is considerable evidence for the beneficial outcome of this approach [6].

However, as angioplasty and stenting are readily available, particularly when risk factors have been controlled, intervention is not unreasonable. Certainly iliac artery disease giving rise to disabling buttock and thigh claudication responds very well to angioplasty and stenting with durable results [7]. Compared to the available surgical options (e.g. aortofemoral and iliofemoral bypass) with the attendant risks and complications, percutaneous intervention is a good option.

Infrapopliteal disease is not so straightforward. Although the results of balloon angioplasty for short stenoses and occlusions are reasonable, more often than not there will be disease of considerable lengths of the superficial femoral artery. Although these lesions can usually be reopened percutaneously, either transluminally or more often subintimally, the medium and long-term patency rates are not great and failure necessitates reintervention or even conversion to surgery.

It is relatively uncommon to proceed with surgery for intermittent claudication in modern practice. This is largely a result of the available endovascular options and the not inconceivable risk of surgery in these patients. However, there are a few exceptions. Younger patients with extensive aorto-iliac disease get the most durable long-term result from direct aortobifemoral bypass. Careful assessment for occult coronary disease is important prior to surgery. Unilateral iliac occlusions can be treated with low risk either by iliofemoral or femorofemoral bypass.

The approach to surgical intervention for infrainguinal occlusive disease has changed considerably. In the 1980s it was common practice to insert prosthetic above-knee grafts in such patients. Time has taught us that often these grafts fail due to disease progression and patients re-present with critical ischaemia. Good-quality long saphenous vein should be the bypass conduit of choice in this setting [8].

The situation in critical ischaemia is different. Investigations will confirm the location and extent of disease. Often disease is present at multiple levels and so this needs to be taken into account. The underlying principle is that good inflow is a must. Often if this is achieved (percutaneously or surgically) and the profunda femoris artery is patent, adequate revascularisation will result.

For infrainguinal disease many surgeons will adopt an angioplasty-first approach as these patients have a poor prognosis (40–50% mortality at 2 years). The BASIL trial indicated that this was reasonable for patients with a short life expectancy but for patients with greater than 2 years’ life expectancy who had good-quality saphenous vein, surgical bypass was the better option [9].

**Carotid artery disease**

Since the early 1990s, clinical practice has been guided by the results of the North American and European Symptomatic Carotid Surgery Trials (NASCET and ECST) [10,11]. For patients with high-grade symptomatic carotid artery stenosis, carotid endarterectomy (CEA) provides significant protection from stroke compared to best medical therapy (BMT). The subsequent asymptomatic carotid surgery trials, albeit the North American trial was stopped early because of the steering committee’s feeling that surgery was significantly superior to BMT, also showed a benefit but far less impressive than for symptomatic patients [12,13]. In the UK,
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most vascular surgeons are fairly selective about offering CEA to asymptomatic patients. The group with greatest benefit appears to be men under the age of 68 years with bilateral high-grade carotid stenoses.

There is increasing evidence to suggest that the benefit of intervention is greatest in the first few days after the index event. There is now an ongoing effort to educate the public and non-vascular medical practitioners to recognise the emergent nature of transient ischaemic attack (TIA) and the need for immediate assessment of these patients. Many hospitals have developed TIA clinics that operate in a similar fashion to acute chest pain clinics. Pathways have been developed for urgent referral to vascular services for CEA within 2 weeks of the index event [14].

There are a lot of data in the Cochrane Database now regarding the conduct of CEA. Conventional endarterectomy with routine patching are recommended. The GALA trial failed to demonstrate a significant advantage in terms of operative stroke, death or complication for the use of regional over general anaesthesia and so it is recommended that the operating team use the approach that yields the best results in their hands. The use of shunts is another controversial subject. Under regional anaesthesia this use can be selective with most surgeons shunting about 10% of patients. Operators using general anaesthesia use either routine shunting or selective shunting based upon intraoperative monitoring (transcranial Doppler, oximetry, EEG, etc.).

Carotid angioplasty and stenting (CAS) is now fairly widespread in clinical practice in Europe and the USA. In both of these regions, the majority of patients treated are asymptomatic. Many randomised trials of variable quality have been performed over the years. Suffice it to say that the jury is still out, especially for the use of this technology in acutely symptomatic patients. The most recently published trial data indicated a significantly higher stroke rate for CAS vs. CEA in symptomatic patients [15].

Subclavian/innominate artery disease

Patients with occlusive disease of these vessels present in a variety of ways depending on the extent of disease. Subclavian artery origin disease is often asymptomatic but can give rise to subclavian steal. Investigations will demonstrate reversal of flow in the vertebral artery affected.

Traditional surgical options are carotid to subclavian bypass or subclavian transposition. The majority of symptomatic patients can be treated by balloon angioplasty and stenting. Similarly innominate stenosis can be managed by endovascular means but there is a not insignificant stroke risk. Also these lesions tend to be heavily calcified and often cannot be reopened by balloon techniques. Occasionally direct surgical bypass from the ascending aorta is necessary. This is a major procedure with considerable operative risk and requires sternotomy. Such operations are seen pretty rarely.

Mesenteric arterial disease

The options for patients with chronic mesenteric ischaemia are either surgical bypass or balloon angioplasty and/or stenting. Surgery is probably the more durable reconstruction but is associated with risk of mortality and morbidity. The traditional procedures are either bypass to the superior mesenteric artery ( SMA) directly from the infrarenal aorta, retrograde bypass from the right iliac system (if the aorta is heavily diseased) or bypass from the supraceliac aorta often combined with coeliac revascularisation. The key is adequate revascularisation of the SMA [16].

There is a growing pool of data on the results of endovascular mesenteric revascularisation. Results are best for short proximal lesions usually combining angioplasty and stenting [17].

Aortic aneurysm disease

Pathology

The development of aneurysmal disease of the aorta is a complex, multifactorial pathology.

There are probably a number of different mechanisms at play including enzymatic degradation of the aortic wall (increased collagenase, metalloproteinase and elastase activity has been demonstrated), inflammatory cell infiltration (macrophages and lymphocytes) giving rise to increased cytokine activity and enzyme secretion, biomechanical factors including wall shear stress in combination with biochemical events and a likely genetic predisposition in combination with these other factors.

Histologically, aneurysm tissue demonstrates loss of elastin and collagen in the tunica media together with decreased numbers of smooth muscle cells, inflammatory cell infiltration and neovascularisation.

About 90% of aortic aneurysms involve the infrarenal aorta, the remaining 10% variably involving
the thoracic and thoracoabdominal aorta. The male:female ratio for infrarenal abdominal aortic aneurysm (AAA) is at least 5:1, whereas that for thoracic and thoracoabdominal aneurysms is about 1.5:1. There is a strong familial tendency with male siblings being ten times more likely to have an aneurysm. The disease affects patients in their 60s and 70s although younger patients can be affected if they have an underlying connective tissue disease such as Marfan syndrome.

Clinical presentation
The majority of patients are asymptomatic. Diagnosis is often made during routine abdominal palpation or more commonly as a coincidental finding when investigations are performed for other reasons (e.g. investigation of the urinary tract). Figure 1.3 shows an abdominal ultrasound scan demonstrating an aortic aneurysm.

As population screening is unfortunately not yet undertaken in most countries, today still a considerable proportion of patients with AAA present acutely with aortic rupture.

Management
Asymptomatic patients with AAA maximum diameter less than 5.5 cm are typically managed in surveillance programmes with serial ultrasound measurement of maximum anteroposterior aneurysm diameter [18]. For AAA between 3 and 4 cm, scans are repeated annually; for lesions between 4 and 5 cm 6-monthly and between 5 and 5.5 cm 3-monthly.

When AAA diameter exceeds 5.5 cm, intervention should be contemplated. At this size annual rupture risk is probably between 3% and 7%, rising to about 10% at 6 cm and exceeding 20% above 7 cm.

The assessment of fitness for intervention is covered elsewhere in this book.

Assuming patients are deemed fit for intervention, the options for treatment are either open surgical repair (OR) or endovascular repair (EVAR). The EVAR I trial demonstrated a significantly lower operative mortality for EVAR vs. OR in patients fit for OR (1.75 vs. 4.7%) [19]. Over follow-up, the survival curves for the two groups remained parallel for about 4 years after which they crossed and when the trial was closed at 8 years, EVAR patients seemed to have fared rather less well in terms of aneurysm-related mortality. All late-ruptured AAA were seen in the EVAR group [20]. The Achilles heel of EVAR is the need for lifelong monitoring of the grafts and reintervention on a proportion of patients. The late results of the trial are a slight cause for concern when considering this modality for relatively young, fit patients.

The decision about the feasibility of EVAR is dictated by various anatomical issues. A proximal landing zone for the stent graft that is adequate to seal the infrarenal aorta above the aneurysm sac is necessary. This should be 15–20 mm in length, be of relatively uniform, undiseased diameter aorta and not be excessively angulated. Distally iliac involvement may dictate where the iliac limbs are deployed and this may lead to the need to sacrifice one of the internal iliac arteries. In order that the sheath containing the stent-graft can be passed safely retrograde from the femoral arteries into the aorta, it is necessary that the (external) iliac artery has a minimum diameter of 7 mm. In Figure 1.4 an endovascular stent can be seen in place following a stent-graft repair of an infrarenal aortic aneurysm.

As stent-graft technology progresses the boundaries can be pushed out to treat shorter, more angulated aneurysm necks and as the devices are mounted on lower-profile delivery devices, so smaller iliac access will not be a barrier to successful EVAR.

When it was first developed, it was envisaged that EVAR would be especially useful in treating patients with large AAA unfit for open surgery. The EVAR II trial compared the outcome for patients thought unfit for open surgery who were treated with EVAR and those treated with open surgery. The results indicated that endovascular repair was a safe and effective alternative to open surgery, with lower morbidity and mortality rates. However, the long-term follow-up is still ongoing and further studies are needed to determine the true benefits and risks of EVAR.
for open repair who were randomised to EVAR or medical therapy [21]. A number of criticisms were levelled at the trial methodology. The definition of ‘unfit for open repair’ was not particularly well defined and so a spectrum of level of fitness was probably randomised. There was a not inconsiderable number of patients who crossed over during the trial from the medical treatment group to EVAR. Even so, at 4 years almost 70% of patients in each group had died from non-aneurysm-related causes. Thus the rather surprising and disappointing conclusion from the EVAR II trial was that patients with AAA who are unfit for open surgery should not be offered EVAR or open repair.

Suprarenal and thoracoabdominal aneurysm repair
The situation for aneurysms involving the suprarenal, thoracoabdominal and thoracic aorta is more complicated. Open surgery requires a more complex strategy as these procedures involve a degree of visceral ischaemia reperfusion. For extensive thoracoabdominal aortic aneurysms (TAAA) this includes potential ischaemic injury to the spinal cord and resulting paraplegia.

Early exponents of thoracoabdominal aneurysm repair had no choice but to adopt the so-called ‘clamp and go’ technique. This involved clamping the aorta above and below the aneurysm (e.g. the proximal descending thoracic aorta and common iliac arteries), opening the entire aneurysm sac and performing proximal aortic and visceral branch anastomoses as quickly as possible. This was extremely stressful for patient and surgeon alike! Not surprisingly the complications, especially spinal cord and renal, increased significantly in direct relation to the duration of clamping. Nowadays, these operations are best performed in centres with extensive experience as the literature confirms a direct correlation between volume and outcome [22].

In addition, surgeons performing TAAA surgery make use of a number of intraoperative adjuncts to help reduce the incidence of spinal cord and abdominal organ dysfunction. The operations are done using a sequential clamp technique. In conjunction with left heart bypass and a centrifugal pump the abdominal organs and spinal cord can be perfused retrograde via a left femoral artery cannula during the procedure. Body temperature can be lowered as required (e.g. to 31–32 °C). When the visceral arteries are being anastomosed their perfusion can be maintained using selective cannulation [23]. The spinal cord is protected in a number of ways including drainage of cerebrospinal fluid, reimplantation of intercostal arteries during the operation and the maintenance of mean arterial pressure above 80 mm Hg during the first few days postoperatively. Some centres also monitor motor evoked potentials during the operation and this helps direct the best intercostals arteries to reimplant [24]. All of these techniques have greatly improved the results of this surgery. In centres of excellence repair of appropriately selected patients with extensive TAAAs is associated with operative mortality of 5–10%, paraplegia rates of 2–4% and dialysis-dependent renal failure rates of under 5%.

Thoracic endovascular aneurysm repair (TEVAR)
Endovascular stent-graft technology has revolutionised the management of thoracic aortic pathology. Isolated aneurysms of the descending thoracic aorta should all be treated using this technology nowadays. Instead of thoracotomy, heart bypass etc. grafts can be deployed via a small groin incision and femoral arteriotomy. Depending on the length of aorta to be covered cerebrospinal fluid drainage may be required and if it is necessary to cover the left subclavian artery proximally, carotid–subclavian bypass is recommended.
to reduce paraplegia and stroke risk. Otherwise these procedures are very straightforward and recovery time is a matter of 2 or 3 days. The procedure can be performed under regional anaesthesia. For thoracic aneurysms involving the aortic arch and thoracoabdominal lesions involving the visceral segment a number of options for endovascular treatment are available [25]. It is often possible to perform extra-anatomic grafts relocating the inflow to the carotid and left subclavian arteries thereby creating an adequate proximal landing zone for stent-graft deployment. Indeed, on occasions it is possible to ligate the origin of the innominate artery, take a graft from the proximal ascending aorta to the innominate bifurcation and perform right carotid to left carotid and left carotid to subclavian grafts creating a proximal landing zone in the ascending aorta. This means that aortic arch aneurysms can be treated by endovascular means without aortic cross-clamp. Although this approach requires a sternotomy, compared to the open surgical option which is hypothermic arrest with all of its associated complications, patients tolerate this procedure well.

Similarly, within the abdomen, retrograde revascularisation of the coeliac axis, superior mesenteric and renal arteries can be achieved with multiple grafts from the iliac arteries [26]. This allows stent-grafts to be deployed within aneurysmal disease of the thoracoabdominal aorta including the abdominal visceral segment.

More recently, stent-graft technology has progressed further such that fenestrated and multi-branched stent-grafts have been developed to allow an entirely endovascular approach to the repair of these complex aneurysms [27]. This technology is in its relative infancy and thus far is limited to highly selected patients in a few pioneering centres. Extensive coverage of the thoracoabdominal aorta during TEVAR is associated with higher paraplegia rates than one sees in open surgery.

Aortic dissection
This refers to an acute tear in the tunica intima of the aorta that leads to blood flowing in a second channel located in the tunica media and extending proximally or distally to a variable extent. The term dissection is somewhat historical (and probably a post-mortem finding in days gone by). Nowadays, largely based upon the sophisticated imaging that we have available the term acute aortic syndrome is a useful term and covers the initiating event. The site of the intimal tear can be located in the ascending or descending aorta and its location dictates the clinical sequelae.

About 60% of acute aortic dissections have an entry tear in the proximal ascending aorta and extend distally into the descending aorta to a variable extent (DeBakey type I). DeBakey type II dissections are restricted to the ascending aorta only. These two subgroups constitute the so-called Stanford type A dissections. Dissections that commence at or just beyond the left subclavian artery and propagate distally are known as DeBakey type III or Stanford type B dissections and account for about one-third of cases. Men are affected twice as often as women. Pre-existing, poorly controlled hypertension is present in almost 80% of patients. Age at presentation is most often 50–70 years of age. Ten per cent of patients will suffer from a connective tissue disorder, most often Marfan syndrome.

Acute Stanford type A dissections are a cardiac surgical emergency. A CT scan of such a dissection is shown in Figure 1.5. If the aorta has not ruptured freely but there has been a contained rupture often with retrograde propagation involving the coronary ostia and aortic valve with or without haemopericardium, emergency surgical replacement of the ascending aorta with reimplantation of the coronary arteries and aortic valve resuspension or replacement if necessary is the treatment of choice. At this procedure the
false lumen is closed distally just proximal to the innominate artery origin, but the remainder of the aorta is left, unless there is evidence of distal visceral or lower limb ischaemia. In a proportion of patients the false lumen remains perfused distally as there are often multiple entry tears and in this situation late aneurysmal change can develop.

Stanford type B dissections can present as acute aortic rupture. As for type A lesions, this is most likely to occur in the first 24 hours after acute dissection, decreasing in incidence with the passage of time. Conventional treatment of uncomplicated type B dissections is conservative with aggressive control of systemic blood pressure. Intervention in the acute phase has traditionally been reserved for contained leak and distal vascular compromise. About 20% of patients who survive the acute event will develop late aneurysmal change in the dissected segment of aorta due to expansion of the thin-walled, perfused false lumen. It is for this reason that some enthusiasts advocate early endovascular intervention for all type B dissections [28]. In theory, by placing a thoracic stent-graft over the entry tear within a short time of the acute event one prevents ongoing perfusion of the false lumen, and encourages flow to be directed through the true lumen thereby allowing the aorta to remodel and avoid aneurysmal change developing later. By the same token, others have argued that up to 80% of uncomplicated type B dissections do not develop late aneurysmal change and so the majority of these interventions are pointless. Most clinicians would look for evidence of early dilatation of the proximal descending aorta on CT scan soon after the acute event. In such circumstances, TEVAR is a very appealing option. Endovascular treatment of chronic type B dissections is notoriously less easy to achieve because of difficulty with adequate length of proximal landing zones and because chronic dissection flaps tend to be very rigid and difficult to seal with stent-grafts.

References
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Vascular surgery is a potent unnatural selector of the fittest. The more likely it is that a patient will die after scheduled surgery the more reason he or she should consult a senior anaesthetist preoperatively. This consultation should be scheduled soon after identification of pathology that could be treated surgically. This includes small abdominal aortic aneurysms (AAAs) detected by chance or through screening. Patients should have as much time as possible to consider their options and shift risk of harm to risk of benefit.

This chapter reviews preoperative survival assessment for vascular surgical patients. Further details are in chapters specific to particular operations. Throughout this chapter the word ‘risk’ is synonymous with ‘chance’. We concentrate on assessment and preparation of patients with AAAs: screening will make this operation more common, postoperative mortality is high and it is uncertain what proportion of patients benefit and what proportion are harmed.

**Benefit versus harm in surgery**

**Health objectives**

The aim of surgery can be palliation of existing symptoms (peripheral arterial bypass surgery for pain). It can also be used to prevent future symptoms (carotid endarterectomy for stroke). Surgery can benefit patients by making life better and possibly longer. Surgery can worsen quality of life and reduce its duration. Preoperative preparation is a cooperative venture between patient, surgeon and anaesthetist intended to quantify the risks of benefit and harm, coupled with attempts to shift their balance in the patient’s favour.

**Evidence that vascular surgery works**

The effect of an intervention is best disentangled from confounding factors by testing in randomised controlled trials (RCTs). Surgery should be justified by improved survival and quality of life. However, RCTs do not support vascular surgery except carotid endarterectomy (CEA) in symptomatic patients.

- RCT evidence does not justify bypass surgery for chronic leg ischaemia (no RCTs).
- RCT evidence does not prefer surgery to exercise for chronic leg ischaemia (one RCT).
- RCT evidence does not justify endovascular stenting for chronic leg ischaemia (two RCTs).
- RCT evidence does not justify elective repair of AAAs between 4 cm and 5.5 cm diameter (two RCTs).
- RCT evidence does not justify elective repair of AAAs larger than 5.5 cm diameter (one RCT).
- RCT evidence for endarterectomy of asymptomatic stenotic carotid arteries is equivocal (three RCTs).
- RCT evidence does not justify elective thoracic endovascular aortic repair for stable type B aortic dissections (one RCT).

Other vascular surgeries continue on the assumption that other sources of evidence, such as observational data, have sufficiently evaded inherent biases that the impression we have of net benefit is correct. For instance people die from ruptured AAAs, mortality after emergency AAA surgery is higher than after elective AAA surgery, larger AAAs have a higher incidence of rupture. Of course many people with AAAs die from other causes. Interpretation of all