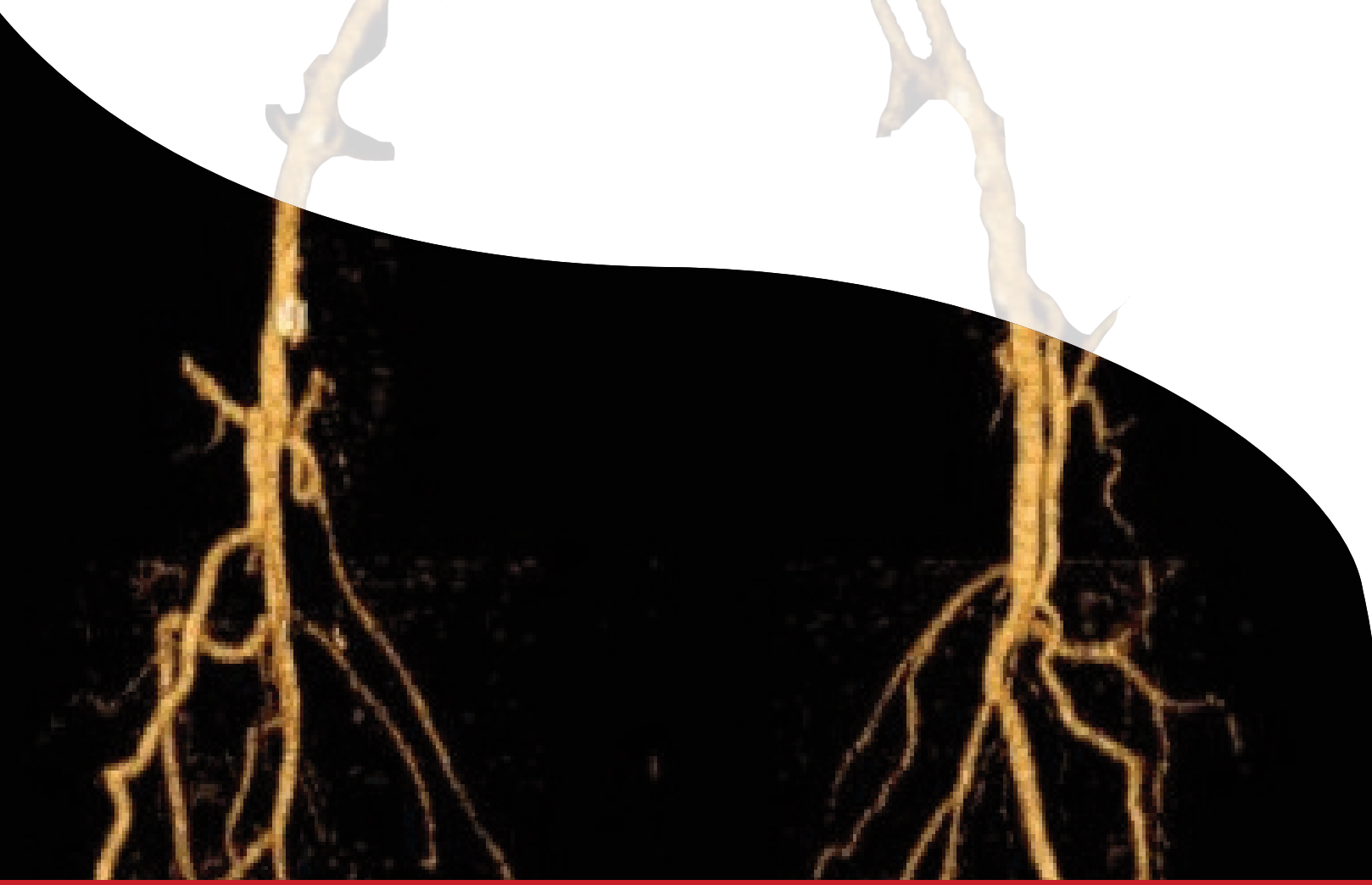
All you need to know about
Vascular Surgery



A GUIDE FOR MEDICAL STUDENTS, EARLY YEAR DOCTORS AND ALLIED HEALTHCARE PROFESSIONALS

VASCULAR SOCIETY

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FOREWORD

Vascular surgery involves the medical and surgical management of patients with diseases of the arteries, veins and lymphatics. I am biased but, in my humble opinion, vascular surgery is the greatest surgical specialty. The surgery is technically challenging and exciting and involves life and limb saving operations for peripheral arterial disease, carotid artery disease and aortic aneurysms. The medicine is also important as our patients are often frail with multiple co-morbidities. In addition much of our practice is evidence based and active vascular research helps us make the right treatment decisions for our patients.

The surgical specialities are often only covered briefly in undergraduate courses and so this book hopes to give you an introduction to vascular surgery and inspire budding doctors to consider training in this brilliant surgical specialty. This beautifully illustrated and easy to read book has been written for medical students, doctors in the early stages of their career as well as allied healthcare professionals who are looking to gain a greater understanding of vascular surgery.

I would like to thank everyone who has helped bring this book to fruition by contributing chapters, editing and illustrating skills – it has been a real team effort to provide this important free resource.

Please enjoy reading or dipping into as a quick reference guide.

Miss Rachel Bell MS FRCS
Consultant Vascular Surgeon
President of the Vascular Society of Great Britain and Ireland

INTRODUCTION

The many contributors to this volume share a love for vascular surgery; its many complexities, its joys, its anatomical range, its beauty and its frustrations. We are unified by the desire to make the lives of vascular patients better, to relieve their pain and to save life and limb. To do this we rely on the expertise of other professionals, our teams. It is therefore intentional that while this book has a surgical emphasis for Foundation doctors and medical students, it is also aimed at those professionals such as specialist nurses, surgical care practitioners and therapists without whom our work would be impossible and not half as rewarding.

The management of vascular disease and the typical vascular patient have changed over the years. We now deal with more elderly comorbid patients but have a wide array of endovascular treatments available. However, the basics of vascular surgical care remain rooted in compassion combined with scientific knowledge and technical skill.

Though the numbers of infrarenal aneurysms and carotid stenoses suitable for surgery may have decreased in some populations, the global prevalence and mortality associated with peripheral arterial disease has increased substantially. The scourge of diabetic vascular disease is upon us. Likewise, venous disease in all forms demands greater expertise and resources. Trauma and vascular injuries secondary to interventional medical procedures seem to increase annually. The need for enthusiastic, committed young people from all backgrounds to train in vascular surgery is clear.

If you are determined to follow another medical specialty, this book will be a good reference when you come up against a vascular problem. If you are thinking about a career in vascular surgery but are still not sure, we hope these chapters will kindle a love for it. If you are already drawn to this brilliant discipline then we hope you enjoy the book and will share it widely.

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OVERVIEW OF VASCULAR SURGERY

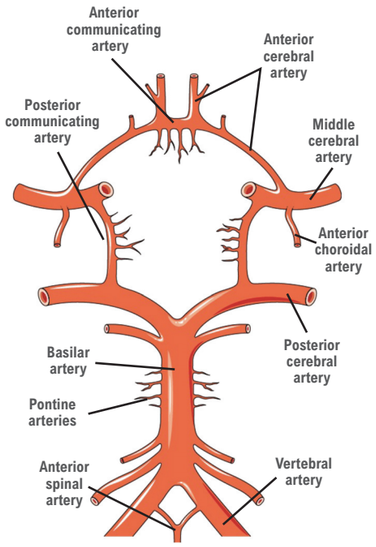
1 VASCULAR ANATOMY – CHAPTER 1

Than Dar, Rob Sayers

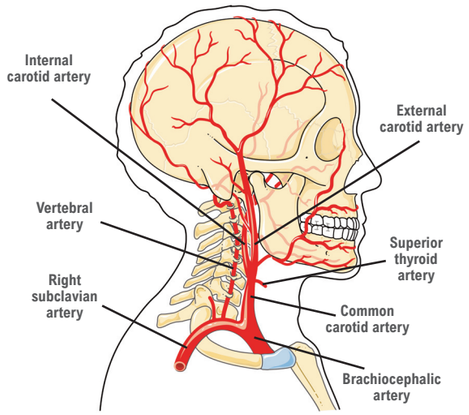
Vascular surgeons operate all over the body, therefore detailed knowledge of the vascular anatomy and surrounding structures is important in understanding vascular pathologies, procedures, and complications.

Head

- Blood supply to the brain arises from the internal carotid and vertebral arteries. The internal carotid arteries are divisions of the common carotid arteries.
- The internal carotid artery enters the cranial cavity through the carotid foramen and runs a tortuous intracranial course to bifurcate into its terminal branches, the anterior and middle cerebral arteries.
- The territory of the anterior cerebral artery includes the midline of the frontal lobe and superior and middle portions of the parietal lobe. A stroke in this artery commonly affects the contralateral lower limb.
- The territory of the middle cerebral artery includes the motor and somatosensory cortices. A stroke in this artery commonly produces such deficits in the face, trunk and upper limbs.
- The vertebral artery arises from the first part of the subclavian artery in the superior mediastinum. It ascends through the transverse foramina of the cervical vertebrae to enter the cranial cavity through the foramen magnum. It goes on to supply the posterior part of the brain, cerebellum, brainstem and spinal cord. Features of a stroke in this artery include ataxia, dizziness and nystagmus.



- The vertebral arteries form an anastomotic connection with the internal carotid arteries at the base of the brain, known as the Circle of Willis. This rich anastomosis between the anterior and posterior circulations to the brain allows cerebral perfusion to be preserved if an artery becomes occluded.

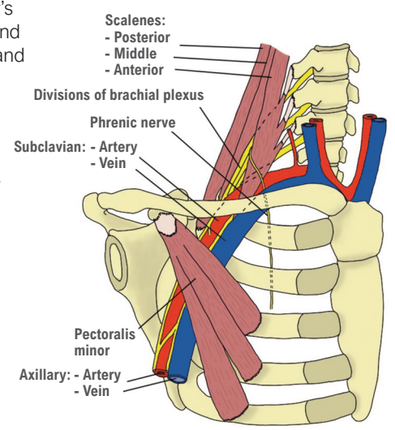


Neck

- It is important to be aware of the many major neurovascular structures in the neck that will be encountered specifically during a carotid endarterectomy.
- The carotid artery lies within the carotid sheath alongside the internal jugular vein and vagus nerve in the anterior triangle of the neck which is bound posteriorly by the anterior border of the sternocleidomastoid, anteriorly by the midline of the neck and superiorly by the mandible.
- The right common carotid artery arises from the brachiocephalic artery. The left common carotid artery arises directly from the aortic arch. In some cases, it can arise from the brachiocephalic artery, which is referred to as a bovine arch.
- The common carotid arteries bifurcate at C3-4 level into the internal and external carotid arteries. The internal carotid artery has no branches in the neck. The external carotid artery gives off eight branches that primarily supply the face.
- The internal jugular vein exits the cranial cavity through the jugular foramen to descend in the carotid sheath.
- Several cranial nerves are closely related to the carotid arteries during their course.
- Damage to cranial nerve IX (glossopharyngeal) produces impairment in the gag reflex and swallowing. Damage to cranial nerve XII (hypoglossal), which innervates the muscles of the tongue, produces tongue deviation towards the side of the lesion.
- Cranial nerve X (vagus) gives off branches to the pharyngeal plexus that innervate the pharyngeal constrictors for swallowing. The muscles of the larynx are supplied by the recurrent laryngeal except cricothyroid, which is innervated by the external branch of the superior laryngeal nerve. A superior laryngeal nerve palsy produces a hoarse voice, reduced vocal frequency range, and increase risk of aspiration.
- The ansa cervicalis is formed by the C1-3 nerve roots. It innervates the infrahyoid muscles involved in speech and swallowing. It can sit on top of the carotid artery and may need to be divided to facilitate carotid surgery.

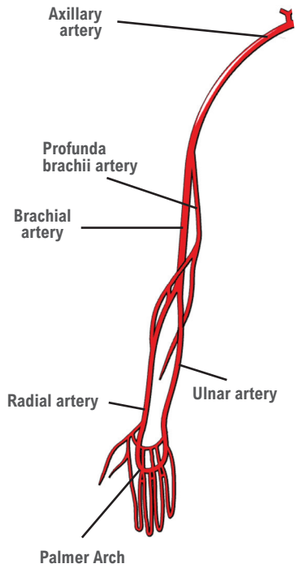
Thorax

- The sympathetic chain runs along the length of the vertebral column and consists of pre- and post-ganglionic fibres and sympathetic ganglia. It is a fundamental part of the sympathetic nervous system, and allows nerve fibres to travel to spinal nerves that are superior and inferior to the one in which they originated.
- The stellate ganglion lies on the neck of the first rib at the thoracic inlet. It provides the majority of the sympathetic nerve signals to the head, neck, arms and aspects of the upper chest. During endoscopic thoracic sympathectomy, a treatment to manage hyperhidrosis, the sympathetic trunk must be divided below the ganglion to avoid Horner's syndrome, recognised as a triad of ptosis, miosis, and ipsilateral facial anhidrosis. The aortic arch begins and ends at the angle of Louis which lies at the T4/5 vertebral disc level. Its three main branches are the brachiocephalic, left common carotid, and left subclavian arteries.
- It continues as the descending thoracic aorta in the posterior mediastinum giving off the bronchial, mediastinal, oesophageal, pericardial, superior phrenic, intercostal and subcostal arteries.
- The brachial plexus and subclavian vessels can be compressed when they exit the thorax to cause thoracic outlet syndrome. This can be due to compression from the anterior scalene muscle, a cervical rib or due to compression between the clavicle and the first rib. Features include upper limb paraesthesia, claudication and weakness.



Upper limb

- Knowledge of the anatomy of the upper limb is helpful to appreciate the steps in procedures such as embolectomies and formation of renal dialysis fistulas.
- The axilla acts as a conduit for neurovascular structures to enter the upper limb. It contains the axillary artery, vein, lymph nodes and the brachial plexus.
- The axillary vein lies medial to the artery. It is formed by the basilic and brachial veins.



- The axillary artery is a continuation of the subclavian artery beginning at the outer border of the first rib. It is divided into three parts based on its relation to pectoralis minor.
- In the arm, it continues as the brachial artery at the inferior border of teres major. One of the branches is profunda brachii which runs with the radial nerve in the spinal groove of the humerus.
- The cubital fossa is a triangular region in the elbow. It is bound laterally by brachioradialis and medially by pronator teres to form a distal apex. The base goes across the two humeral epicondyles. Beneath the deep fascia of the forearm, the following structures can be seen from lateral to medial: bicipital tendon, brachial artery and median nerve.
- The brachial artery divides into the radial artery laterally and ulnar artery medially. They continue in the forearm to form the deep and superficial palmar arches that supply the hand.
- The main superficial veins in the upper limb are the basilic and cephalic veins which can be seen on the ulnar and radial aspects of the forearm respectively.

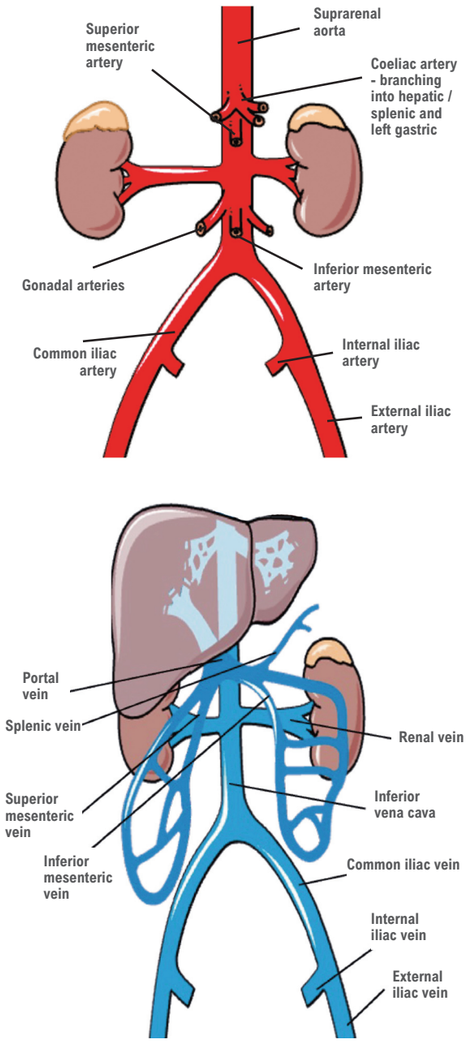
2 VASCULAR ANATOMY – CHAPTER 2

Harj Rayt, Than Dar[2]

Vascular surgeons operate all over the body, therefore detailed knowledge of the vascular anatomy and surrounding structures is important in understanding vascular pathologies, procedures, and complications.

Abdomen and pelvis

- The abdominal aorta is a retroperitoneal structure that begins at T12 as it descends from the diaphragmatic crura to L4, where it bifurcates into the common iliac arteries.
- The abdominal aorta gives rise to paired visceral, unpaired visceral, and parietal branches.
- The coeliac trunk arises at T12 to supply the foregut from the lower third of the oesophagus to the second part of the duodenum. It gives off three branches: the left gastric, splenic, and common hepatic arteries.
- The superior mesenteric artery arises at L1 to supply the midgut from the second part of the duodenum to two thirds along the transverse colon.
- The inferior mesenteric artery arises at L3 to supply the hindgut from the distal third of the transverse colon to two thirds along the rectum.
- The common iliac arteries bifurcate at the sacroiliac joints to become the internal and external iliac arteries.
- Change text to: The inferior vena cava begins at L5 where the common iliac veins unite. It ascends in the abdomen to the right of the abdominal aorta to enter the diaphragm at T8.
- The renal vein on the left is longer than the right, generally drapes over the aorta and receives drainage from multiple tributaries including the left gonadal vein, whereas the right gonadal vein drains directly into the inferior vena cava.



Clinical notes

- **May-Thurner syndrome**

  This is characterised by the presence of an anatomical variant in which the right common iliac artery compresses the left common iliac vein against the lumbar spine. The resultant reduction in blood flow increases the risk of deep vein thrombosis.

- **Nutcracker syndrome**

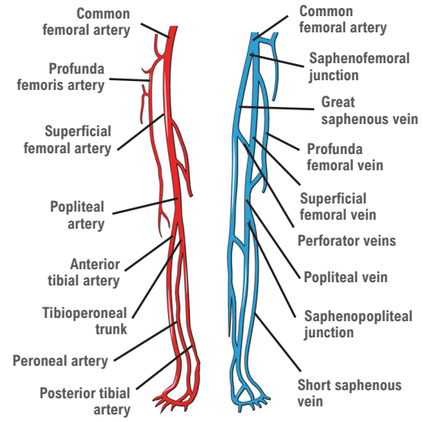
  This is characterised by compression of the left renal vein, typically between the abdominal aorta and superior mesenteric artery. Clinical symptoms of this syndrome can include haematuria and abdominal or flank pain.

- **Varicocele**

  This is a scrotal swelling due to dilated veins in the pampiniform plexus. As the left testicular vein drains into the left renal vein, a left-sided varicocele should raise suspicion of left-sided renal cell carcinoma as it can spread along the course of the renal vein.

Lower limb

- The external iliac artery continues as the common femoral artery in the femoral triangle, which is bound superiorly by the inguinal ligament, laterally by sartorius, and medially by adductor longus.
- The common femoral artery branches into the profunda femoris and superficial femoral arteries. The superficial femoral artery continues its course anteromedially in the subsartorial (adductor) canal. It emerges at the adductor hiatus of adductor magnus to become the popliteal artery.
- The popliteal fossa is a diamond shaped space behind the knee. It is bound by the hamstrings superiorly and the two heads of gastrocnemius inferiorly. Its contents include the popliteal vein and artery, tibial, sural, and common peroneal nerves, and lymph nodes.



- At the inferior border of popliteus, the popliteal artery divides into the anterior tibial artery and tibioperoneal trunk. The anterior tibial artery passes through the interosseous membrane between the tibia and fibula IN THE ANTERIOR COMPARTMENT down the leg. It crosses the ankle joint to become the dorsalis pedis artery, which lies lateral to the extensor hallucis longus tendon. It joins the lateral plantar artery in the foot to complete the plantar arch.
- The tibioperoneal trunk divides into the posterior tibial and peroneal arteries. The posterior tibial artery descends deep to gastrocnemius and soleus IN THE POSTERIOR COMPARTMENT. It passes behind to the medial malleolus through the tarsal tunnel before dividing into the medial and lateral plantar arteries. The peroneal artery passes behind the fibula and descends IN THE LATERAL COMPARTMENT of the leg.
- The main superficial veins in the lower limb are the great and small saphenous veins.
- The great saphenous vein runs anterior to the medial malleolus. It ascends medially up the lower limb, close to the saphenous nerve, to drain into the femoral vein in the groin. The small saphenous vein runs posterior to the lateral malleolus. It ascends posteriorly up the leg, close to the sural nerve, to drain into the popliteal vein.
- The deep veins follow the course of the major arteries in the lower limb, for example the femoral vein and artery accompany each other in the femoral sheath. In the presence of deep vein thrombosis, flow can be directed into the superficial veins via the perforating veins.

Clinical notes

- **Surface landmarks**

  The common femoral artery lies at the mid-inguinal point, halfway between the anterior superior iliac spine and the pubic symphysis. This is important in locating the common femoral artery for endarterectomies or endovascular procedures.

- **Acute compartment syndrome**

  Intermuscular septa divide the leg into four compartments: anterior, lateral, superficial posterior, and deep posterior. The swelling or bleeding that ensues after trauma or ischaemia increases the pressure within these compartments, which can lead to compression of their contents. This is a limb threatening condition that requires emergency four compartment fasciotomy to relieve the pressure.

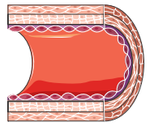
3 BIOLOGY OF VASCULAR DISEASE

Fabio Stocco, Marc A Bailey

WHAT CAUSES VASCULAR DISEASE?

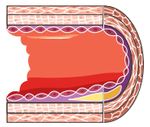
- Vascular disease encompasses pathology of the circulatory system (blood vessels) and the consequences of coagulation. This includes diseases of arteries (aorta, peripheral, or carotid), veins, lymphatic channels and both arterial and venous thrombosis / embolism.
- Common risk factors for vascular disease are genetics, modifiable factors (e.g., smoking, high cholesterol, high blood glucose, high blood pressure), trauma or infections.
- The most common cause of vascular disease is atherosclerosis, which describes the build-up of a cholesterol-laden fatty plaque inside the artery wall causing narrowing of the lumen and reduced blood supply to target organs – in the leg arteries this causes intermittent claudication.

ATHEROSCLEROSIS

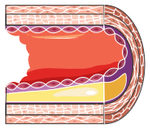


Several factors drive chronic endothelial injury that triggers formation of atherosclerosis:

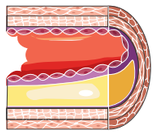
• Hyperlipidaemia • Hypertension • Haemodynamics • Smoking
• Toxins • Viruses • Immunity



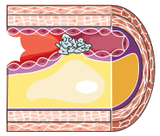
Endothelial injury results in endothelial dysfunction and increased permeability. Monocytes adhere to the dysfunctional endothelium and migrate into the intima. They differentiate into macrophages.



Within the intima macrophages are activated. Vascular smooth muscle cells (VSMC) are recruited by factors released from activated platelets, macrophages and the generally inflamed vascular wall milieu.

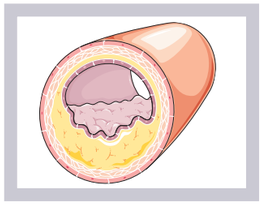


The VMSC de-differentiate and adopt a macrophage like phenotype. These activated cells and invading macrophages engulf cholesterol (foam cells) releasing further inflammatory cytokines; a positive feedback loop.



The VSMC proliferate and synthesise collagen and abnormal extracellular matrix. Extracellular lipids are engulfed. The lesion grows with a lipid rich core and a protective fibrous cap made of VSMC. Arterial thrombosis occures when there is rupture of the fibrous cap

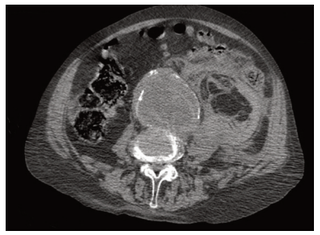
Peripheral arterial disease



- Atherosclerotic plaque causes arterial lumen narrowing and reduced end organ perfusion. An example of this is in peripheral arterial disease (PAD): this causes reduced blood flow to the limbs, most commonly the legs, which can cause intermittent claudication or chronic limb threatening ischaemia.
- Acute thrombosis occurs where there is disruption of the plaque fibrous cap exposing its thrombogenic lipid core resulting in the development of intraluminal thrombosis. This can either occlude the lumen (commonly a cause of acute myocardial infarction) or result in embolic disease more distally (a common cause of stroke).
- An embolism is a blockage of a blood vessel caused by material that has travelled from a more distant source. This is usually a blood product but can also be caused by fat / air or amniotic fluid. The most common source is cardiogenic (e.g. atrial fibrillation [AF] or left ventricle [LV] thrombus) or peripheral (e.g. unstable plaque or aneurysmal mural thrombus). In the venous circulation it can cause a pulmonary embolism and the common source is a DVT.

Aneurysmal development

Aneurysm: Defined as a progressive, focal, permanent dilatation of an artery (>1.5x normal). Clinically sequelae are rupture with a high associated mortality rate or less commonly thrombosis or embolism.



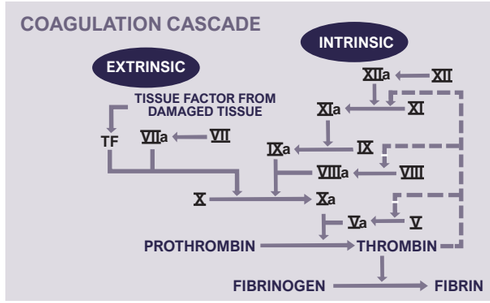
In genetically susceptible patients, environmental factors such as smoking, hypertension and atherosclerosis cause a dysfunction and weakening of the aortic wall.

Elastin and collagen are lost and the media degenerates. The wall loses tensile strength and dilates forming an aneurysm (aorta >3 cm). AAA grow at ~2 mm per year. As size increases so does the wall shear stress and eventually the aorta will rupture. Elective repair is therefore offered when the AAA is large (>5.5 cm) to remove the risk of rupture.

Thrombosis

Thrombosis describes the clotting of blood in the arterial or venous circulation. It can be triggered by (i) hypercoagulability, (ii) vascular wall pathology or (iii) altered blood flow (known as Virchow's triad).

Arterial injury (including surgery) or atherosclerotic plaque rupture can lead to exposure of subendothelial proteins: the von Willebrand factor binds to platelet glycoproteins leading to platelet aggregation and thrombus formation. Reduced blood flow due to lumen narrowing via atherosclerotic plaque, NIH or stasis in diseased veins is an important driver of thrombosis in vascular surgery. Genetic thrombophilia (e.g. Factor V Leiden) increases the risks of thrombosis.



Neointimal hyperplasia (NIH)

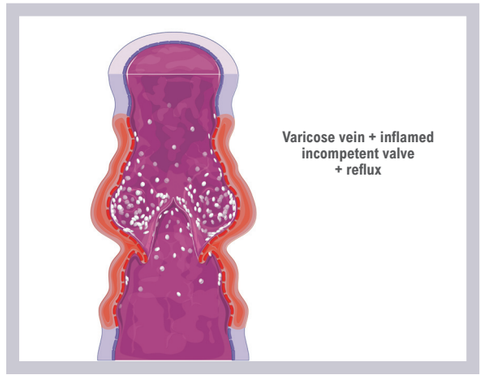
Following revascularisation (e.g. angioplasty, bypass graft), treatment failure is a major clinical problem. NIH describes the unwanted pathological vascular remodelling response following revascularisation, leading to re-stenosis and eventually occlusion.

Endothelial injury leads to stimulation of VSMC proliferation and migration. This causes release of inflammatory cytokines and synthesis of new extra-cellular matrix. This causes thickening of the intima, narrowing the lumen with a fibrous plaque in a similar way to atherosclerosis. The altered flow in the treated segment of artery (or bypass graft) increases the risk of thrombosis and graft occlusion / failure.

Venous disease

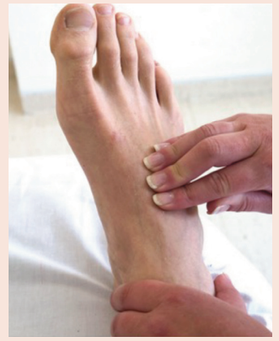
Chronic venous hypertension (CVH) is a common disorder of the lower extremities which can manifest as varicose (dilated and tortuous) veins or deep venous insufficiency. It is caused by dysfunctional valves causing venous reflux, increasing the venous pressure in the leg. Deep venous obstruction, secondary to venous thrombosis, can also cause CVH.

Venous reflux can be caused by dysfunction of pressure-sensing in the vein wall which leads to pathological vascular remodelling and inflammation.

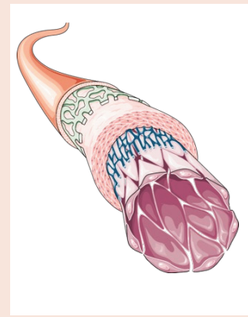


Further, following a deep vein thrombosis (DVT) the thrombus formation can damage the valves in the deep veins. The pooling of blood in abnormal superficial veins makes them prone to thrombosis: thrombophlebitis describes thrombosis and inflammation of superficial veins, whereas deep veins are subject to DVT that can lead to pulmonary embolism (PE), which can be so severe to be fatal.

BOX 1 THE STRUCTURE OF BLOOD VESSELS



1. **Tunica intima** (endothelium): the inner layer of the blood vessels is a single cell layer of endothelial cells that is anti-thrombitic and in direct contact with flowing blood. Implicated in vascular diseases.
2. **Tunica media** (middle or medial layer): primarily consists of vascular smooth muscle cells, elastin and collagen. The thickest layer and more prominent in arteries than veins. Provides support for the vessel, propagates the pulse wave, allows contraction / dilation to regulate blood flow and blood pressure. Implicated in vascular diseases.
3. **Tunica adventitia** (outermost layer): contains connective tissue with varying amounts of elastic and collagenous tissue. Mainly fibroblasts. Attaches the vessel to the surrounding tissue and provides general support.



4 VENOUS IMAGING

Mujahid Abdalla, Fatemeh Sakhinia

WHAT IS VENOUS IMAGING?

- Vascular surgeons commonly see patients with disorders of the veins.
- The majority of problems affect the veins in the legs but there are also recognised issues with the veins in the arms.
- Imaging is key to providing an accurate diagnosis of patients with venous disorders.

LOWER LIMB VENOUS IMAGING FOR DEEP AND SUPERFICIAL VENOUS DISEASE

Continuous-wave Doppler ultrasound

This is utilised as a non-invasive initial screening test for chronic disease of the superficial lower limb veins. It involves using a pencil ultrasound probe and is a relatively inexpensive examination. Acoustic signals are used to render venous blood flow and incompetence of the great saphenous vein can be detected. However, this method is less accurate in diagnosing incompetence of the lesser saphenous vein or the deep venous system due to anatomical variations and operator-dependent differences.

Colour-flow Duplex ultrasound

This is another non-invasive modality and is relatively simple to perform. It facilitates functional and morphological examination of both the deep and superficial venous system. In particular, deep vein thrombosis (DVT) of the lower limb can be diagnosed with high accuracy via dynamic venous compression using the ultrasound probe itself.

In addition, the dynamic assessment capabilities of ultrasound can aid in identification of the anatomical level of venous incompetence. This is done through utilisation of a tilt table (or standing the patient up) as well as manual calf compression by the operator(s) to induce venous reflux. Simultaneous venous waveforms traces can be rendered using the pulse-wave function on most modern ultrasound machines to register venous reflux digitally.

Venous duplex scanning of the popliteal fossa.

There are however diagnostic limitations with this modality in assessment of the pelvic veins due to their deep location. Also, in some cases, it is not possible to definitively assess the deep veins of the lower leg due to variations in individual patient anatomy.

Computed Tomography (CT) and Magnetic Resonance (MR) Venography

These modalities are most useful in the evaluation of the more proximal veins and their surrounding structures but can also depict abnormal superficial veins (varicose veins) relatively well. They allow for accurate assessment of intrinsic venous obstruction or extrinsic venous compression.

Optimal imaging with these modalities necessitates the injection of intravenous contrast material with appropriate timing of image acquisition to coincide with venous filling, in order to obtain a 'venogram'. Both CT and MR venography can be used to delineate complex venous anatomy, such as iliofemoral venous obstruction, prior to intervention.

Intravascular ultrasound (IVUS)

This technique involves the use of a catheter-based ultrasound probe to image peri-luminal vascular anatomy in order to detect stenotic or obstructive disease of the venous system.

IVUS appears to be superior to venography in the estimation of morphology and severity of central venous stenosis as well as in visualising detailed intraluminal anatomy. This superior detection of stenosis severity has resulted in increased venous percutaneous interventions for the treatment of chronic lower limb venous disease. In particular, IVUS has been shown to be ideal for identifying pelvic venous lesions (namely iliac) that are occult on conventional ultrasound or venography.

Conventional contrast venography

Contrast venography allows for direct visualisation of the venous system via either an ascending or descending approach.

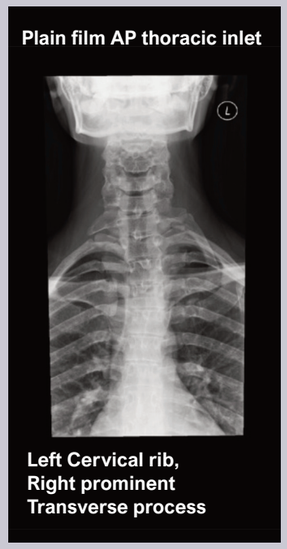
Ascending venography is defined as injection of contrast in the veins of the dorsum of the foot and subsequent visualization of contrast travelling cephalad in the deep venous system of the lower limb. This provides detailed imaging of venous anatomy that can help guide surgical interventions and can also aid in distinguishing primary from secondary disease.

Descending venography involves proximal injection of contrast with the patient in a semi-vertical position (using a tilt table) and requesting the patient to perform the Valsalva manoeuvre. It is most useful in identifying reflux in the common femoral vein and at the saphenofemoral junction, but may be used to assess other locations also.

UPPER LIMB VENOUS IMAGING

Radiography (conventional x-ray)

Anatomical abnormalities potentially causing thoracic outlet syndrome (TOS) such as prominent cervical ribs, fracture callouses or compressive tumours can often be demonstrable on chest, shoulder or spine radiographs.



Plain film AP thoracic inlet

Left Cervical rib, Right prominent Transverse process

Colour-flow Duplex ultrasound

In cases of suspected vascular TOS, colour-flow Duplex ultrasound is a highly sensitive and specific imaging modality. It is also non-invasive and inexpensive and is the initial imaging test of choice.

This technique permits functional and morphological examination of both the deep and superficial venous system. As in the lower limbs, DVT can similarly be diagnosed with high accuracy in the upper limbs via dynamic compression of the veins using the ultrasound probe. Extrinsic causes of upper limb venous compression from adjacent anatomical structures may also be diagnosed, especially when imaging with dynamic assessment, i.e. imaging in real time during active/passive movement of the upper limb.

Computed Tomography (CT) and Magnetic Resonance (MR) Venography

These modalities are useful in differentiation of equivocal cases of TOS and can provide additional anatomic detail required for surgical planning.

MR imaging is the preferred modality for investigation of suspected TOS and is particularly preferred to CT given its lack of ionising radiation (especially beneficial in the generally younger affected patient population). However, absolute or relative contra-indications to MR (e.g. incompatible implanted device, severe dialysis-dependent renal failure, claustrophobia) may preclude use of this modality. In such cases, CT with intravenous contrast is then the preferred imaging modality (see below) or time-of-flight non-contrast MR imaging.

MR imaging is acquired with gadolinium-based intravenous contrast material. The intravenous catheter should be placed on the asymptomatic arm in order to minimise T2* artefact caused by concentrated gadolinium contrast that may obscure the axillosubclavian vasculature on the symptomatic side. Several series of images are then performed with the upper limbs adducted and abducted to allow detection of positional stenoses of the vessels.

CT is excellent for assessment of bony anatomy and therefore detection of anomalous ribs or fractures that may predispose to TOS. The investigation of TOS with CT is typically acquired with intravenous iodinated contrast material and, as with MR imaging, the intravenous catheter should be placed on the side opposite to the symptoms in order to prevent streak artefact from dense contrast material.

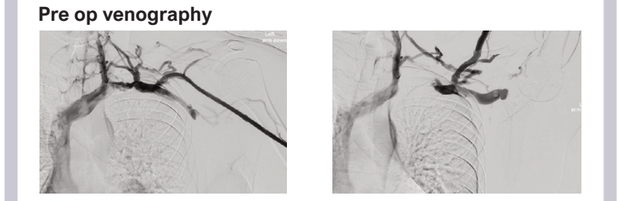
Intravascular ultrasound (IVUS)

IVUS allows for detection of stenotic or obstructive disease of the venous system.

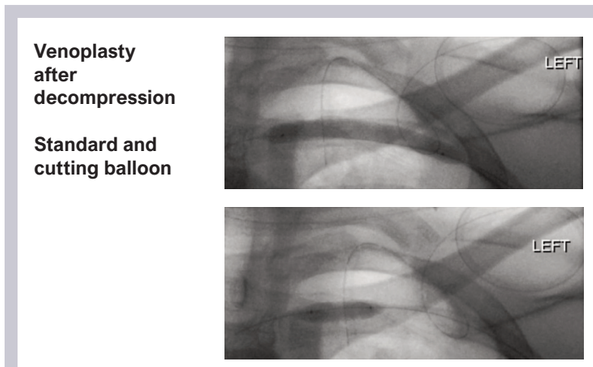
it probably has a similar sensitivity in identifying upper limb venous lesions that are occult on venography or conventional ultrasound.

Conventional contrast venography

These techniques are invasive and involve the injection of contrast material intra-arterially or intravenously in order to visualise vessels using x-rays via fluoroscopy. They can be used to demonstrate extrinsic compression of the upper limb veins. Venography is particularly useful in venous TOS when dynamic imaging is required to demonstrate occlusion of upper limb veins on hyperabduction and as part of intervention with lysis for acute DVT and post operative venoplasty.

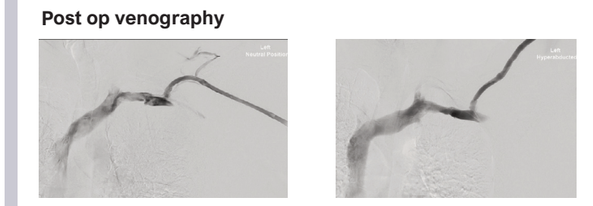


Pre op venography



Venoplasty after decompression

Standard and cutting balloon



Post op venography

5 ARTERIAL IMAGING

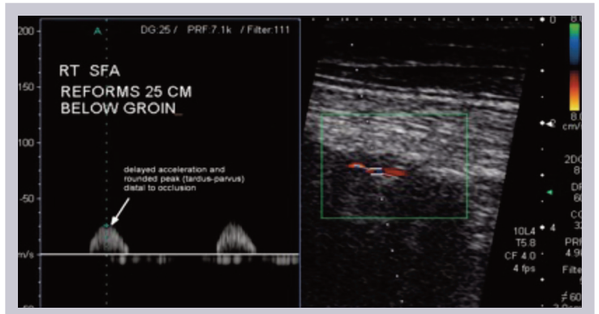
Patrick Coughlin

WHAT IS ARTERIAL IMAGING?

- Imaging of the arterial tree is key to confirming an accurate diagnosis and determining an optimal treatment strategy.
- The imaging strategy used will be determined by the access to the imaging modalities below and often more than one imaging modality may be used in patients.

Arterial Duplex

Duplex is a non-invasive ultrasound imaging modality technique that incorporates two modes of ultrasound - Doppler and B-mode. The B-mode aspect obtains an image of the vessel being studied with the Doppler aspect able to measure speed and blood flow.



Duplex is commonly the first line arterial investigation.

It is specifically used for:

- Assessment of carotid disease
- Investigation of upper and lower limb arterial disease – considered as first line imaging by NICE for lower limb PAD.
- It has significant benefits in those patients who require surveillance following intervention (e.g. post infrainguinal bypass surgery or stenting, surveillance of EVAR, etc) due to the repeated requirements of such investigations

There are a number of advantages to its use:

- The ultrasound machine is portable and the imaging modality is non-invasive
- It does not requires use of contrast / nephrotoxic agents
- The use of both Doppler and B-mode allows for a more precise estimation of the degree of significance of the arterial disease
- It allows for visualisation of the entire artery (including the wall) and not just the lumen, providing information on plaque characterisation
- It is low cost

There are some disadvantages to its use:

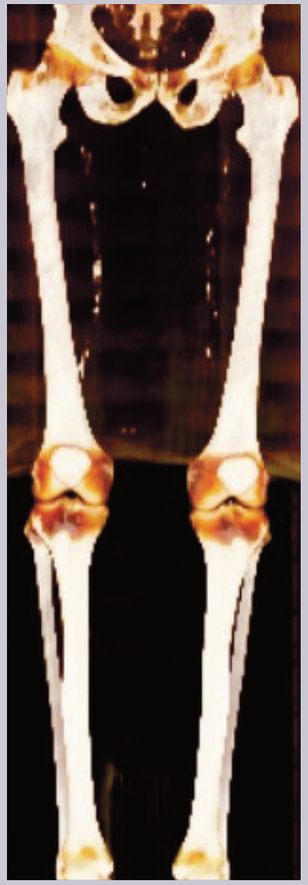
- Operator dependence with a learning curve associated with performing these scans
- Challenges in assessing intra-abdominal vessels, especially in more obese patients

CT angiography

CTa allows the cross-sectional imaging of arterial vessels throughout the body using the injection of contrast material to evaluate the arterial tree.

CTa has a number of advantages

- It is non-invasive
- It is rapid to perform and tends to be available 24 hours per day, 7 days per week in most hospitals.



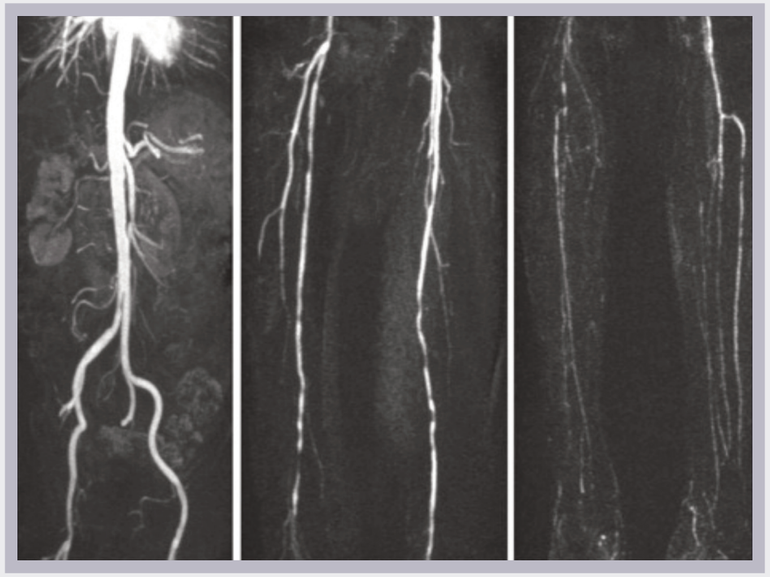
- It is not user dependent but appropriate protocols need to be in place for accurate image acquisition
- It is able to provide information on every artery within the body including those within the thorax and abdomen
- It provides precise anatomical detail including assessment of the lumen, arterial wall and extra-arterial anatomy

The disadvantages

- Exposure to radiation
- Small risk of contrast allergy
- Small risk of contrast nephropathy in patients with chronic kidney disease
- CT angiography may overcall the degree of stenosis in patients with lower limb PAD
- It can be challenging to accurately determine the presence and severity of atherosclerosis in the infrapopliteal arteries in patients with significant medial artery calcification (e.g. patients with diabetes mellitus and renal failure)

Magnetic Resonance Angiography (MRA)

MRA is a further imaging modality that can provide cross-sectional imaging of arteries within the body.

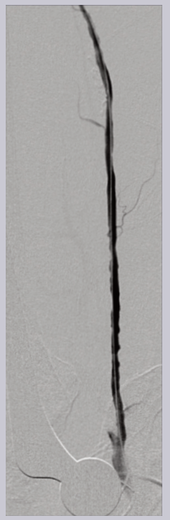


MRA has a number of advantages:

- It is non-invasive
- It does not require exposure to ionising radiation
- It is not user dependent but does require appropriate protocols for image acquisition
- It has the potential for non-contrast examination but commonly the use of contrast is still required

MRA does also have a number of disadvantages:

- It is not as quick as CT angiography for image acquisition
- There are a number of MRI contraindications around implanted medical devices – specifically pacemakers and defibrillators (although newer devices are increasingly becoming MRI compatible)
- MR angiography tends not to be available 24 hours per day / 7 days per week within the UK
- MRA may not be able to see and capture images of calcium deposits within the arterial tree



Intra-arterial digital subtraction angiography (IADSA)

IADSA is still deemed to be the “gold standard” method of angiography.

This involves direct puncture into an artery (usually the common femoral artery) and insertion of a sheath following this. IADSA is rarely used in isolation as a diagnostic imaging modality but most commonly as a bridge to some form of concomitant endovascular intervention. In most cases, duplex or cross-sectional angiography would provide the detail needed to plan an endovascular intervention.

IADSA is used diagnostically either to determine the quality of a distal target infrapopliteal artery when there is uncertainty of its quality to support a femoro-distal surgical bypass or to accurately determine the blood supply in the foot.

The advantages of IADSA are:

- Comprehensive evaluation of the lower limb PAD
- There are no real issues with medial artery calcification
- It is able to provide detailed anatomy of the arterial supply to the foot which can be important in patients with diabetes

The disadvantages of IADSA are:

- Contrast is required and so patients with CKD may be susceptible to contrast nephropathy
- The procedure is invasive with associated risks
- The procedure requires the use of radiation

6 STRATEGIES FOR REVASCULARISATION

Penelope PJ Sucharitkul, Christopher P Twine

WHAT IS REVASCULARISATION?

- Revascularisation is a surgical procedure which aims to restore blood flow to an area of the body where the arteries have been blocked, usually by atherosclerosis
- Restoring blood flow can prevent amputation and save lives as well as healing wounds, reducing pain and increasing quality of life
- This can be achieved with 'endovascular', 'open' or 'hybrid' surgical operations
- A patient's disease can often be treated by more than one operation type. Choosing the best option requires balancing a number of factors and careful shared decision making

Endovascular revascularisation

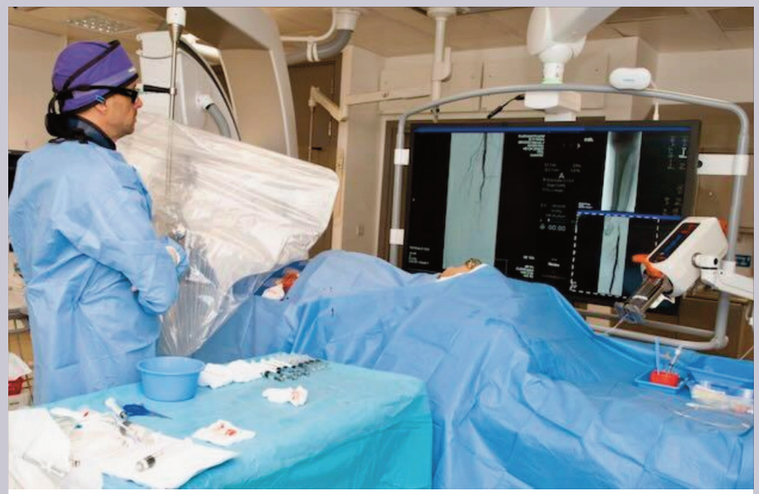
- 'Endo' comes from the Greek word, éndon, meaning within.
- A minimally invasive approach to revascularisation, 'Endovascular' refers to image guided surgery where a wire is threaded into the artery and through the blockage. Devices such as balloons and stents are passed over the wire and used to re-open the blocked or narrowed artery.
- An 'Angioplasty' is always performed during an endovascular intervention, this describes the process of opening up the blocked or narrowed artery.
- It is often performed under local anaesthetic, using ultrasound to access the artery with a needle.
- Multiple devices may be used for one endovascular intervention, the choice is different for each artery treated
- Not all patterns of disease are suitable for endovascular surgery but, as technology and devices advance, so do the indications.
- Generally, endovascular surgery is lower risk for the patient in the short term but may not be as durable as open or hybrid surgery in the long term.
- A more co-morbid patient (see Box 1) may benefit more from the lower risk nature of endovascular intervention.

BOX 1
Patient selection

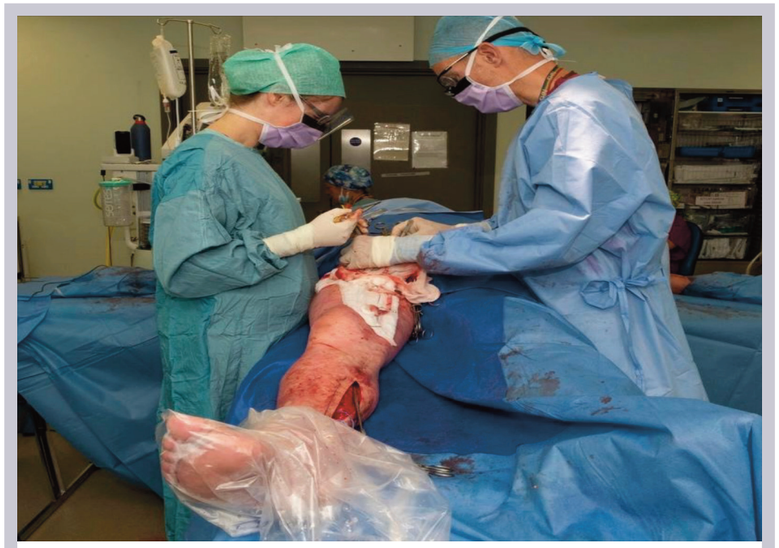
- Vascular patients are often elderly and co-morbid with other medical problems such as diabetes, previous myocardial infarction or stroke.
- Patients may experience various complications after a revascularisation operation (see Box 2).
- Endovascular surgery is generally lower risk than open or hybrid surgery. Depending on the artery affected and pattern of atherosclerosis, it may not be as durable as open surgery.
- Open surgery has higher short term risks to the patient. It may be more durable than endovascular or hybrid surgery in the long term.
- If a patient has a pattern of atherosclerosis suitable for endovascular or open surgery, many factors need to be considered to make the best decision on which option to choose.
- A more medically fit patient may benefit more from open surgery, whereas a more comorbid patient may benefit more from endovascular treatment. However, this varies depending on the artery.
- Patients undergoing arterial intervention will be discussed at a multi-disciplinary team meeting comprising surgeon, radiologists, anaesthetists and other team members because of the complex nature of the decision making.

Open revascularisation

- 'Open' refers to procedures where a surgical incision is made to access the artery.
- This requires large cuts in the skin and needs a general anaesthetic or 'block' such as a spinal anaesthetic.
- Less diseased arteries before and after the blockage are exposed and a tube or 'conduit' is sewn to each. The blood travels through the conduit and bypasses the blockage, thus restoring blood flow to the foot.
- The conduit is usually taken from the leg, where a healthy section of vein, for example the saphenous vein, is harvested for the bypass. This carries its own complications, such as swelling due to reduced venous outflow.
- All patterns of disease may be treated by open surgery, so patient selection (see Box 1) and shared decision-making understanding the benefits and risks is very important.
- Open revascularisation procedures are higher risk for the patient in the short term but may be more durable in the long term.
- Patients who are less comorbid may be suitable for open surgery (see Box 1), while those with multiple comorbidities may be better suited to endovascular treatment.



Endovascular intervention in action in the IR Suite.



Surgeons undertaking a lower limb bypass.

BOX 2
Complications

Vascular surgery comes with risks, and highly co-morbid patients

- **Site infection** – open surgery carries higher risks of post-operative infection.
- **Bleeding** – arterial bleeding in the leg commonly requires further surgical intervention.
- **Occlusion** – clot in bypass and risks of having to re-intervene.
- **Cardiovascular events** – patients are at high risk of myocardial infarction and must be commenced on best medical management (smoking cessation, diabetic control, high intensity statin therapy, antihypertensive and antiplatelet therapies) to try and mitigate this risk.
- **Hospital associated infections** – long stays in hospital can result in exposure to resistant organisms.
- **Amputation** – occlusion of the bypass may result in amputation.
- **Death** – mortality in this cohort remains high, despite intervention.

Support for patients

The Circulation Foundation is a UK based vascular charity which provides information and support for patients with vascular diseases, https://www.circulationfoundation.org.uk/

Hybrid revascularisation

- Hybrid procedures combine an open operation with an endovascular operation.
- A theatre with suitable imaging and operative capacity is needed to perform a hybrid operation.
- X ray equipment is needed to perform a hybrid procedure. This is operated by a radiographer, emphasising the need for a team approach.
- These procedures were previously conducted via an open approach, or with separate endovascular and open components, but with new imaging equipment and theatres, these can now be performed with reduced invasiveness for the patient.
- A common hybrid operation is a common femoral endarterectomy with iliac stenting where blockages in the common femoral artery are removed using open surgery, then the blood flow to this area from the aorta is opened with balloons and stents.

THEME 2

PERIPHERAL ARTERIAL DISEASE AND LOWER LIMB DISEASE

7 BEST MEDICAL THERAPY

Natalie Yonan, Claire Dawkins

WHAT IS BEST MEDICAL THERAPY?

To reduce progression of PAD but also morbidity related to cardiovascular disease, particularly cerebral and cardiac ischaemic events, all patients diagnosed with PAD should be initiated on:

- Antiplatelet therapy – usually clopidogrel 75mg
- Statin – usually atorvastatin 80mg

There should also be optimisation of hypertension and diabetes management.

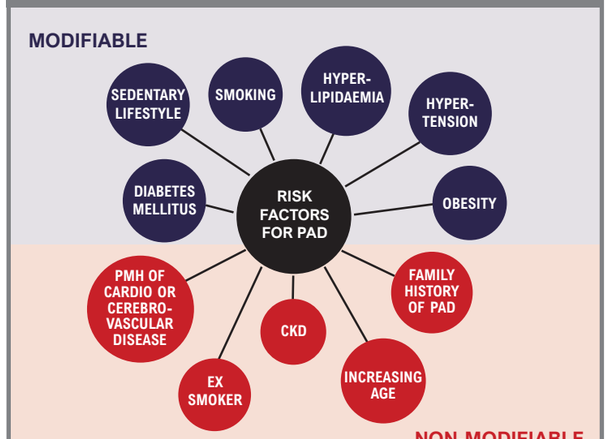
Alongside medical management it is important to give appropriate lifestyle advice:

- Smoking cessation – through counselling, medications such as nicotine replacement treatment, and smoking cessation programmes.
- Encourage exercise (including through supervised exercise programmes), healthy diet and weight loss.

WHY IS BEST MEDICAL THERAPY IMPORTANT?

Peripheral arterial disease (PAD) is most commonly caused by atherosclerosis, the causative factor also in coronary and cerebrovascular disease. Patients with PAD have a higher risk of having a stroke or a myocardial infarction, with a 2-3 times risk of cardiovascular mortality compared with the control population. Optimising cardiovascular risk factors is therefore paramount in patients with PAD to reduce cardiovascular morbidity and mortality.

RISK FACTORS FOR PERIPHERAL ARTERIAL DISEASE, SPLIT INTO MODIFIABLE AND NON-MODIFIABLE



Lipid modification

Dyslipidaemia is a strong risk factor for the development of PAD alongside cardiovascular disease. First line lipid modification therapy of choice is **80mg Atorvastatin daily**.

In patients who have cholesterol levels within the normal range, there is still benefit to statin therapy with further reduction of lipid levels and the anti-inflammatory effect of statins.

If statins are contraindicated then alternative treatment can be considered, for example, ezetimibe in primary hypercholesterolaemia.

MECHANISM OF ACTION - Statins

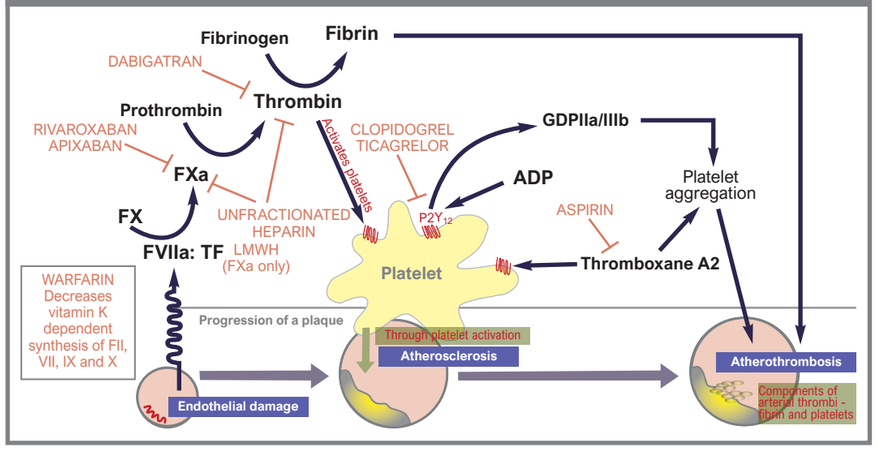
Statins are competitive inhibitors of 3-hydroxy-3-methylglutaryl coenzyme A (HMG-CoA), which is a step in the synthesis of cholesterol.

- This impacts lipid metabolism, and importantly reduces low-density lipoprotein cholesterol (LPL-C) levels.
- There is also an associated anti-inflammatory response, thought to be due to their inhibition of isoprenoids, which helps with plaque stabilisation

Important recommendations when initiating a statin:

- Measure non-fasting lipid profile prior to starting. Repeated at 3 and 12 months, with the aim to lower non-HDL-C by >40%.
- Measure non-fasting lipid profile prior to starting. Repeated at 3 and 12 months, with the aim to lower non-HDL-C by >40%.
- Important drug interaction: Clindamycin. Statins should always be stopped prior to commencement.

MECHANISM OF ACTION OF COMMON ANTIPLATELETS AND ANTICOAGULANTS FOR TREATMENT OF ATHEROSCLEROTIC DISEASE



MECHANISM OF ACTION - Clopidogrel

Clopidogrel is an inhibitor of the adenosine diphosphate (ADP) P2Y12 platelet receptor, which inhibits platelet aggregation. This is an irreversible process, thus platelets are affected for their lifespan, approximately 7 days.

Antiplatelet therapy and anticoagulation

Patients with PAD should be commenced on antiplatelet therapy. **Clopidogrel 75 mg daily is the antiplatelet of choice in peripheral arterial disease** with evidence (CAPRIE trial) demonstrating a greater relative risk reduction for major vascular events in clopidogrel when compared with aspirin.

Proton pump inhibitors (PPI) are often prescribed for gastro-protection; however, caution is needed with omeprazole and esomeprazole due to interactions with clopidogrel.

Evidence (VOYAGER PAD, COMPASS) has shown low dose rivaroxaban (2.5 mg) with aspirin (75 mg) is of benefit in patients with peripheral arterial disease and stable cardiovascular disease. In practice this may be used for high risk patients under specialist vascular input and bleeding risk must be considered.

Anticoagulation is commonly used for patients with embolic or thrombotic events. Intravenous heparin is often used in the short term for its immediate action, ease of control and reversibility. This is often changed to Low Molecular Weight Heparin (LMWH) after 24-48 hours to reduce the requirement for monitoring and improve flexibility. Longer term warfarin or DOACs can provide appropriate anticoagulation orally, with DOACs being many patients' choice although these are used off-label for the treatment of arterial thrombus.

Management of diabetes

Close diabetes management, for both type 1 (T1DM) and type 2 (T2DM) diabetes mellitus, with tight glycaemic control reduces the risk of macro and microvascular complications.

The aim of treatment is usually a HbA_{1c} <48mmol/L (6.5%) for both T1DM and T2DM. For those with T2DM, this is initially through lifestyle advice, but often requires medication.

Anti-diabetic drugs:

- Metformin – first line for T2DM
- Combination therapy – DPP-4 inhibitors, pioglitazone, sulfonylurea, SGLT-2 inhibitors are all options and escalate from dual to triple therapy
- Insulin – only treatment for T1DM, and for poorly controlled T2DM following initial therapies.

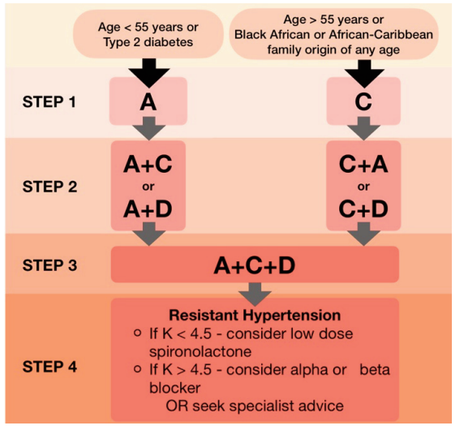
All patients with PAD should be screened for diabetes, initially with HbA_{1c} measurement.

Management of hypertension

Hypertension, with resultant increased pressure and damage to vessel walls, is another risk factor for PAD. NICE guidelines recommend a target blood pressure of <140/90 mmHg (<150/90 if over 80 years of age). The recommended approach is shown in Figure 1.

There is discussion regarding use of ACE inhibitors in the absence of hypertension, for reduction of cardiovascular disease; however, there are no current guidelines recommending this.

FIGURE 1: SUMMARY OF MANAGEMENT OF HYPERTENSION (NICE)



A = Angiotensin converting enzyme inhibitor or Angiotensin receptor blocker, C = Calcium channel blocker, D = Thiazide like diuretic

https://www.nice.org.uk/guidance/ng136/resources/visual-summary-pdf-6899919517

Symptomatic relief

In patients with PAD where revascularisation is not possible or ill-advised, vasodilators may offer some improvement to select patients. **Iloprost** infusions can offer some improvement in patients with unmanageable symptoms. **Naftidrofuryl oxylate** can be used for a 3-6 month trial in patients with claudication. Treatment should be discontinued if no improvement is seen. Cilostazole, Pentoxiphylline and Inositol nicotinate are not recommended.

8 INTERMITTENT CLAUDICATION

Paddy Coughlin

WHAT IS INTERMITTENT CLAUDICATION (IC)?

- The classical symptom of IC is that of muscular pain, usually cramp-like in nature, that is precipitated by walking and relieved by rest. The pain usually comes on quicker if walking up hill
- The muscle group(s) affected are those downstream of the significant arterial disease. As the superficial femoral artery is the most common site of significant PAD, the calf is the usual symptomatic muscle. The thigh (and calf) can be affected when the arterial disease is proximal to the profunda femoris artery and the buttock affected when the disease occurs proximal to the internal iliac artery. If there is significant aortic disease then both legs can be affected and in men this can also cause impotence (Leriche's syndrome)
- Pain results from ischaemic neuropathy involving small unmyelinated sensory fibres (types A delta and C) and from local intramuscular acidosis from anaerobic metabolism (lactate buildup)

How to diagnose intermittent claudication

History

- Muscular pain brought on by walking and relieved by pain. The initial claudication and maximal walking distances tend to be very reproducible

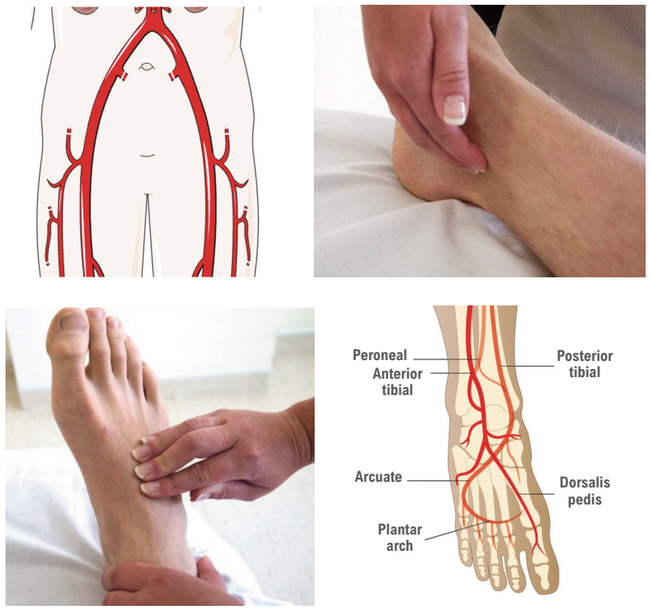
Examination

- Assessment of lower limb pulses (Figure 1).
- Ankle brachial pressure index (ABPI) measurement (Figure 2)

Imaging

- Imaging should only be undertaken if uncertainty of diagnosis or if planning for revascularisation. Its aim is to provide a "roadmap" of the site and severity of the lower limb atherosclerosis
- First line arterial imaging is usually an arterial duplex (combination of doppler and ultrasound) with CT or MR angiography useful cross-sectional imaging when planning revascularisation

FIGURE 1: ASSESSMENT OF LOWER LIMB PULSES



The femoral pulse is located – It can be palpated midway between the anterior superior iliac spine and pubic symphysis, just inferior to the inguinal ligament

ABPI = Highest ankle pressure

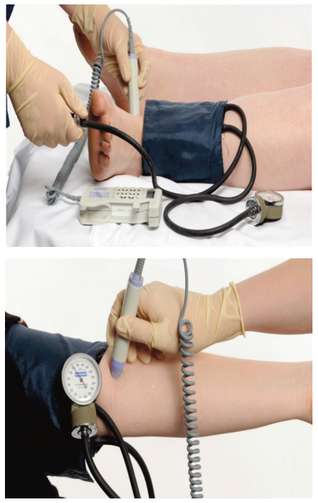
Higher arm systolic pressure

ABPI: 0.9-1.4. Normal value. Pressure usually higher in arm than leg.

ABPI <0.9: Diagnostic for PAD. If value is <0.5 this is suggestive of severe PAD.

ABPI >1.4: suggestive of a non-compressible vessel. This is commonly found in patients with diabetes mellitus and / or renal failure and is caused by calcification in the wall of the artery which makes it resistant to collapse by the blood pressure cuff.

FIGURE 2: ANKLE BRACHIAL PRESSURE INDEX (ABPI) MEASUREMENT



What is the differential diagnosis?

Exertional leg pain is not uncommon. Other conditions to consider are:

- Spinal stenosis: the neurogenic claudication symptoms are usually caused by ischaemia or mechanical compression of nerve roots. Pain may not only be present on walking but also on standing and on upright exercises. A key discriminator is its relationship to posture with lumbar flexion reducing the pain (e.g. leaning forward or lying down)
- Entrapment syndromes: primarily popliteal artery entrapment syndrome where the popliteal artery undergoes muscular compression during exercise. This is usually caused by an aberrant gastrocnemius anatomy and is usually seen in younger patients (<40 years of age).
- Compartment syndrome: this condition occurs due to a marked increase in tissue pressure within the confinement of a closed fascial space during exercise – commonly the calf. It tends to present in athletes. Other symptoms may include numbness / tingling in the dermatomal distribution of the nerve running through the compartment or weakness of the affected muscle.
- Venous claudication: thought to be an exercise induced pain resulting from venous outflow impairment which leads to an intense 'squeezing' type pain throughout the affected leg. There is usually a history of previous deep vein thrombosis (commonly of the iliofemoral veins).
- Osteoarthritis

Management of intermittent claudication

- Optimal management of cardiovascular risk to reduce overall cardiovascular morbidity and mortality (see chapter 7).
- The natural history of the leg in intermittent claudication is relatively benign (75% of patients with symptoms either staying the same or improving their walking distance) and this should influence the management of such patients. The indication for treatment is whether the symptoms negatively affect the patient's quality of life.
- Supervised Exercise Programme (SEP) – see Box 1
- Pharmacotherapy
  - NICE guideline suggests consideration of naftirorfuryl oxalate only when SEP has not led to satisfactory improvement and the patients prefers not to undergo revascularisation. Naftidrofuryl acts as a vasodilator. Review the clinical benefit after 3–6 months and stop the medication if no benefit.
- Revascularisation strategies:
  - Aorto-iliac disease

    In the case of short stenosis/occlusion (<5 cm) of iliac arteries, endovascular therapy gives good long-term patency with a low risk of complications.

    In cases of ilio-femoral lesions, a hybrid procedure is indicated, usually endart-erectomy or bypass at the femoral level combined with endovascular therapy of iliac arteries, even with long occlusions.

    If the occlusion extends to the infra-renal aorta, covered endovascular reconstruction of an aortic bifurcation can be considered. Open surgery (aortobifemoral grafting) can also be considered.
  - Femoro-popliteal disease

    If revascularisation is needed and there is stenotic disease or a short occlusion, then endovascular revascularisation is first line – usually with balloon angioplasty.

    For more extensive atherosclerotic disease then, where possible, a surgical bypass with autologous vein is preferable, although in higher risk patients then an endovascular approach may be appropriate.

BOX 1

Supervised Exercise Programme

For IC, enrolment in a supervised exercise programme (SEP) is effective and improves symptoms, increases walking distance and improves quality of life. It has also been proven to be cost-effective. The ideal SEP takes place 3 times a week for a minimum of 3 months duration. Exercise programmes commonly use a mixture of walking and lower limb strength exercises.

SEP is more effective than unsupervised exercise programme. However, in the UK, there are a lack of appropriate numbers of SEP for IC. As such, although home-based walking therapy is not as effective as SEP, it is a useful alternative when compared to walking advice alone.

Alternative exercise therapies including cycling / strength training / upper arm ergometry may be an alternative when walking therapies are not an option for patients.

ABPI VALUES AND MANAGEMENT

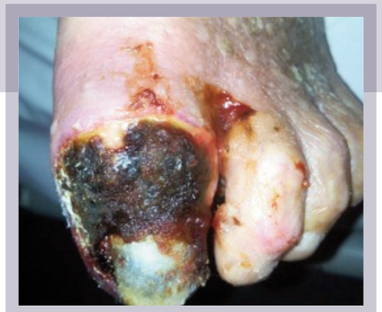
ABPI value	Interpretation	Recommendation
>1.4	Arterial calcification	Manage claudication in primary care initially. If foot / leg wounds or evidence of CLTI needs urgent referral to vascular specialist.
0.9 – 1.4	Normal	Nil
0.8 – 0.9	Some minor arterial disease	Risk factor modification. Manage claudication in primary care initially. Patients are able to have compression bandaging therapy with these ASBPI readings.
0.5 – 0.8	Moderate arterial disease	Risk factor modification. Manage claudication in primary care initially. If foot / leg wounds or evidence of CLTI needs urgent referral to vascular specialist. Patient should not have compression therapy applied with these ABPI readings.
<0.5	Severe arterial disease	Risk factor modification. Manage claudication in primary care initially. If foot / leg wounds or evidence of CLTI needs urgent referral to vascular specialist. Patient should not have compression therapy applied with these ABPI readings.

9 CHRONIC LIMB THREATENING ISCHAEMIA

Kaji Sritharan, Georgios Koufopoulos

WHAT IS CHRONIC LIMB THREATENING ISCHAEMIA?

- Chronic Limb Threatening Ischaemia (CLTI) is the advanced stage of peripheral arterial disease (PAD). It occurs due to the presence of severe PAD, whereby the blood supply to the foot is insufficient for the needs of the tissues. This results in a combination of rest pain, gangrene or lower limb ulceration which is present for a duration of 2 or more weeks; and associated with one or more haemodynamic abnormalities.
- Rest pain is discomfort within the forefoot which typically occurs at night when the foot is elevated in the bed. The pain will often wake the patient from their sleep and is relieved by hanging the affected limb off the bed. This is because gravity helps blood flow to return to the ischaemic leg.



How to diagnose CLTI

History

- Pain in the feet which disrupts sleep and is relieved by hanging the foot from the bed.
- Ulceration or gangrene.

Examination

- Pale foot with evidence of tissue loss.
- Foot pulses are not palpable.
- Positive Buerger's test with a Buerger's angle of less than 20 degrees (see Box 1).
- Assessment of haemodynamics (see Box 2) and documentation of the WIfI score should also be performed (see Chapter 10).

WIfI score

- The WIfI system is based on three key factors: Wound, Ischaemia, and foot Infection.
- The composite WIfI score, which is calculated by adding the Wound, Ischaemia, and Infection sub-scores, is a predictor of the likelihood of amputation, limb salvage and wound healing and can identify those patients who may benefit from revascularisation.

Rutherford classification

- The Rutherford classification is a commonly used system to categorise the severity of PAD. It includes both a clinical descriptor and an objective assessment of haemodynamics using ankle pressures (AP) and toe pressures (TP).
- Rutherford categories 4 to 6 characterise CLTI (shown below).

Rutherford Stage	Clinical Presentation
0	Asymptomatic
1	Mild intermittent claudication
2	Moderate intermittent claudication
3	Severe intermittent claudication
4	Ischaemic rest pain
5	Minor tissue loss
6	Major tissue loss

BOX 1

Buerger's test

Buerger's test assesses the adequacy of the arterial supply to the leg. It has two parts:

1. Assessment of elevation pallor
With the patient supine, raise both legs at the same time to an angle of 45 degrees and hold for one minute. Observe the colour of the feet and the angle at which they become pale. Pallor is a sign of ischaemia and it occurs when the peripheral arterial pressure is not sufficient to overcome the effects of gravity. In a limb with a normal circulation, the toes and sole of the foot will remain pink, even when the limb is raised by 90 degrees. The poorer the arterial supply, the less the angle to which the legs have to be raised for them to become pale. A vascular angle of less than 20 degrees indicates severe PAD.

2. Assessment for reactive hyperaemia.
Then ask the patient to hang their legs down over the side of the bed. Gravity will help blood flow to return to the ischaemic limb and the foot will slowly turn pink and soon after red (so-called ischaemic rubor or sunset foot). This occurs due to the dilatation of the arterioles in an attempt to remove the metabolic waste products that have built up in a reactive hyperaemia. The foot then returns to its normal colour.

If possible, examine both legs at the same time, as the changes are most apparent when one leg has a normal circulation.

BOX 2

Non-invasive haemodynamic tests

Ankle pressure (AP) and ABPI

- Measurement of ankle pressure (AP) and the calculation of ABPI is recommended as the first-line non-invasive test.
- AP <50 mm Hg or ABPI <0.4 is typically seen in severe PAD, BUT a significant proportion of patients, particularly those with CKD and diabetes, will have incompressible blood vessels due to calcification, leading to artifactually elevated readings.
- If this is suspected, toe pressures (TP) and toe-brachial index (TBI) or other haemodynamic measurements, should be considered.

Toe pressures (TP) and toe-brachial index (TBI)

- TP is measured by placing an appropriately sized mini-cuff around the base of the great toe. This is attached to a standard manometer. A photoplethysmographic or continuous-wave Doppler flow detector is then used to determine when flow returns while the inflated cuff is slowly deflated.
- The digital arteries are relatively spared from calcification and TP is therefore less often affected by incompressibility, unlike AP.
- TBI <0.7 is abnormal and a TP <30 mmHg is associated with severe PAD.

Several other non-invasive tests also exist but are not recommended for routine use.

Imaging

- Imaging is performed for the purposes of evaluating the pattern of PAD which will help when planning intervention.
- First line arterial imaging is usually an arterial duplex (combination of doppler and ultrasound) with CT or MR angiography providing useful cross-sectional imaging for planning revascularisation. In some cases, invasive diagnostic digital subtraction angiography (DSA) can be helpful for planning surgery.

Prognosis

- CLTI is associated with premature death, with a mortality rate of 20% to 25% in the first year after presentation, mainly due to cardiovascular events.
- The 5-year all-cause mortality for patients with CLTI is as high as 70% and this is greater than that for some cancers.

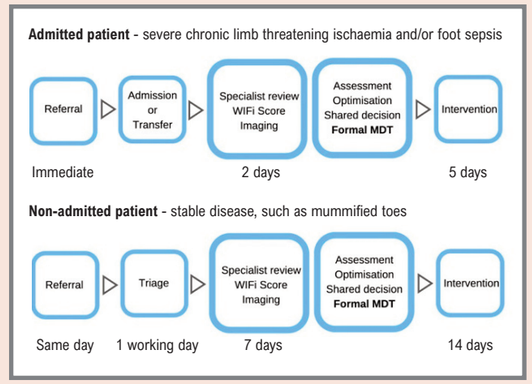
Management

- Optimal management of cardiovascular risk factors will reduce cardiovascular mortality and morbidity .
- Left untreated, the estimated overall risk of limb loss in CLTI is approximately 25% at 1 year. However, the risk of amputation remains high even in those who have undergone a successful revascularisation.
- Patients who present late and with the greatest degree of tissue loss are at the highest risk of major amputation.
- Early revascularisation will prevent limb loss, and the delays to treatment can be prevented by developing well organised networks with clear referral pathways.
- The effectiveness of non-revascularisation therapies, such as, spinal stimulation, pneumatic compression, prostanoids, and hyperbaric oxygen, is not yet established.

BOX 3

Peripheral Arterial Disease Quality Improvement Framework (PAD-QIF)

- The PAD-QIF aims to reduce unwanted variation in the delivery of services for people with PAD and describes the care pathways, workforce and facilities required to improve outcomes for patients with PAD.
- It advocates equitable access for people with PAD to timely revascularisation. Patients with CLTI who are admitted to hospital, should be treated within 5 days; non-admitted patients with CLTI should be treated within 2 weeks.



- Every patient should receive a multi-specialty assessment, including shared decision-making over treatment options. The core members of the multi-disciplinary team are shown below.

CORE MEMBERS
Formal lower limb MDT meeting
Vascular surgeon - *at least two*
Interventional radiologist - *at least two*
Vascular specialist nurse
Vascular anaesthetist
Consultant in care of elderly and frailty
Clinical vascular scientist
MDT administrator

Revascularisation strategies

- Improvement of the blood supply to the foot can be achieved by using minimally invasive angioplasty techniques, open surgery (endarterectomy or bypass operation) or a combination of angioplasty and open surgery, so-called hybrid interventions.
- CLTI usually occurs as a result of arterial disease at multiple levels. The level(s) of arterial disease within the lower limb (iliac, common femoral, superficial femoral, popliteal and tibial) arteries, the nature (occlusion versus stenosis) and the extent of disease (i.e. length of lesions), along with the fitness of the patient and WIfI score, will influence which strategy is best employed.
- In some patients, primary amputation or palliation may be the management strategy of choice. For example, those patients with significant co-morbidities or limited life-expectancy, complex or no options for revascularisation, extensive tissue loss or infection.

10 DIABETIC FOOT DISEASE

Chris Ashmore, Dave Russell

WHAT IS DIABETIC FOOT DISEASE?

- Diabetic foot disease is a common complication of diabetes mellitus (DM). It is a constellation of pathologies which includes neuropathy, ulceration, peripheral arterial disease, neuro-osteoarthropathy, gangrene and infection.
- Diabetic foot ulceration describes any disruption of the epidermis of the foot. The severity of an ulcer can vary depending on the site, size, depth and degree of infection or ischaemia.

Epidemiology

- Of the 5 million adults with DM, 10–25% will develop a diabetic foot ulcer in their lifetime.
- 50–80% of lower limb amputations are attributable to diabetes. Survival following a diabetic foot ulceration is 60% at 5 years, and 50% at 2 years following a major lower limb amputation.

Aetiology of diabetic foot ulcers

- Diabetic foot ulcers are classified as neuropathic, neuroischaemic or ischaemic.
- Identify the specific aetiology of a foot ulcer by considering the following:
  - **Diabetic sensorimotor neuropathy.** Caused by advanced glycation end products and microvascular disease of the vasa nervosa. This leads to foot deformity (clawed toes, prominent metatarsal heads) and loss of protective sensation (LOPS) in the foot, compounding traumatic or pressure damage which may lead to skin breakdown.
  - **Peripheral arterial disease (PAD).** Arises due to hyperglycaemia, oxidative stress-induced endothelial dysfunction and atherosclerosis. Impaired tissue perfusion results in aberrant wound healing or ischaemic necrosis.
  - **Precipitating event.** Epidermal ulceration can be caused by macrotrauma, such as standing on a nail, or repeated microtrauma due to foot deformity.

Prevention

- All people with diabetes should be screened for diabetic foot ulcers. The frequency of screening, ranging from annual to monthly check-ups, can be determined using the IWGDF Risk Stratification Score.
- Strategies for ulcer prevention include the provision of structured foot care education, advice or the provision of appropriate footwear, and the treatment of any modifiable risk factors.

Clinical assessment

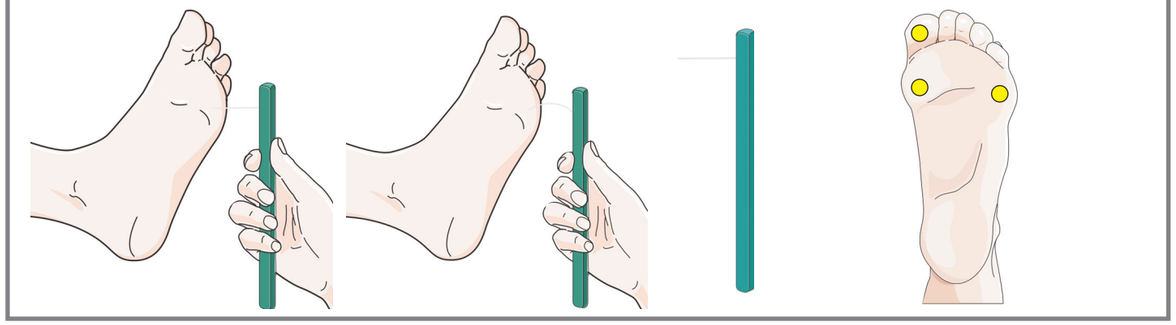
- Assess the patient holistically, obtaining clinical observations, a full vascular examination and a neurologic examination of the foot.
- All ulcers can be assessed using the SINBAD system. This provides a structured approach to ulcer assessment and can facilitate communication between healthcare professionals.
  1. **Site.** Describe the location of the ulcer with reference to the dorsal or plantar surface, digital, interdigital, forefoot, midfoot or hindfoot.
  2. **Ischaemia.** Full assessment of lower limb pulses. Insonation of foot vessels with a handheld doppler and quantifying metrics of arterial pressure (such as ABPI, toe pressures or transcutaneous pressure of oxygen).
  3. **Neuropathy.** 10 g Semmes-Weinstein monofilament assessment of intact plantar skin is used to describe the presence and distribution of neuropathy (see Figure 1).
  4. **Bacterial infection.** Diagnosis made by the presence of two signs of infection (redness, swelling, heat, pain, purulent discharge). Osteomyelitis should be suspected in a positive probe to bone test.
  5. **Area.** Measure the surface area of the ulcer.
  6. **Depth.** Using a sterile probe, describe the depth of the ulcer with reference to the skin, subcutaneous tissues, muscle or bone.
- The presence of fever, tracking cellulitis, lymphangitis, abscess, purulent discharge, rapidly progressing necrosis or crepitus indicate that urgent surgery may be needed.

Toe pressures

- Diabetes causes medial arteriosclerotic calcification resulting in incompressible vessels and a falsely elevated ABPI in people with diabetes and LOPS.
- Toe pressures measure the pressure required to occlude digital arteries and are less affected by LOPS.
- If the toe pressure value is <40 mmHg, the wound is less likely to heal without revascularisation.

Toe Pressure (mmHg)	Interpretation
>60	No significant or mild arterial disease
40–59	Moderate peripheral vascular disease
30–39	Severe peripheral vascular disease
<30	Critical ischaemia

FIGURE 1: SHOWING CORRECT USE OF SEMMES-WEINSTEIN MONOFILAMENT TO TEST FOR GLOVE AND STOCKING DISTRIBUTION OF DIABETIC SENSORY NEUROPATHY, AS DETAILED IN IWGDF 2023 PRACTICAL GUIDELINES



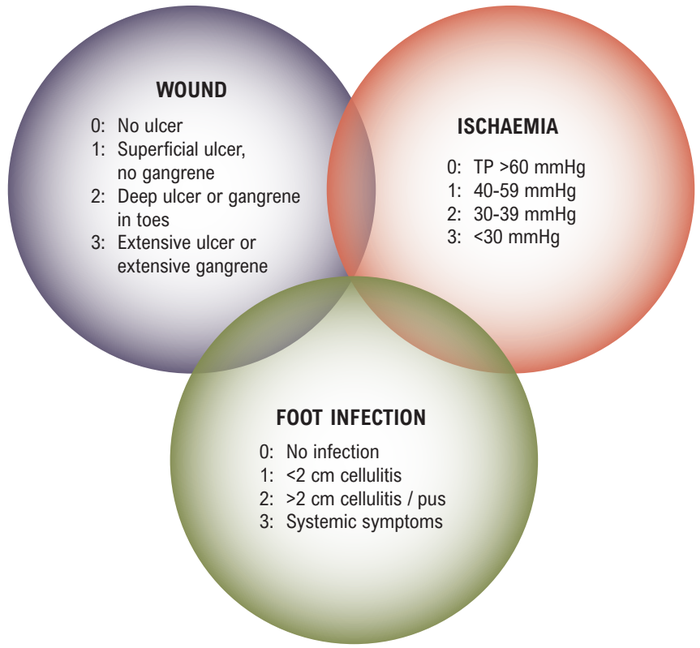
Imaging

- All patients with a deep ulcer or suspected Charcot foot should have an X-ray to assess for fractures, deformity and osteomyelitis.
- Where diagnostic uncertainty persists, MRI of the foot can be considered.
- Vascular imaging should be reserved for patients who are likely to require intervention.

WIfI score

- The WIfI score is a classification score for diabetic foot disease based on the presence and severity of the wound, distal ischaemia and foot infection.
- It is used to predict the risk of amputation and the benefit from revascularisation .
- The score is not added but is reported by each component e.g.WIfI: 2,0,1, with a stage of risk calculated using a scoring matrix or App.
- See below.

Diagrammatic representation of the Wound, Ischaemia and foot Infection (WIfI) staging system



Management

All patients with a diabetic foot ulcer (DFU) should have a referral made to a specialist multidisciplinary diabetic foot service within 24 hours.

Mechanical offloading

- This is the mainstay of management for all DFUs
- Offloading techniques can be divided into orthotic and surgical
  - Orthotic footwear can shift weight away from the affected area of the foot.
  - Non-removable devices are superior to removable devices, largely due to patient compliance.
  - Plantar ulcers should be treated with below knee casts/walker boots unless contraindicated.
  - Surgical techniques include achilles tendon lengthening, flexor/extensor digital tenotomy, metatarsal head resection, joint arthroplasty, or metatarsal osteotomy.

Soft tissue infection

- Superficial soft tissue infection can generally be treated by systemic antibiotics covering Gram positive organisms. Need for intravenous antibiotics or hospital admission depends on the extent of infection and presence of systemic symptoms.
- Surgical debridement, if required, should be performed urgently as ‘time is tissue’.

11 LOWER LIMB AMPUTATION

Ismay Fabre, Rosannah Williams, Ian Massey, David Bosanquet

GOALS OF AMPUTATION SURGERY

- Removal of all compromised, necrotic or grossly infected tissue.
- Achievement of primary healing
- Create a functional residual limb to preserve independent ambulatory ability in capable patients
- Pain relief

Amputation surgery should be performed to preserve life or preserve quality of life.

Indications for lower limb amputation

- Acute or chronic limb ischaemia
- Infection
- Trauma
- Malignancy
- A "non-functional" limb
- Symptom management in palliation (in cases where life cannot be saved, quality of death should be considered)

Amputation is indicated when the lower limb is "dead", "deadly" or "dead useless".

After a period of ischaemia, the tissue may be non-viable or "dead" and therefore can no longer be salvaged.

Uncontrollable infection, or release of waste products from necrotic tissue may pose a threat to life, making the tissue "deadly".

An ischaemic limb may lose its function due to paralysis, irremediable contracture, or destruction of the major weight bearing portions of the foot, making it "dead useless".

Levels of amputation

How do you decide what level to perform a lower limb amputation?

To determine the level of amputation the surgeon must balance the goals of amputation surgery, particularly the likelihood of primary healing creating a functional residual limb.

The higher the amputation level is, the higher the likelihood of primary healing (due to higher chances of sufficient blood supply), but the functionality of the residual limb is lower.

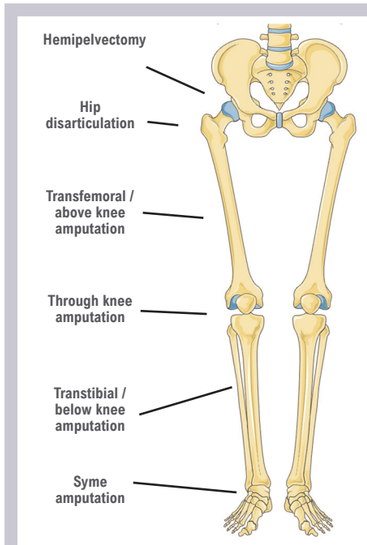
The optimal site is the lowest amputation level that will heal, therefore providing maximum rehabilitation potential.

CLASSIFICATION OF AMPUTATIONS

PRIMARY	VS	SECONDARY
Amputation undertaken as primary operation when there is no option for limb salvage, or in a palliative setting for symptoms management		Amputation undertaken when limb salvage, i.e. revascularisation efforts, have failed.

Major Lower Limb Amputation	VS	Minor Amputation
Hip Disarticulation Transfemoral Amputation Knee Disarticulation Transtibial Amputation Ankle Disarticulation		Trans-metatarsal amputation Digital Amputation

Minor amputations are often performed in combination with revascularisation procedures for distal necrosis to ensure healing of the amputation site. If possible, they are preferable as they can preserve foot function.



The MDT

- An MDT approach should be applied to all patients.

The Amputation MDT:
Vascular surgeon
Vascular anaesthetist
Specialist nurse
Interventional radiologist
Rehabilitation consultant
Physiotherapist
Occupational therapist
Prosthetist
Acute pain specialist
Diabetes team (if appropriate)
Clinical psychology

- Revascularisation and limb salvage should be considered in all patients.
- Patient-centered care and shared decision making is crucial.

Risks of amputation

Pain: Post-operative pain is common; many centres will place a perineural nerve catheter during MLLA to deliver local anaesthetic directly to the nerves. Acute pain team review is recommended after surgery.

Phantom Limb Pain or Sensation: The feeling that the removed limb remains attached, commonly pain or itching. Management is with neuropathic analgesics.

Infection: Surgical Site Infections (SSI) are a leading cause of morbidity and mortality after amputation. Vascular patients are often high risk due to common co-morbidities such as diabetes and smoking.

Wound healing problems & need for debridement or more proximal amputation: Failure of primary wound healing is a common problem after amputations of an ischaemic limb due to poor perfusion, seroma, haematoma and SSI. Wound complications occur in 12-34% of BKA and 6-16% of AKA.

Non-healing wounds may require debridement or revision of amputation. Revision could be at the same level, or revision to a higher level. Rates of conversion from BKA to AKA is 9-28%.

Mortality: The 5-year mortality after major amputation varies from 30-70%; it is higher for AKA than BKA.

Other: All patients undergoing surgery are at risk of developing chest infections post-operatively and thromboembolism including DVT, PE, CVA and MI. Early mobilisation and VTE prophylaxis reduce this risk.

FACTORS THAT INCREASE RISK OF WOUND COMPLICATIONS

Pre-operatively	Peri-operatively	Post-operatively
• Diabetes • Smoking • Renal failure • High body mass index • Anaemia • Poor perfusion	• BKA have a higher rate of primary healing failure than AKA. This is likely due to AKA level amputations having a more reliable perfusion status.	• Development of infection, seroma or haematoma • Compartment Syndrome • Poor wound care

Amputation technique

1. Dissection through skin an underlying fascia
2. Muscle groups are identified and transected approximately 1–2 inches longer than the planned level of bone cut to ensure adequate tissue coverage for the residual limb.
3. Major arteries and veins should be identified, dissected, ligated and transected.
4. The major nerves should be identified and sharply transected to reduce the risk of neuroma formation.
5. The bone/s are then transected and filed to ensure no sharp edges.
6. Closure is done in layers, including the fascia, subcutaneous tissue and skin. Some surgeons may also use myodesis (suturing muscles to drill holes in the bone), or myoplasty (suturing agonistic to antagonistic muscles) to provide more stability and better soft tissue coverage.

Rehabilitation

Steps in fitting a prosthetic limb

1. Physiotherapy and Occupational therapy work with the patient to ensure fitness for a prosthetic limb.
2. The Prosthetist makes a 'mould and model' of the residual limb. This may be with plaster, fibreglass or digital imaging.
3. A socket is created around the model.
4. The socket is sometimes set up on a 'diagnostic prosthesis' to asses function and comfort before the definitive socket is made.
5. Multiple appointments may be required to ensure the prosthesis is optimised to the individual.
6. Once the prosthesis is functionally optimised, an external finish can be added as deemed appropriate for each patient.

Factors improving rehabilitation potential

- **Medical optimisation**. Cardiovascular optimisation is important as energy expenditure of ambulation increases <40% after a BKA, and <70% after AKA.
- **Creation of a dynamic residual limb** with muscle stabilising procedures and good soft tissue coverage creates a more balanced, functional residual limb to interact with the prosthetic.
- **Good wound care** ensures a healthy interface for interaction with a prosthetic.
- **Early mobilisation** with physiotherapy improves long term outcomes.
- **Safe discharge planning** with occupational therapy.
- **Psychology, counselling and support groups** should be offered to all patients and their family, as undergoing an amputation is a life changing event.
- **Regular assessment with prosthetist** is important as the residual limb may evolve over time, requiring re-fittings.

THEME 3

DISEASES OF THE AORTA

12 ABDOMINAL AORTIC ANEURYSM

Conor Dooley, Ciarán McDonnell

WHAT IS ABDOMINAL AORTIC ANEURYSM?

- An aneurysm is defined as a localised swelling of an artery which exceeds 50% of it's normal diameter.

Diagnosis

- AAAs are usually asymptomatic until they are either rupturing or in imminent danger of doing so.
- The majority are detected coincidentally either during a routine clinical examination or on abdominal imaging such as ultrasound or CT being performed for some other reason.
- An ultrasound-based screening programme to detect AAA in males aged 65 or over operates in the UK and in many other countries.

Natural history and rationale for treatment

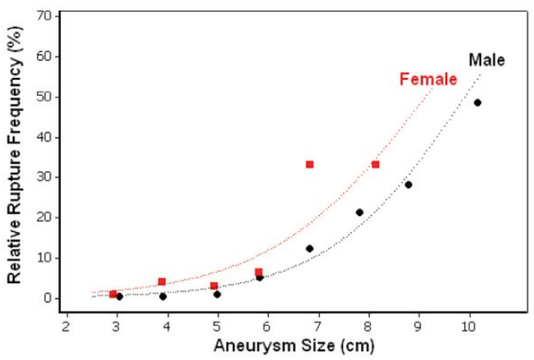
The natural history of aortic aneurysms is continuous enlargement, leading ultimately to rupture with catastrophic bleeding.

Elective surgical repair is the only option to prevent this. The purpose of surgical repair is therefore to prevent death from rupture, which still carries a 75–80% mortality risk, relatively unchanged over the last 40 years.

Aneurysm diameter is the best predictor of rupture risk, with current guidelines stating that when an AAA reaches a maximal diameter of 5.5cm then elective repair should be considered in asymptomatic cases.

AAAs less than 5.5cm in diameter are usually just kept under surveillance with regular ultrasound scanning.

Any Symptomatic AAA should be considered for urgent repair irrespective of AAA sac size. Symptoms include lower back or abdominal pain with no other identifiable cause or a palpable tender AAA on examination.



Surgical management

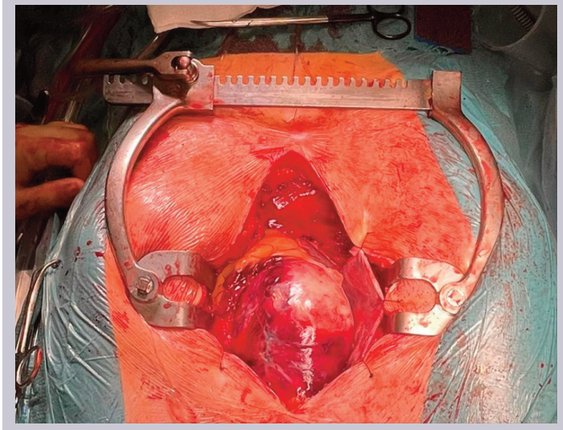
The principle of surgery is to exclude the aneurysm from the circulation by insertion of a synthetic graft inside the aneurysm sac. This prevents the aneurysm from expanding and ultimately rupturing, causing catastrophic internal bleeding. The difference between the two types of aneurysm surgery – Open Surgical Repair (OSR) and Endovascular Aneurysm Repair (EVAR) is related to how the graft is introduced.

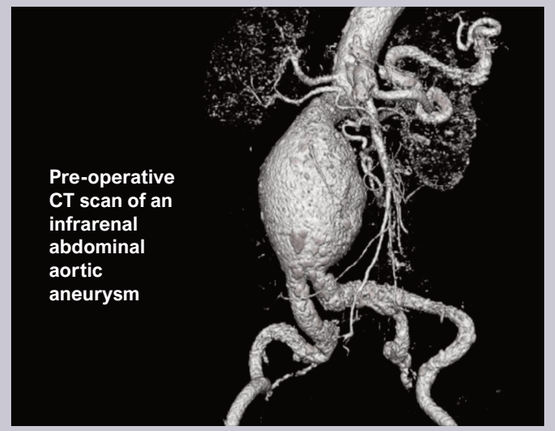
Open Surgical Repair (OSR)

- Open Surgical Repair is performed via a laparotomy, most commonly a longitudinal midline incision but sometimes via a transverse incision. The aorta is clamped proximal and distal to the aneurysm which is then opened and a synthetic graft (usually made from Dacron or PTFE) is then sutured into the normal aorta above and below. The clamps are released and blood flow to the lower torso restored.

Endovascular Aneurysm Repair (EVAR)

- This is performed by means of introducing a stent (endograft) usually through the femoral arteries. The stent consists of a metal exoskeleton with synthetic graft material stitched onto it and is usually scrunched-up within a long narrow tube (a sheath) which is introduced into the lumen of the femoral vessels over a guidewire and passed up into the aneurysm sac under X-ray guidance. When the stent is in the correct position it is uncovered and expands, sealing the aneurysm at the proximal and distal ends and containing blood flow within the endograft and not within the aneurysm sac.





Complications of aneurysm repair

Open Surgical Repair is characterised by three major physiological insults: Laparotomy, Aortic Cross Clamping and Ischaemia-Reperfusion Injury, none of which occur during EVAR. This is reflected in differences between the complications of the two types of procedures.

Complications of OSR

Apart from the obvious potential complication of bleeding from the suture line, there are many others:

SURGICAL INSULT	
Laparotomy	- Ileus - Bowel/ureteric/viscus injury - Laparotomy wound dehiscence or later incisional hernia - Respiratory complications secondary to impaired respiratory movement due to pain
Aortic Cross Clamping	- Increase in cardiac afterload with cardiac complications, M.I., C.C.F., arrhythmia etc.
Ischaemia-Reperfusion Injury	- Hypotension - Myocardial depression - Cardiac irritation and Arrhythmias - Acute lung injury - Acute renal failure

Application of the aortic clamp renders the lower torso relatively ischaemic, and when blood flow is restored this washes out various metabolites into the systemic circulation which can cause a systemic inflammatory type response.

Loss of blood flow through the inferior mesenteric artery can sometimes produce large bowel ischaemia.

Late complications which can occur are pseudoaneurysm formation at the anastomotic sites, and the development of an aorto-enteric fistula, where the exposed aortic suture line erodes through overlying bowel wall, producing catastrophic (and almost invariably fatal) bleeding. Hernias and adhesive small bowel obstruction are further recognised complications.

Complications of EVAR

Persistent blood flow within the aneurysm sac (endoleak) is a unique complication of EVAR. Endoleaks are classified depending on their source:

TYPE	NATURE OF LEAK	RELEVANCE
I	Loss of the sealing zone at the proximal or distal ends of the aneurysm	High velocity blood flow within the AAA. Requires urgent treatment.
II	Persistent back bleeding from lumbar arteries or inferior mesenteric artery back into the aneurysm sac	Usually low velocity and very often self-limiting.
III	Separation of the components of the graft within the aneurysm sac	High velocity flow within the AAA which requires urgent attention
IV	Due to porosity of the graft material	Described in the earlier phase of endograft development – rarely if ever seen now.
V	Expansion of the aneurysm sac with no demonstrable endoleak	Controversial. May require graft explantation and conversion to open repair if continuous.

Iatrogenic arterial injury can occur as a consequence of the endograft being introduced through the iliac arteries.

WHICH METHOD OF REPAIR – OPEN OR ENDOVASCULAR?

Not surprisingly, the lesser surgical insult of EVAR is reflected in a lower 30-day mortality (1.8%) compared to OSR (4.8%). This survival advantage is not maintained, however, with longer term survival being similar. The potential for the development of endoleak at any stage postoperatively requires lifelong follow up with serial imaging (usually annual ultrasound +/- xray) of EVAR patients whereas this is usually not necessary in those undergoing OSR. There is also a higher reintervention rate in those treated with EVAR vs. OSR. The decision as to which procedure to undertake is usually made by the surgeon in consultation with the patient, taking into account the individual anatomical and physiological factors unique to each case.

13 RUPTURED ABDOMINAL AORTIC ANEURYSM

Conor Dooley, Ciarán McDonnell

WHAT IS RUPTURED ABDOMINAL AORTIC ANEURYSM?

- A ruptured abdominal aortic aneurysm is a surgical emergency where the integrity of the abdominal aorta is compromised due to a breach within the aortic wall leading to leakage of blood out of the circulation.
- The aneurysmal nature of the aorta means that the aortic wall is weak.
- This is a life threatening emergency reflected in the fact that a ruptured abdominal aortic aneurysm carries a mortality rate in excess of 70%.

Presentation

The classical presentation of rAAA is a triad of:

1. **Back / abdominal pain**
2. **Hypotension**
3. **Pulsatile abdominal mass**

However only 30–50% will present with all three symptoms. Symptoms can differ depending on the site of rupture. Posterior wall rupture is usually contained via the retroperitoneum and causes compressive symptoms on surrounding nerves causing pain. An anterior wall rupture ('free rupture') enters the peritoneal cavity and usually presents with sudden collapse and death.

Alternative presentation of rAAA:

- Flank/Groin/Scrotal Pain
- Haematuria
- Collapse

Common misdiagnoses of rAAA:

- Renal Colic
- Diverticulitis
- Perforated Viscus

Management

Unstable patient

Patients who present haemodynamically unstable and have a known aneurysm or suspected rupture should be taken immediately to the operating theatre for repair.

Stable patient

Patients who arrive haemodynamically stable and conscious have sufficient time to be further assessed with imaging (CT angiogram). Two large IV cannulas should be placed for medication, fluids and blood products.

Co-morbid patient

Patients who are elderly, who have significant co morbidities and who are unlikely to survive the operation should be considered for palliation. This is after discussion with the patient, if possible, family members and medical / anaesthetic teams.

Investigation

Ultrasound	• Can be performed at the bedside in the Emergency Department • Can effectively assess the patient's aorta size (e.g. if the Aorta is normal then a rAAA can be ruled out) • NOT reliable to exclude a rupture
CT Angiogram	• The gold standard form of imaging • Can be performed rapidly and is available in most Emergency Departments quickly confirming the diagnosis • Added benefit of assessing suitability for endovascular repair (EVAR)

Permissive hypotension

A key element in managing a patient with rAAA allowing a relatively low systolic blood pressure of 80–100 mmHg by avoiding large amounts of fluid resuscitation. Aggressive fluid replacement can disturb the tamponade that has formed at the rupture site and risks of re-rupture of the contained retroperitoneal hematoma with catastrophic bleeding.

Surgical management

OPEN SURGICAL REPAIR – DIFFERENCES FROM ELECTIVE OPEN SURGERY

THE 30 MINUTE RULE:
A patient with a suspected rAAA should be assessed, scanned and transferred to theatre within 30 minutes.

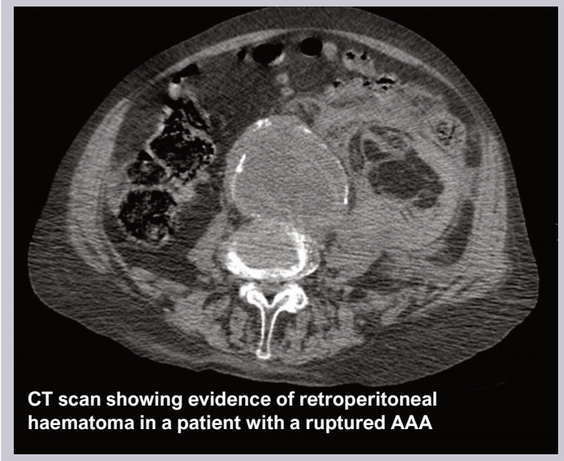
- The patient is prepped and draped while awake before the induction of general anaesthesia. Rapid sequence induction is performed, followed by an immediate midline laparotomy. (muscular relaxation from the anaesthetic can cause a loss of tamponade of the retroperitoneal haematoma and therefore this time needs to be limited)
- The aortic neck is dissected and clamped, once proximal control has been achieved, time is given to the anaesthetic team to replace fluids and blood products and stabilise the patient. Aortic repair with a synthetic graft is then performed in a similar approach to an elective repair.

Endovascular Repair (EVAR)

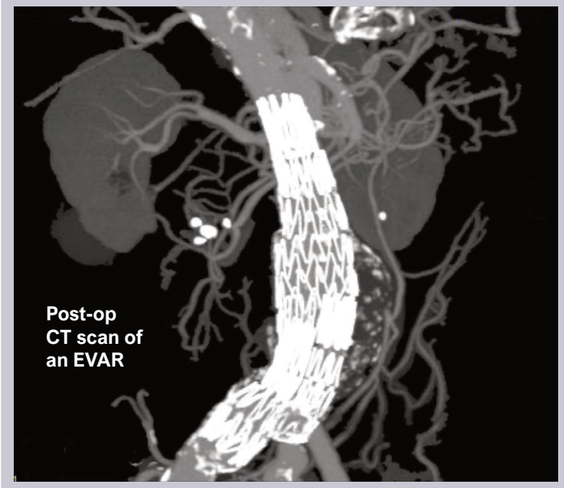
- If the aorta has suitable anatomy, then an endovascular repair may be performed preferentially under local anaesthesia and mild sedation (although general anaesthesia can be performed). Patients need to be haemodynamically stable to permit an endovascular repair of a rAAA as the capacity to achieve rapid arrest of aortic bleeding by application of an aortic cross clamp such as in an open repair, does not exist. In the case of significant hypotension an intra-aortic balloon placed in the proximal aorta may aid with haemodynamically stability but makes the EVAR more challenging to perform.

EVAR – differences from Elective Surgery

- It is preferable that repair is achieved using a bifurcated device where possible. However, in certain circumstances, an EVAR can be performed using an aorto uni-iliac type graft. This allows blood flow into one iliac artery only while the other one is blocked using a special vascular occluder. The leg supplied by this vessel is then revascularised using a fem-fem crossover bypass. This method is generally faster than the standard bifurcated EVAR device and thus allows for more rapid control of bleeding if required.



CT scan showing evidence of retroperitoneal haematoma in a patient with a ruptured AAA



Post-op CT scan of an EVAR

Abdominal Compartment Syndrome (ACS)

A significant complication that can manifest after a ruptured aortic aneurysm repair. Abdominal compartment syndrome is defined as organ dysfunction with abdominal hypertension (>25 mmHg). This may present with elevated peak airway pressures, decreased tidal volumes and increased difficulty ventilating the patient as well as decreased urine output due to acute renal failure. Abdominal pressures can be estimated via bladder pressure measurements. There should be heightened vigilance for ACS after endovascular cases as these patients do not have the retroperitoneal haematoma evacuated during the procedure as would routinely happen during an open repair.

Management

Conservative measures include nasogastric decompression, muscle relaxants, abdominal TAP blocks and, if these do not work, operative decompression laparotomy. In these cases, the abdomen is left open with a sterile abdominal dressing to protect the cavity and is closed later.

A Study to Know

The IMPROVE Trial - The Immediate Management of Patients with Rupture: Open Vs Endovascular Repair

Multicentre trial that randomised patients with a diagnosed rupture to either EVAR if anatomically suitable or Open repair

- No difference in mortality between the two groups
- EVAR was associated with a shorter length of stay in ICU and hospital
- Patients who underwent EVAR were also more likely to be discharged directly home

14 TYPE B AORTIC DISSECTION

Millie Sucharitkul, Rachael Forsythe

WHAT IS AORTIC DISSECTION?

- A tear in the innermost layer of the aorta allowing blood to flow between the intimal and medial layers of the vessel wall. This creates a 'false lumen', causing the layers to separate.
- Dissections arising in the ascending aorta or arch proximal to the left subclavian artery (Type A aortic dissection; TAAD) are surgical emergencies requiring immediate intervention by a cardiac surgery team.
- Dissections arising distal to the left subclavian artery (Type B aortic dissection; TBAD) are managed by the vascular surgery team. They do not always require immediate surgical intervention, but should be managed initially in a critical care environment. TBAD can be complicated by retrograde TAAD dissection and a joint management approach may be required.
- TBAD is one of three 'Acute Aortic Syndromes' alongside Intramural Haematoma and Penetrating Aortic Ulcer. The principles of management are similar for these pathologies
- TBAD most commonly presents with acute severe chest pain, inter-scapular pain or signs of end-organ ischaemia such as abdominal pain, lower limb ischaemia or paralysis
- Fatal complications include rupture of the aorta and visceral malperfusion leading to ischaemia of abdominal organs

ANATOMICAL CLASSIFICATION OF AORTIC DISSECTION

Most widely adopted classification systems are detailed below:

Stanford - Type A: Entry tear proximal to left subclavian artery, Type B: Entry tear distal to left subclavian artery.

DeBakey – Type 1: Entry tear in the ascending aorta, extending throughout, Type 2: Entry tear in the ascending aorta, isolated to ascending aorta, Type 3A: entry tear in descending aorta, distal extent above diaphragm, Type 3B: entry tear in descending aorta, distal extent below diaphragm.

SVS/STS – Type A: Entry tear in Zone 0, Type B: Entry tear in Zone 1 onwards. Distal extent also denoted by zones. B1,7 indicates entry tear in Zone 1, dissection extending to Zone 7.

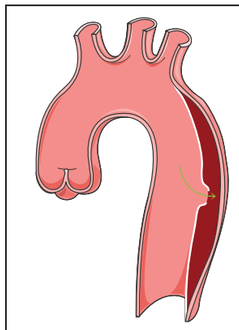
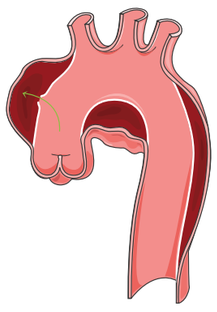


Diagram showing a type B aortic dissection with an entry tear distal to the left subclavian artery.



Type A aortic dissection with an entry tear within the ascending aorta and the dissection extending down in to the descending thoracic arota

Clinical classification of type B aortic dissection

Acute, subacute and chronic
TBAD is defined by the time from symptom onset to clinical presentation. (acute: 1–14 days, subacute: 14–90 days, chronic: >90 days).

Uncomplicated versus complicated
A patient with an uncomplicated TBAD has not sustained a rupture and/or has no features of end-organ malperfusion. A complicated aortic dissection refers to a dissection that has ruptured or caused end-organ malperfusion and/or displaying at least one of the high risk features:

- Refractory pain and/or hypertension
- Visceral ischaemia and malperfusion, even if solely radiological
- Entry tear ≥10 mm
- False lumen diameter ≥22 mm
- Aortic diameter ≥40 mm
- Retrograde dissection
- Entry tear at lesser curve
- Bloody pleural effusion
- False lumen thrombosis

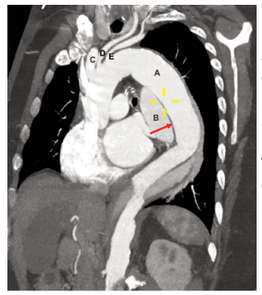
Diagnosis of aortic dissection

History
- Sudden onset, severe, tearing pain in the chest or between scapulae
- Radiation to limbs, suspect involvement of brachiocephalic or iliac arteries
- Radiation to abdomen, suspect mesenteric ischaemia
- Symptoms of other end organ dysfunction: lower limb paraesthesia or paralysis
- Syncope and sweating

Investigations
- Blood pressure: patient is usually hypertensive, a hypotensive patient with symptoms of dissection should be investigated for rupture
- Improvement of pain upon blood pressure control is a strong indicator for aortic dissection
- D-dimer: studies show the potential of D-dimer to aid diagnosis of aortic dissection. A raised level in conjunction with typical symptoms is sensitive in detecting dissection.

Imaging
- Contrast CT Angiography (CTA) is diagnostic – images acquired show longitudinal division of the aortic wall forming a double-lumen. Cardiac gating allows synchronisation of images to the cardiac cycle

PREOPERATIVE SAGITTAL CTA OF TBAD

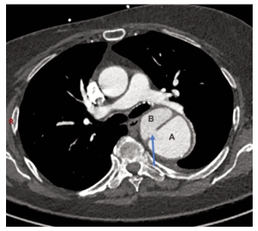


Location of entry tear (yellow cross)

Dissection flap (red arrow)

A. True Lumen;
B. False Lumen;
C. Innominate Artery;
D. Left Carotid Artery;
E. Left Subclavian Artery

PREOPERATIVE TRANSVERSE CTA OF TBAD



Entry tear (blue arrow)

A. True Lumen;
B. False Lumen

- Transthoracic and transoesophageal echocardiography can both be used to visualise the proximal aorta and, as such, is used mostly for TAAD. Transoesophageal echocardiography is more sensitive.

Risk factors for aortic dissection

- Hypertension, most common cause
- Abrupt significant increase in blood pressure: heavy weight-lifting, use of drugs with sympathomimetic effects (e.g. cocaine, ecstasy)
- Existent aortic aneurysm
- Certain conditions can predispose individuals to development of aortic dissections:
  - Connective tissue disorders – Marfan Syndrome, Ehlers-Danlos Syndrome
  - Congenital malformations – bicuspid aortic valve, coarctation of the aorta

Differential diagnosis

- Chest X-ray and ECG may be used to exclude other cardiac pathologies presenting with acute chest pain (e.g. cardiac tamponade, myocardial infarction, pulmonary embolism)
- Other acute aortic syndromes should also be considered upon presentation
- Confirmation on CTA rules out other pathologies

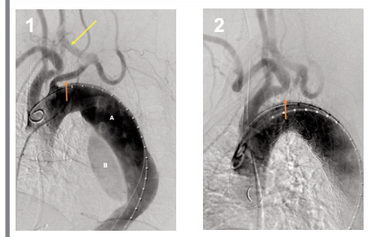
Management of TBAD

- The aims of initial management for TBAD are to prevent or treat rupture of the aorta and organ malperfusion. This starts with immediate control of blood pressure and involvement of critical care teams
- The INSTEAD trial 5 proposes a 'complication-specific' approach, whereby optimal medical management (through strict BP control and close surveillance) is preferred in the event of an uncomplicated dissection. Management with TEVAR at this stage provides no significant survival benefit to patients.

Surgical management

- Thoracic Endovascular Aortic Repair (TEVAR) has emerged as the preferred surgical option for patients with complicated TBAD. Stent grafting to cover the intimal entry tear and stabilise the vessel wall prevents life-threatening sequelae. TEVAR requires adequate proximal and distal sealing zones (≥20 mm) and may necessitate revascularisation of the neck vessels to create a suitable length of proximal aorta on which to land the stent graft. Most commonly, this is a left carotid-subclavian bypass. In an acute setting, covering the left subclavian for revascularisation at a later date may be acceptable.
- Open repair of TBAD is rarely performed in the acute setting due to the significantly higher risk of complications than TEVAR in this typically elderly, frail and comorbid population. Open repair is more commonly required for younger patients with aneurysmal change as a result of TBAD.
- Acute complicated TBAD – initial control of blood pressure and heart rate with anti-impulse therapy, followed by surgical intervention (typically TEVAR) to exclude the false lumen and restore blood flow to compromised organs.
- Acute uncomplicated TBAD – managed medically in the first instance, but requires close surveillance to ensure early detection and treatment of complications. Early TEVAR (± neck revascularisation) may be required during the subacute phase if there is evidence of high risk features.

PRE (1) AND POST (2) TEVAR ANGIOGRAM OF TBAD



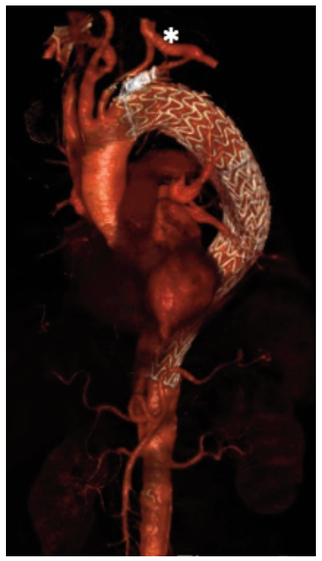
The true lumen **(A)** and false lumen **(B)** both fill with contrast. The left subclavian artery (orange arrow) and carotid-subclavian bypass (yellow arrow), are similarly visualised.

The false lumen no longer fills with contrast. The origin of the subclavian artery has been covered by the stent graft – hence the bypass. Therefore, the subclavian does not fill proximally (orange arrow). The corrugated pattern of the TEVAR graft stent can be seen along the aorta.

Lifelong surveillance

- Follow-up CTA should be scheduled regularly – for example, at 1 month, 6 months, 1 year, and yearly thereafter. This includes all patients diagnosed with aortic dissection, irrespective of means of management. This is due to the high incidence of aneurysmal change following diagnosis or treatment of dissection.

3D RECONSTRUCTED CTA OF TBAD



5 days postoperatively

The TEVAR stent graft is shown clearly, along with the coiled origin of the subclavian artery (seen as a white patch) and patent left carotid-subclavian bypass (*)

15 THORACIC AND ARCH ANEURYSMAL DISEASE

Becky Sandford

WHAT IS THORACIC AND ARCH ANEURYSMAL DISEASE?

- Aneurysms of the aortic arch and descending thoracic aorta may arise as a result of different underlying pathology
- Both are more common in men than women, and rupture of a thoracoabdominal aneurysm affects 10–15 per 100,000 person years
- They may be associated with connective tissue disorders (CTD) such as Marfans or Loeys-Dietz syndrome or related to an underlying vasculitis
- Occasionally such aneurysms may be mycotic in nature

Connective Tissue Disorders

Marfan syndrome

A genetic disorder that affects connective tissue and can cause pathological processes to occur across multiple systems in the body. MS is caused due to a mutation in one of the genes that makes fibrillin (FBN1) which leads to a weakened connective tissue. It is autosomal in nature and in 75% of cases is inherited (in the remaining 25% it occurs due to a new mutation). Such patients are at risk of developing cardiac valvular issues and are also at risk of aneurysmal degeneration of the ascending aorta and the arch of the aorta.

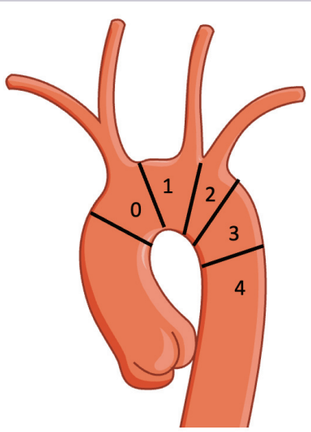
Loeys–Dietz syndrome

An autosomal dominant condition that can affect a number of genes that encode transforming growth factors. The condition can present with premature arterial aneurysm formation or dissection (even can happen in children).

Vascular Ehlers–Danlos syndromes (vEDS)

vEDS is one of a number of syndromes classified as Ehlers Danlos syndrome. vEDS is the most severe of the causes. It is commonly caused by mutations in the COL3A1 gene which results in extremely fragile tissues in general but specifically blood vessels. Patients commonly present with early aneurysmal formation (with rupture) or arterial dissection. Treatment is challenging due to the arterial fragility.

Anatomy



- The location of the aneurysm reflects the complexity of treatment options
- Important to understand which great vessels may be affected and the aortic arch and thoracic aorta can be split into zones (The Ishimaru's classification scheme – see Table 1)
- Thoracoabdominal aneurysms are described according to the Crawford classification system, with Type II aneurysms being the most extensive and carrying the greatest risks involved with repair.

TABLE 1: SHIMARU'S CLASSIFICATION

0	Involves the origin of the innominate artery
1	The origin of the left common carotid artery (LCCA)
2	The origin of the left subclavian artery
3	The proximal descending thoracic aorta down to the T4 vertebral body
4	The remainder of the thoracic aorta

Clinical Features

History

- Most patients are asymptomatic and the aneurysm is detected as an incidental finding often following a chest xray (CXR) or cross-sectional imaging (CT or MRI).
- A CXR will commonly show one or all of the following:
  - widening of the mediastinum
  - enlargement of the aortic knob
  - displacement of the trachea from the midline
- If patients are symptomatic they classically present with chest pain or back pain which is usually interscapular.
- If the patient presents with symptoms then there is an urgent need to exclude rupture of the aneurysm.

Family History

- Family history of dissection, aneurysm, connective tissue disorder, or sudden death at a young age may be present.

Examination

- Findings may be normal in the absence of aortic rupture.
- Patients may be hypotensive / tachycardic if there is an aortic rupture.

Investigations

- If there is a clinical suspicion of thoracic aortic pathology then a CT angiogram should be undertaken.

BOX 1

Indications and considerations for repair

Indications for ascending aortic repair:

- 5.5 cm as a general threshold for surgical intervention for non-syndromic/non-bicuspid aortic valve patients
- Any aneurysms that have a growth rate greater than 0.5 cm/ year are considered for surgical repair.
- The size limit is reduced to 4.5–5.0 cm for syndromic aortopathy

Indications for arch / thoracic descending aortic repair

- Aneurysms >60 mm should be considered for repair
- In patients with CTD, a lower threshold of 50 mm may be used

Symptomatic patients should be considered for urgent aortic repair

Risks of repair need to be balanced with risks of aortic rupture when deciding optimum management plans.

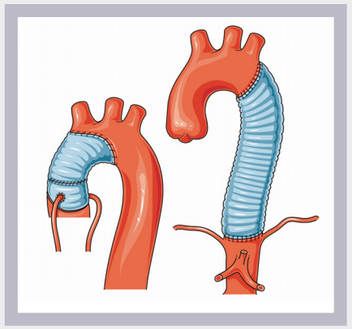
Management options

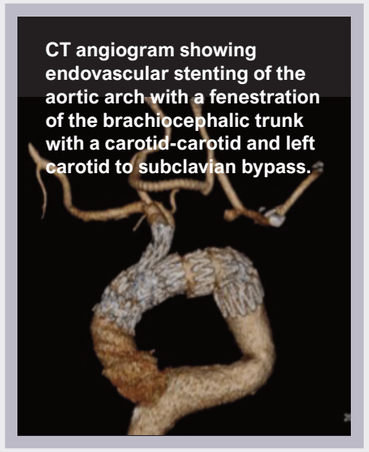
Open surgery

- Often more suitable for younger patients and those with connective tissue disorder or those without good endovascular options due to the aortic arch anatomy.
- Needs collaboration with cardiac surgeons and facilities for cardiopulmonary bypass.
- Higher complication risks associated with such an approach. Risks of open surgery need to be balanced against benefits.

Endovascular surgery

- The development of more complex endovascular techniques has meant that more patients can be treated this way.
- Often more suitable for atherosclerotic aneurysms in older patients.
- Usually incorporates branched or fenestrated graft design to maintain perfusion to visceral vessels





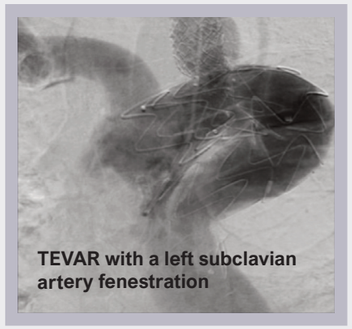
CT angiogram showing endovascular stenting of the aortic arch with a fenestration of the brachiocephalic trunk with a carotid-carotid and left carotid to subclavian bypass.

- Can incorporate branches for the left subclavian, left carotid and innominate arteries in some cases.

Hybrid intervention

Combination of open arch surgery and endovascular management of the thoracic aortic aneurysm.

- The frozen elephant trunk (FET) technique is a relatively new technique that involves surgical repair of the aortic arch which then facilitates an endovascular repair of the descending thoracic aorta. It is increasingly used in more complex aortic pathologies where there is a lack of a proximal landing zone for a thoracic endovascular stent graft.
- The proximal part of the FET consists of a standard aortic graft that has multiple branched grafts coming of the main body which can be anastomosed to the individual arch vessels (there is also often a branch that can facilitate distal body perfusion at the time of surgery) The distal part of the graft is a self-expandable stent graft that is positioned in the descending thoracic aorta which can provided a stable landing zone for a subsequent TEVAR.



TEVAR with a left subclavian artery fenestration

BOX 2

Prevention of complications

Spinal cord ischaemia from covering the spinal arteries can occur after thoracoabdominal aneurysm repair.

Spinal cord perfusion = Mean Arterial Pressure – intraspinal pressure

Increasing MAP is vital to maintain spinal cord perfusion

Spinal Cord protection relies on strict physiological parameters, e.g.:

- Hb >100 g/L
- Sats >96%
- MAP >90 mmHg
- CSF drainage can also be considered in high risk cases

THEME 4

VENOUS DISEASE

16 VARICOSE VEINS AND CHRONIC VENOUS DISEASE

Victoria Bristow, Tristan Lane

WHAT ARE VARICOSE VEINS AND WHAT IS CHRONIC VENOUS DISEASE?

- Varicose veins are common and are the visible component of superficial venous disease. Up to 40% of the population are affected to a lesser or greater extent.
- Deep venous disease is due to either incompetence or obstruction of the deep veins which is often secondary to deep vein thrombosis (DVT).
- Chronic venous disease is where chronic venous hypertension has led to permanent skin damage which increases the risk of skin ulceration.

How is venous disease diagnosed?

Venous disease is diagnosed with three key components:

1. Clinical history
2. Clinical examination
3. Clinically directed imaging – venous duplex ultrasound and rarely magnetic resonance venography and direct invasive venography

Clinical history

A detailed clinical history should be undertaken that includes:

- Symptoms should be discussed with a focus on any pain, swelling, itching of the legs and risk factors identified such as prolonged periods of standing. It is important to also gauge what the individual's biggest concern is?
- Family history – focussing on thrombotic conditions.
- Past medical history – any previous deep vein thrombosis (DVT) or central venous access?
- Current medication including anticoagulants, hormonal therapies.
- Previous surgical history – any previous varicose vein procedures? Has the vein previously been used in other procedures e.g., coronary artery bypass grafts (CABG) or arterial bypasses?

Clinical examination

An examination should be performed assessing the legs bilaterally for visible varicosities, leg oedema and skin changes. The Clinical-Etiology-Anatomy-Pathophysiology (CEAP) classification can be used to give an objective number of the severity of chronic venous disease (Table 1). With progressive venous hypertension, skin changes will develop.

Lower limb pulses should also be checked to rule out any peripheral arterial disease.

Clinical directed imaging

Imaging should be undertaken with a venous insufficiency duplex ultrasound scan to assess for superficial and deep venous reflux. Duplex means the use of colour imaging to show flow in addition to the picture (B-mode) image.

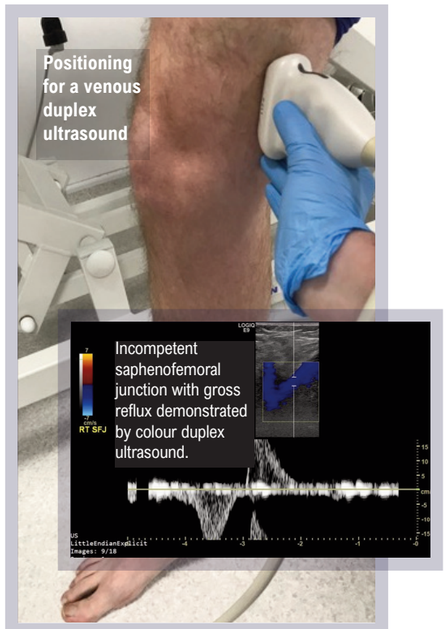


TABLE 1: CEAP CLINICAL STAGES

C0	C1	C2	C3	C4	C5	C6
No signs of venous disease	Telangiectasia or thread veins	Varicose veins- (Figure 1)	Limb swelling	Venous skin changes (haemosiderin, atrophie blanche, lipodermatosclerosis) (Figure 2)	Healed venous ulceration	Active venous ulceration (Figure 3)

FIGURE 1: C2 DISEASE – VARICOSE VEINS

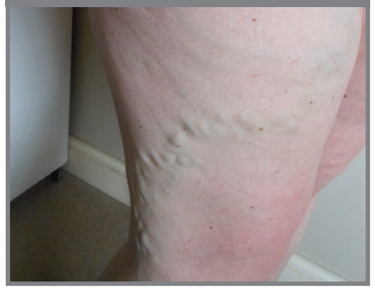


FIGURE 2: C4 DISEASE – CHRONIC SKIN CHANGES

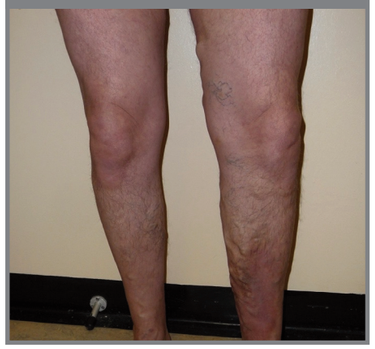
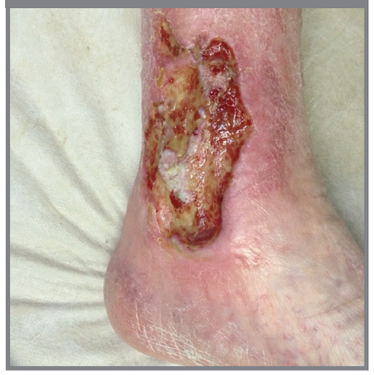


FIGURE 3: C6 DISEASE – VENOUS ULCERATION



Management of venous disease

Treating venous disease has been shown to offer significant benefits to patients at low cost with good expectations of a positive result.

There are many options:

Conservative management

Conservative management of venous disease is heavily reliant on patient compliance with compression hosiery or compression wraps. Patients often find these garments difficult to apply and wear. NICE guidance does not recommend this approach in patients where intervention is an option.

Compression strength can range from light (liners at 10 mmHg) to strong (European Class 4 stockings >49 mmHg). Herbal medication such as red vine leaf extract and horse chestnut extract can also be recommended to the patient. These medications have been shown to reduce signs and symptoms of chronic venous insufficiency.

Superficial venous disease

In instances where conservative management has failed, or the patient is symptomatic including having episodes of superficial venous thrombosis, bleeding, skin changes or active ulceration, there are several methods of treatment.

NICE guidelines recommend the following treatment preference order for truncal veins (great saphenous, small saphenous or anterior accessory saphenous vein):

1. thermal treatment (radiofrequency ablation or endovenous laser treatment)
2. chemical ablation (ultrasound guided foam sclerotherapy)
3. open surgery (high tie and stripping).

The tortuous veins that remain can be treated with phlebectomies (performed using open surgery by hooking the veins out through small skin incisions) or ultrasound guided foam sclerotherapy.

The vast majority of varicose vein procedures can be completed under local anaesthetic and most patients can have some type of treatment. This is one of the most common procedures performed in the NHS.

Deep venous disease

The management of deep venous disease heavily depends on patients' symptoms. The deep venous system may be obstructed or incompetent. Incompetent deep venous systems are managed conservatively as described above.

Obstruction is primarily caused by DVT, and this may lead to post-thrombotic syndrome, the severity of which can be scored with the Villalta score. Those who are asymptomatic are managed conservatively with compression hosiery and anticoagulation, this is the vast majority of patients.

In those with severe symptoms due to obstruction, this can be acute or chronic. Acute DVT can be treated with thrombus removal, which is most effective in the first 14 days post event, often followed by stenting of a narrowing in the vein. Chronic deep venous occlusion that affects the ilio-caval system may be amenable to deep venous stenting. This is complex and specialised limited to experienced centres to achieve good results.

Differential diagnoses for venous disease

Varicose veins are easy to identify; however, the additional signs and symptoms seen with varicose veins may be caused by other conditions.

Lower limb oedema

Occurs without venous insufficiency from the following causes:

- Lower limb dependency / immobility / lack of activation of the foot and calf muscle pumps.
- If unilateral, rule out causes of extrinsic iliac vein compression (e.g. malignancy)
- Heart failure commonly causes bilateral leg swelling.
- Lymphoedema should be considered.
- Obesity leads to relative venous hypertension and subsequent oedema.

Leg pain

- Aching legs can also be a sign of peripheral arterial disease. In which case an ankle brachial pressure index (ABPI) of less than 0.8 would be indicative of the presence of peripheral arterial disease which may need further investigations and risk factor modification.
- Leg pain can also be related to spinal disease. The symptoms of spinal disease often include neuropathic pain, they often differ from those with venous disease by having a positive sciatic stretch test on clinical examination.

Skin changes

- Skin rashes, psoriasis and eczema can mimic chronic venous skin changes.

Superficial vein thrombosis (SVT)

- This condition has historically been known as superficial thrombophlebitis.
- It typically, presents as a painful and tender lump or cord, with redness and heat, located in an area of pre-existing varicose veins, particularly along the course of the great saphenous vein (GSV).
- Misdiagnosis as an infective process is common and frequently results in the unjustified use of antibiotics.
- Diagnosis is made clinically and with the use of a venous duplex scan.
- Any SVT <3 cm from the saphenofemoral junction (SFJ) should be treated as a DVT.
- If the SVT is >3 cm from the SFJ and >5 cm in length, then the patient should be anticoagulated for 6 weeks.
- In any other circumstances then symptomatic treatment should be considered. This is usually is in the form of non-steroidal anti-inflammatory agents.

All patients are entitled to a specialist vascular review and assessment, but treatment is dependent on funding according to current local guidance.

17 VENOUS LEG ULCERATION

Leanne Atkin

WHAT IS VENOUS LEG ULCERATION?

- A leg ulcer is defined as a wound on a lower limb, below the knee but above the level of the malleolus with chronic venous insufficiency (CVI) the most common cause. CVI is the failure of the veins to carry blood back to the heart appropriately resulting in abnormal increase in pressure within the veins (chronic venous hypertension).
- CVI can occur due to problems in either the superficial or deep veins. The most common issue is failure of the valves to work in the veins causing reverse flow of the blood (reflux). Less common, venous obstruction usually due to thrombus can cause venous hypertension.

Introduction

- Increased venous pressure alters the balance of fluid in the tissue and in the capillaries resulting in fluid being forced from the vein into the surrounding tissue causing oedema and a local inflammatory process within the soft tissues. This leads to the changes of skin pigmentation skin (haemosiderin staining – Figure 1) and tissue fibrosis (lipodermatosclerosis – Figure 2).
- Other factors can contribute to venous hypertension. These include obesity and poor mobility. Other causes of leg swelling (e.g. heart failure) may also contribute to ulceration.
- Venous ulceration is a direct consequence of venous hypertension and is regarded as 'end stage' of chronic venous disease.

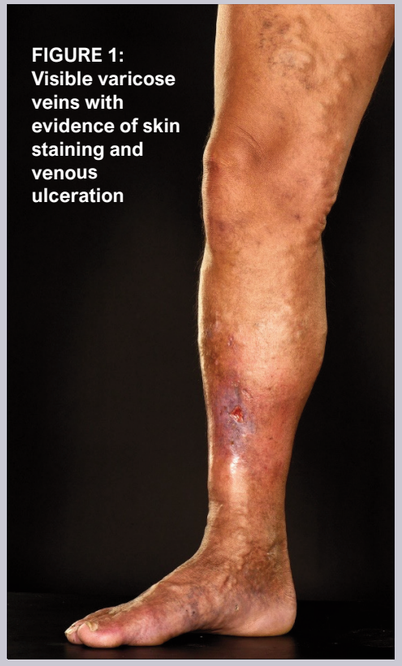


FIGURE 1: Visible varicose veins with evidence of skin staining and venous ulceration

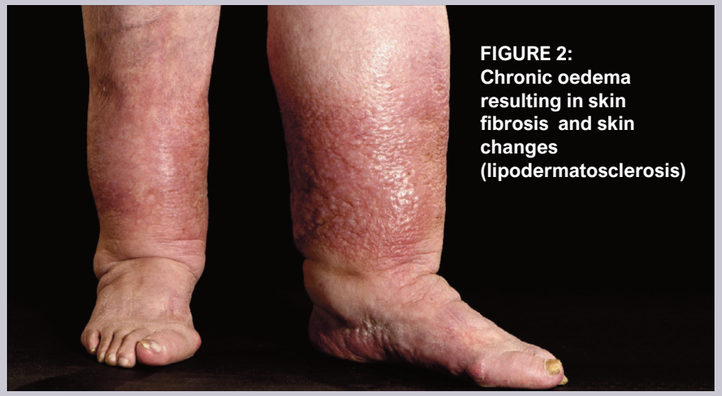


FIGURE 2: Chronic oedema resulting in skin fibrosis and skin changes (lipodermatosclerosis)

Assessment

- A holistic assessment of the patient should include obtaining an appropriate medical history, considering the patient factors, the appearance of the limb and wound assessment.
- Other causes of ulceration be considered including lower limb peripheral arterial disease (PAD) which can occur alongside venous hypertension (i.e. mixed ulceration).
- When lower limb ulceration is present, the arterial supply of the affected leg should be objectively assessed by both palpation of the lower limb pulses and by recording the patient's ankle brachial pressure index (ABPI) or toe pressure. This will confirm or refute the diagnosis of PAD and act as a guide for the suitability of compression therapy.
- If the ABPI is greater than 0.8 then strong compression therapy can be safely applied.
- All patients with venous ulceration should have a lower limb venous duplex scan. This will determine whether the venous hypertension is caused by problems within the superficial veins (i.e. varicose veins – which is amenable to treatment) or within the deep veins (reflux disease – which is not amenable to treatment – or occlusive disease).
- Table 1 highlights the classic characteristic between a venous and arterial ulcer, however, in many patients the differential features may not be so pronounced
- Where the ulcer is chronic and failing to heel despite treatment then one should consider malignant transformation and a biopsy of the ulcer edge should be performed.

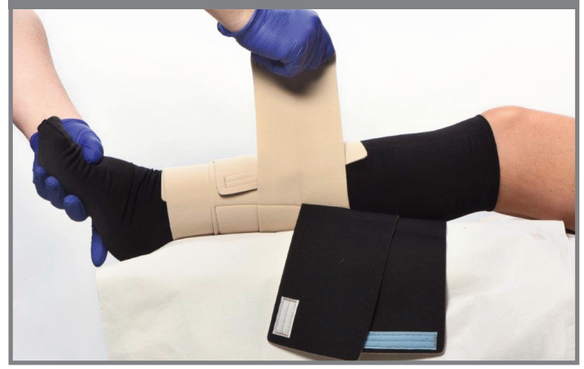
Treatment

- Strong compression therapy (>/= 40 mmHg at the ankle) is the cornerstone of effective management of patient a venous leg ulcer.
- Strong compression is known to decrease time to healing and reduce the risk of recurrence. Strong compression therapy can be applied in many ways – compression hosiery kits (Figure 3), multilayer compression bandages (using 2 or 4 components) (Figure 4) and compression wrap systems (Figure 5). They are all designed to provide 40 mmHg of compression at the ankle.

FIGURE 3: COMPRESSION HOSIERY KIT

FIGURE 4: 2 LAYER COMPRESSION BANDAGE SYSTEM

FIGURE 5: COMPRESSION WRAP SYSTEM



- The choice of which compression system is based on a number of factors, the therapeutic reason for compression (to aid healing or prevent recurrence), the amount of reducible oedema, the levels of exudate and the patients desire to be able to self care. Where indicated patients should be considered for superficial venous intervention (see Chapter 16). There is strong evidence to support a strategy of treating superficial venous reflux in the context of an active / healed venous ulcer.
  - **The ESCHAR study**. This study showed that once a venous ulcer was healed, superficial venous intervention reduced the episodes of recurrence at 4 years in patients wearing compression stocking from 31% to 56%.
  - **The EVRA study**. This study looked at the effect of superficial venous intervention in patients with an active ulcer. It found that there was a reduction in healing time from 82 days in the control group (compression only) to 56 days in the treatment group (compression and venous intervention). Further in the treatment group wound healing was achieved in 85.6% of patients at 24 weeks post treatment.
- If the iliac veins are occluded due to a previous deep vein thrombosis then iliac vein stent insertion can be considered with the aim of reducing the degree of venous hypertension.
- Patients with venous leg ulceration should heal within 12 weeks, if failing to heal despite evidence-based care consider escalation to specialist services.

TABLE 1: CLASSIC DIFFERENCES BETWEEN VENOUS AND ARTERIAL ULCERS

	Venous ulcer	Arterial ulcer
Patient factors	High Body Mass Index	History of arterial problems in other vascular beds (e.g. stroke, heart attack)
	Previous history of a deep vein thrombosis (DVT)	History of factors that are associated with arterial disease – diabetes mellitus, smoking, hypertension
	Immobility	A history in keeping with intermittent claudication (i.e. muscle cramps brought on by walking and relieved by rest)
Limb related assessment	Presence of oedema	
	Evidence of skin changes in keeping with venous hypertension: • Haemosiderin deposition • Venous eczema • Erythema around gaiter area • Lipodermatosclerosis • Evidence of varicose veins	Pain (although may be absent in patients with diabetes due to neuropathy)
		Shiny skin
		A red foot ('sunset foot')
	Wound in gaiter area	Wound on the foot
		Hairless skin
Wound assessment	Flat	Punched out
	Irregular	Round
	High Exudate	Often dry
	Evidence of granulation / sloughy tissue	Sloughy / necrotic tissue
	Develops over weeks	Quick to develop

18 DEEP VENOUS DISEASE

Sam Galea, Emma Wilton

WHAT IS DEEP VENOUS DISEASE?

- Chronic venous disease is due to elevated ambulatory pressure within the lower limb leading to venous hypertension.
- This can be due to either deep or superficial venous disease.
- The most severe outcome of chronic venous disease is the formation of a venous leg ulcer.

Aetiology

Deep venous obstruction

- Acute - deep venous thrombosis (DVT)
- Chronic
  - Thrombotic
    - Post-thrombotic syndrome (PTS)
  - Non-thrombotic
    - Non-malignant iliac vein and IVC outflow obstruction
      - Congenital
      - May-Thurner syndrome (secondary to overlying crossing artery) / non-thrombotic iliac vein lesion (NIVL)
    - Malignant iliac vein / IVC external compression

Deep venous incompetence

- Valvular damage secondary to DVT
- Congenital

DEEP VENOUS OBSTRUCTION

Acute DVT

The annual incidence of acute DVT is 1 per 1000 people/year.

Aetiology

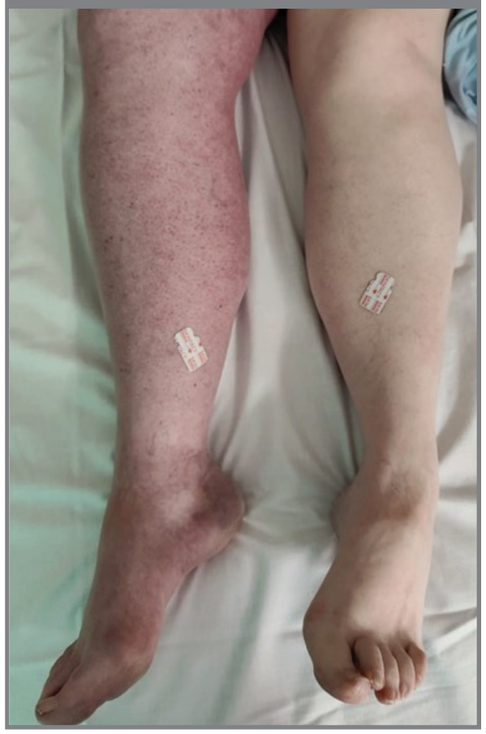
- Unprovoked DVT – unknown cause; pose a higher long-term risk of recurrence.
- Provoked DVT - secondary to changes in any of the domains of the Virchow Triad

Clinical presentation

Symptoms and signs (Figure 1):

- Pain in the leg
- Swelling of the leg
- Warm / hot skin of the leg
- Red skin of the affected leg
- Tenderness
- Venous claudication
- Phlegmasia cerulea dolens
  - The most extreme clinical presentation of DVT
    - Occurs when there is occlusion of the common femoral vein and external iliac veins, completely obstructing the outflow of all deep and superficial veins of the limb, as well as collaterals.
    - Symptoms include sudden severe ischaemic pain, massive congestion of the limb, cyanosis, sensory / motor disturbance, tachycardia and shock. It may also be complicated by massive PE, compartment syndrome and, potentially, lead to venous gangrene.

FIGURE 1: RIGHT LOWER LIMB ACUTE DVT



Pre-test probability score

Wells DVT score

Investigation

- Duplex ultrasound (including compression ultrasound)
- Cross-sectional imaging
  - Computed tomography venography
  - Magnetic resonance venography

Treatment

Aims to reduce the risk of PE and propagation of the DVT. Treatment also aims to reduce the risk of developing PTS.

- Anticoagulation with the use of warfarin or direct oral anticoagulant
- Compression therapy as tolerated - check for pedal pulses
- Limb elevation
- Early thrombus removal in selected patients with symptomatic iliofemoral DVT
  - Endovenous thrombectomy +/- iliac vein stenting
    - Mechanical thrombectomy
    - Aspiration thrombectomy
    - Pharmacomechanical thrombectomy
- Catheter directed thrombolysis +/- iliac vein stenting

Chronic deep venous obstruction

Post thrombotic syndrome (PTS)

Aetiology

The fibrinolytic pathway completely dissolves acute thrombus but in 30–50% of patients this is incomplete and results in some form of deep venous scarring e.g., synechia, residual obstruction and valvular incompetence. This is termed post-thrombotic syndrome (PTS). It has a significant impact on quality of life. PTS is graded using the Villalta score. The risk of PTS increases in proximal DVTs. Up to 10% of patients will go on to develop severe PTS with the formation of venous leg ulceration over a 10-year period. Symptoms of PTS usually develop within 2 years of the DVT. This also adds a significant healthcare burden.

Clinical presentation

Symptoms and signs:

- Aching / cramping / heaviness / venous claudication
- Pruritus
- Limb swelling / skin discolouration / venous ulceration
- Varicose veins

PTS risk factors

- Proximal DVT (2–3 times higher)
- Previous DVT
- Obesity
- Pre-existing venous insufficiency
- Higher baseline Villalta score
- Persistent venous symptoms at 1 month
- Residual thrombus on US

Investigation

- Venous duplex of lower limb with full assessment of deep and superficial systems
  - DVT / chronic scarring / deep venous reflux / superficial venous reflux
- CT venogram/ MR venogram
  - Deep vein obstruction / fibrotic scarring / collaterals / May–Thurner syndrome (MTS) compression / malignancy
- Endovenous imaging
  - Direct venography and intravascular ultrasound – to confirm adequate inflow prior to undertaking venous stenting in appropriately selected patients.

Treatment

- Compression therapy / limb elevation / Exercise

  In severe cases of PTS endovascular iliac vein stenting can be considered in appropriately selected patients. For this to be successful it is imperative to have adequate inflow from the femoral / profunda vein. Patients undergoing stenting will need to adhere to a post-operative anti-coagulation and surveillance protocol which is tailored to the individual's risk profile.

Non-thrombotic iliac venous outflow obstruction

Aetiology

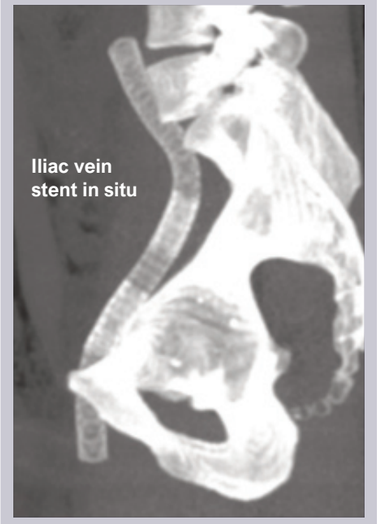
Non-thrombotic iliac vein lesions (NIVLs) are due to extrinsic compression of the iliac vein, commonly between arterial structures and the spine, without associated thrombosis. It is present in up to 70% of the asymptomatic population. The compression results in intrinsic vein stenosis characterised by wall fibrosis or intraluminal webs / spurs. The most common cause of a NIVL is commonly referred to as MTS. This is due to compression of the left common iliac vein by the right common iliac artery.

Clinical presentation

- Asymptomatic
- Symptoms of chronic venous disease e.g., leg discomfort, limb swelling, varicose veins and venous leg ulceration
- Pelvic venous insufficiency – pelvic pain and dyspareunia

Treatment

For symptomatic patients with NIVL, percutaneous iliac venous stenting is a safe and effective treatment option compared with medical management alone.



DEEP VENOUS INCOMPETENCE (DVI)

Aetiology

DVI most commonly results from venous reflux due to faulty valve function developing as a long-term consequence of DVT and recanalisation. It may also be due to primary valvular incompetence without previous episode(s) of DVT. The prevalence of DVI is 10–15% and it is more common in women.

Clinical presentation

Symptoms of chronic venous insufficiency (CVI):

- Pain / ache in limbs / swelling of the limb / oedema
- Venous skin changes / venous leg ulceration

Investigation

- Duplex ultrasound
- Venous anatomy
- Valve incompetence (reflux)
  - Retrograde blood flow with duration >1s in common femoral vein, femoral vein and popliteal vein is pathological
  - Retrograde blood flow with duration >0.5s in superficial and perforator veins is pathological
- Venous obstruction

Treatment

This is focused on symptom control and prevention of venous leg ulcer formation.

- Compression therapy – compression bandaging/hosiery (see Chapter 17)
- Exercise
- Novel experimental therapies such as endovenous valve creation/insertion of neo-valve

THEME 5

MISCELLANEOUS

19 CAROTID ARTERY DISEASE

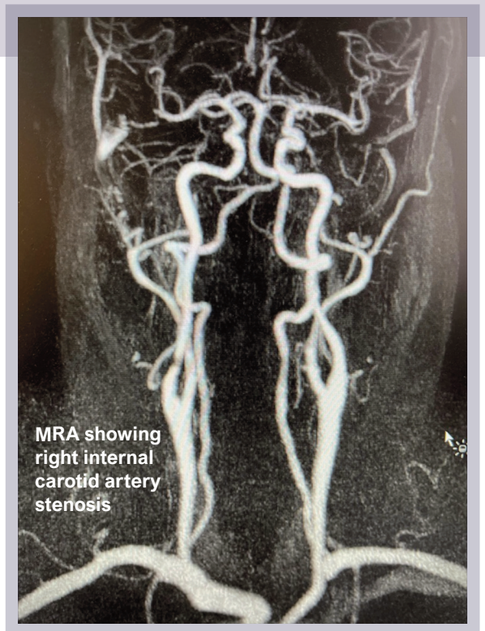
Lewis Meecham

WHAT IS CAROTID ARTERIAL DISEASE?

- Carotid arterial disease refers to the narrowing or blockage of the carotid arteries, which are responsible for supplying blood to the brain. This is primarily due to atherosclerosis.
- As the plaque evolves, it can lead to significant stenosis with a risk of plaque rupture or complete occlusion of the carotid arteries, disrupting blood flow to the brain and increasing the risk of stroke.

Clinical presentation

The vast majority of carotid artery disease is asymptomatic. However, when symptoms do arise, they may include transient ischaemic attacks (TIAs), strokes or amaurosis fugax. TIAs manifest as temporary episodes of neurological dysfunction, such as sudden weakness or numbness in the face, arm, or leg, difficulty speaking, and visual disturbances in the form of transient vision loss (amaurosis fugax) with resolution of symptoms within 24 hours of onset. Strokes, on the other hand, result in more permanent neurological deficits.



MRA showing right internal carotid artery stenosis

Diagnosis

History

- A detailed patient history is crucial in identifying symptoms associated with carotid arterial disease. Symptoms of focal neurological deficits; arm/leg weakness, vision loss, facial droop, inability to speak, etc are all suggestive of a cerebrovascular accident occurring in the carotid artery territory. Unilateral symptoms are a key predominant sign. Cardiovascular risk factors should also be addressed.

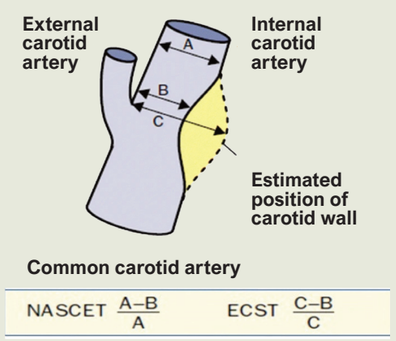
Examination

- Neurological examination should also be performed to assess for any focal deficits.

Imaging

- Duplex ultrasound is the initial imaging modality of choice, providing information about the degree of stenosis and plaque morphology. NASCET (North American Symptomatic Carotid Endarterectomy Trial) and ECST (European Carotid Surgery Trial) criteria are used in assessing the degree of stenosis on duplex (see Table 1). Significant stenosis is defined either as a 50% NASCET or 70% ECST stenosis. Computed tomography angiography (CTA) and magnetic resonance angiography (MRA) are also valuable tools for further assessment and management planning. In the UK, two forms of imaging are used to determine / confirm the stenosis prior to undergoing carotid artery intervention.

TABLE 1 CAROTID ARTERY STENOSIS MEASUREMENT CRITERIA ON DUPLEX ULTRASOUND



NASCET	ECST
30	65
40	70
50	75
60	80
70	85
80	91
90	97

Approximate equivalent degrees of internal carotid artery stenosis used in NASCET and ESCT according to recent direct comparisons

Differential diagnosis

Carotid arterial disease leading to TIA or stroke can share symptoms with other conditions, necessitating a careful evaluation to establish an accurate diagnosis. Differential diagnoses may include temporal arteritis, vertebrobasilar insufficiency, cervical spine pathology, cardiac sources of emboli, and other vascular disorders such as vertebral artery stenosis, intracranial arterial stenosis, and vasculitis. Cardiac sources of emboli, such as atrial fibrillation or valvular heart disease, are the most common and should be evaluated through electrocardiogram and echocardiography.

Management of carotid arterial disease

The management of symptomatic carotid vascular disease involves both medical and surgical interventions. Medical management aims to control risk factors and reduce the risk of further sequelae of the disease. This includes lifestyle modifications, and pharmacological therapy.

Surgical interventions are often considered for patients with significant carotid stenosis (>50% NASCET criteria) in the form of carotid endarterectomy (CEA) or carotid artery stenting (CAS). CEA involves the surgical removal of plaque from the carotid artery, while §CAS involves the placement of a stent to widen the narrowed artery. Both procedures aim to reduce the plaque burden within the artery to the brain and reduce the risk of stroke. The choice between CEA and CAS depends on various factors, including the patient's age, comorbidities, and anatomical considerations.

Medical management
This should involve lifestyle modifications and optimisation of medical therapy, known as best medical therapy (BMT). This includes smoking cessation and aggressive control of hypertension, hyperlipidaemia, and diabetes. Antiplatelet therapy, such as clopidogrel or aspirin, should be prescribed to reduce the risk of thromboembolic events and patients should have optimal lipid lowering therapy usually in the form of high intensity statin therapy (Atorvastatin 80 mg nocte).

Revascularisation strategies
Revascularisation is considered in patients with significant symptomatic carotid stenosis (>50% NASCET criteria). A decision on intervention is determined based upon the risk of stroke versus the risk of intervention and is determined by the degree of stenosis and time from event. Two main revascularisation approaches include carotid endarterectomy (CEA) and carotid artery stenting (CAS).

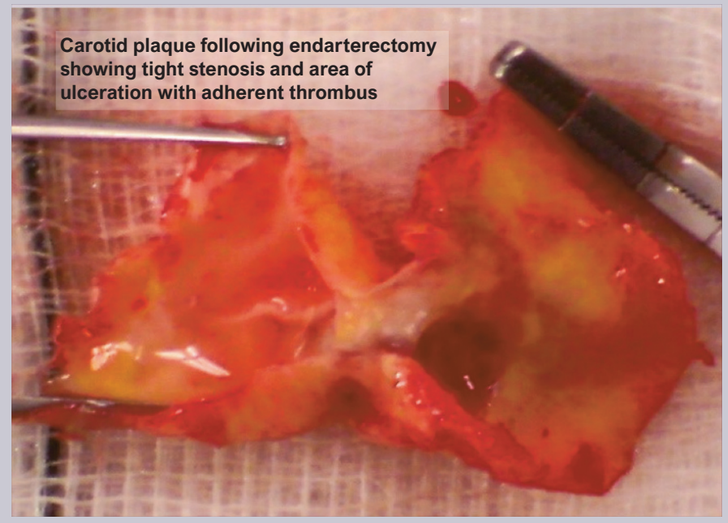
The choice of procedure depends on various factors such as the patient's overall health, comorbidities, and individual risk profile.

Carotid endarterectomy (CEA)

This involves making an incision in the neck, exposing the carotid artery, and removing the plaque causing the stenosis. It is considered the gold standard for revascularisation in patients with carotid arterial disease. CEA has been shown to significantly reduce the risk of stroke in symptomatic patients with significant carotid stenosis. Complications may occur and include stroke, myocardial infarction, bleeding, infection, and cranial nerve injury. Patient selection is important and should consider factors such as age, comorbidities, and the presence of contralateral carotid occlusion.

Carotid artery stenting (CAS)

A less invasive alternative to CEA. It involves the placement of a stent into the narrowed carotid artery to open it up and improve blood flow. CAS is typically performed using a percutaneous approach in the groin. CAS is typically considered in patients who are at high surgical risk or have anatomical factors that make CEA challenging. An example of this is a patient who has had previous radiotherapy to the neck. CAS carries a higher risk of early stroke when compared to CEA with other associated risks including stent-related complications. However, it comes with less risks to the heart and a limited risk of any nerve injury. Careful patient selection and operator expertise are crucial for optimal outcomes with CAS.



Carotid plaque following endarterectomy showing tight stenosis and area of ulceration with adherent thrombus

Management of asymptomatic carotid artery stenosis

Carotid artery stenosis affects around 3% of individuals aged > 60 years and is responsible for up to 15% of all ischaemic strokes. While patients with asymptomatic carotid stenosis (defined as the presence of carotid arterial disease but not having had a previous stroke or TIA) have a low risk of stroke with BMT, it is uncertain whether the benefits of carotid surgery outweigh the risks. The European Society for Vascular Surgery (ESVS) latest guidelines recommend that individuals with an asymptomatic carotid stenosis of 60–99% and average surgical risk be considered for carotid endarterectomy (CEA) only if they exhibit certain characteristics associated with a higher risk of stroke. In high-risk patients who are not suitable for surgery, carotid artery stenting (CAS) may be indicated as long as the peri-procedural risk of stroke or death is less than 3% and the patient has a life expectancy of over 5 years.

20 THORACIC OUTLET SYNDROME AND VASOSPASTIC DISEASE

Chandana Wijewardena, Aminder Singh, Henry Bergman, Andrew Winterbottom

WHAT IS THORACIC OUTLET COMPRESSION SYNDROME (TOCS)?

- The thoracic outlet, located between the thorax and the axilla, is the region where the subclavian artery, subclavian vein and brachial plexus travel from the thorax through to reach the upper limb.
- TOCS includes a variety of symptoms due to compression of neurovascular structures within the thoracic outlet. Symptoms are related to the structure being compressed.
- Common compression points include between scalene muscles, between clavicle and first rib and under pectoralis minor. A cervical rib is an important bony cause of TOCS.
- TOCS affects women more than men.
- Over 90% of patients have neurological symptoms such as pain or weakness from compression of nerve roots of the brachial plexus.
- Compression of the subclavian artery can lead to upper limb claudication or acute limb ischemia. Compression of the subclavian vein can result in deep venous thrombosis.

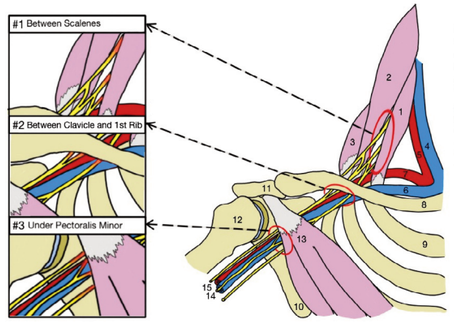
Neurological TOCS

- **History**: commonest symptoms include pain, paraesthesia, and weakness. Location of paraesthesia is determined by which nerve root is compressed.
- **Examination**: tenderness and paraesthesia in response to pressure over scalene muscles may be present. Asking the patient to rotate their head and tilt away from affected side may reproduce pain. Roo's test involves abducting arm to 90°C in external rotation with finger clenching to reproduce symptoms.
- **Diagnostic tests**: a scalene muscle block with local anaesthetic leading to symptom improvement suggests surgical treatment would be effective. A duplex ultrasound to assess the subclavian artery and vein is required, as well as Xray for cervical rib (Figure 1). Neurophysiology and MRI can exclude alternative causes such as cervical root compression.
- **Differential diagnosis**: nerve compression at cervical root (e.g. disc herniation) or in the upper limb (e.g. carpel tunnel) causing neurological symptoms.
- Conservative management is first line with targeted physiotherapy and often significantly improves symptoms in most patients.
- Surgery is offered to those failing conservative management. It involves resection of the 1st rib on the affected side and other bony abnormalities identified, such as a cervical rib.

Arterial TOCS

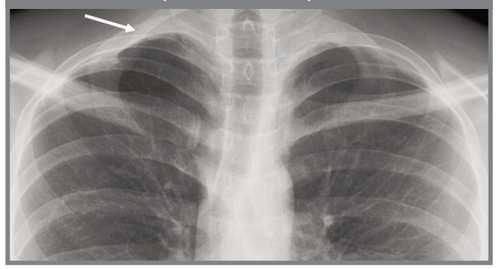
- Usually associated with compression of subclavian artery from bony structures (clavicle and first rib) with time leads to arterial wall damage and post stenotic dilatation.
- Clots formed within the post stenotic dilatation can lead to distal embolisation.
- **History**: upper limb claudication or thromboembolic disease-causing ischaemia. Severe cases may present with digital gangrene.
- **Examination**: full vascular examination. Look for signs of ischaemia such as ulcers or embolic changes.

Demonstration of common compression points between (1) scalene muscles, (2) between clavicle and first rib and (3) under pectoralis minor.



1. Anterior scalene
2. Middle scalene
3. Posterior scalene
4. Internal jugular vein
5. Common carotid artery
6. Subclavian vein
7. Subclavian artery
8. Clavicle
9. First rib
10. Scapula
11. Acromion process
12. Humerus
13. Pectoralis minor
14. Axillary vein
15. Axillary artery

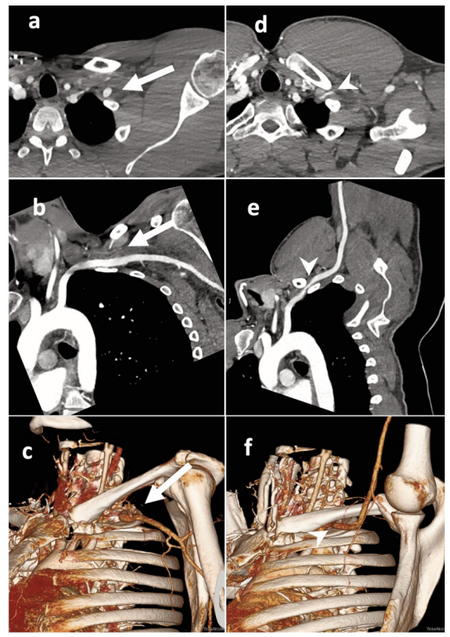
FIGURE 1: CHEST X RAY SHOWING A RIGHT CERVICAL RIB (WHITE ARROW)



- **Diagnostic tests**: Duplex ultrasound of arteries in neutral and stress positions. Computed tomography (CT) with arterial contrast with arms up and down (dynamic CT) positions can demonstrate arterial compression (Figure 2). MR angiography.
- **Differential diagnosis:** Can mimic Raynaud's phenomenon.
- Arterial TOCS with subclavian artery damage should be treated with surgery. If the cause of the compression is a cervical rib, this should be resected. If the compression is proven to be between the 1st rib and the clavicle, resection of 1st rib is indicated. Depending on the extent of the damage to the subclavian artery, arterial reconstruction with patch repair or bypass graft may be undertaken at the time of 1st rib or cervical rib resection identified, such as a cervical rib.

FIGURE 2: VOLUME RENDERED CT ANGIOGRAM IN ARMS DOWN POSITION

a) with normal thoracic outlet and normal subclavian artery (white arrow). Volume rendered CT angiogram
b) and curved plane reformat
c) in arms up position
d-f) showing significant compression of the subclavian artery by the clavicle and 1st rib in the abnormal thoracic outlet (white arrow head).



Venous TOCS

- Primary subclavian vein thrombosis due to compression is also known as Paget-Schroetter syndrome.
- **History**: swelling of arm, pain worse with exercise, cyanosis, venous claudication.
- **Examination**: unilateral swelling of arm. Pulses should be present. Rare cases can have signs of venous gangrene.
- **Diagnostic tests**: Duplex ultrasound is first line followed by CT or MR venography.
- **Management**: conservative management with arm elevation and a compressive sleeve provides symptomatic relief in mild to moderate cases. Pharmacological management with anticoagulation. Catheter directed thrombolysis or surgical thrombectomy can be considered in those with a severe presentation. Surgical resection of first rib would be required after initial management to prevent further episodes.

Vasospastic disorders

- Due to vasospasm of digital arteries leading to reduced blood flow. Usually affects fingers.
- Initially digit colour turns white due to vasospasm, then blue from cyanosis and then red from reactive hyperaemia.
- Predominantly affects females.

Raynaud's phenomenon

- The exact cause is unknown, but it is suggested abnormal red blood cell, white blood cell and platelet function lead to aggregation in the microcirculation. Dysfunction in the peripheral nervous system can lead to vasospasm alongside inflammatory conditions.
- Primary Raynaud's phenomenon occurs on its own and it's the most common type.
- Secondary Raynaud's could be due to connective tissue disease, vascular disease, mechanical or environmental factors or drugs (Table 1).
- **Diagnostic tests**: hand cooling and pressure drop assessment or nail fold capillaroscopy. Blood tests to investigate secondary causes include full blood count, urea and electrolytes, erythrocyte sedimentation rate, rheumatoid factor, and antinuclear antibodies.
- **Management**: conservative with advice of using heating aids which can help with symptom relief. Drug therapy with calcium channel blockers or prostaglandins can be used to promote vasodilation. Sympathectomy and surgery for more significant ischaemia is rarely required. In secondary Raynaud's, treatment of the underlying cause is essential.

TABLE 1: SECONDARY RAYNAUD'S CAUSES

Connective tissue disease	Rheumatoid arthritis Scleroderma Sjogren's syndrome Systemic lupus erythematosus Takayasu's arteritis
Vascular disease	Atherosclerosis Thromboangiitis obliterans (Buerger's disease) Thromboembolism TOCS
Mechanical or environmental	Jobs involving repeated actions or vibration Smoking Trauma
Drugs	Beta blockers Chemotherapy Combined oral contraceptive pill Ergot derivatives Interferon
Other	Diabetes Hypothyroidism Malignancy

21 MANAGEMENT OF PATIENTS WITH RENAL DISEASE

Jing Yi Kwan, Jon De Siqueira

DIALYSIS ACCESS IN PATIENTS WITH END-STAGE RENAL DISEASE

- The ideal vascular access should provide safe and effective haemodialysis by enabling the removal and return of blood via an extracorporeal circuit.
- Routes for haemodialysis vascular access include (1) arteriovenous fistula (AVF), (2) prosthetic graft, and (3) venous catheters (tunnelled and non-tunnelled). The Renal Association guidelines (UK) recommend use of AVF first, AV graft as the second option and venous catheters as the last option.
- Peritoneal dialysis is an alternative where peritoneal catheters are placed to allow dialysate to be instilled directly into the intraperitoneal space where it remains in contact with the peritoneal membrane before being removed.

Assessment of a patient for haemodialysis access creation

History

- Hand dominance, previous vascular access, upper and lower extremity venous thrombosis, symptoms of hand ischaemia.

Investigations

- Duplex venous ultrasound – A non-phlebitic, non-calcified vein of minimum 3.0 mm diameter is preferred. The best conduit is superficial, easily identified, straight and of large calibre.

Arteriovenous fistula creation

- The Society of Vascular Surgery recommends placing access as distal in the upper extremity as possible to preserve future central access while giving preference to the non-dominant arm.
- The preferred order of fistula creation is a radial artery to cephalic vein (radiocephalic) fistula, brachiocephalic fistula, and then transposed brachiobasilic fistula. A radiocephalic fistula has a high rate (30–50%) of non-maturation.
- When the upper arm cephalic, basilic, or brachial veins are being utilised, a two-stage approach is advocated due to increased patency rates. The first stage is the anastomosis of a primary artery to vein through a local incision. The second stage involves the transposition of a mature vein superficially for identification in cannulation.

Arteriovenous loop graft

When there is no autogenous option for AV fistula creation then a non-autogenous reconstruction is created. There are several options:

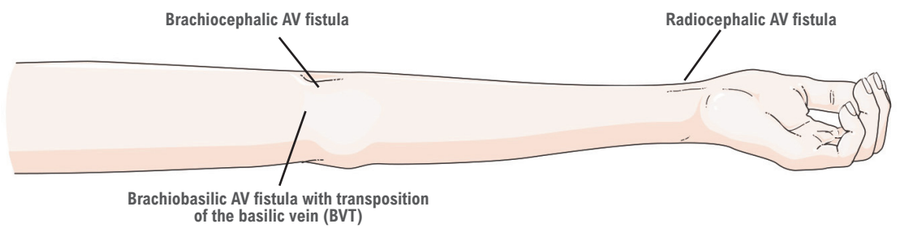
- Expanded polytetrafluoroethylene (PTFE) grafts can be cannulated 2–3 weeks after graft creation.
- A new PTFE-based graft known as the Accuseal graft allows for cannulation within 24 hours by way of a mid-layer sealing membrane.

Complications

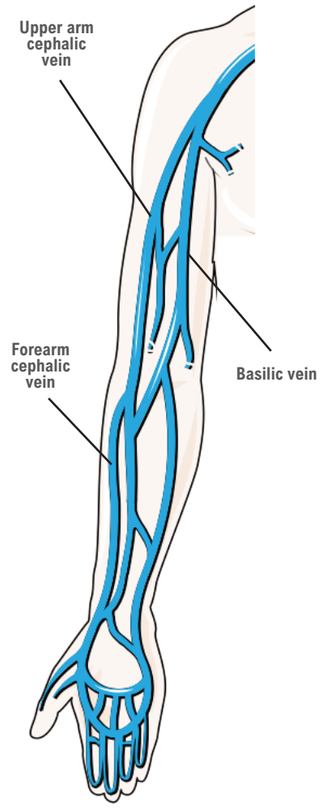
- Immediate complications – Pain, bleeding, haematoma, oedema, loss of thrill secondary to acute thrombosis or intraarterial flap.
- Early / late complications – Failure of maturation, infection, stricture, venous hypertension, ischaemic steal syndrome, aneurysm formation, neuropathy.

Endovascular or surgical interventions may be warranted to revise the existing fistula.

COMMON SITES FOR AV FISTULA FORMATION



DIAGRAMMATIC REPRESENTATION OF THE CEPHALIC AND BASILIC VEINS.



Central venous catheters (CVCs)

Indications

- Advanced age at initiation of HD, patients awaiting AVF maturation.

Contraindications

- Local infection, thrombosis or stenosis of target vein.

Complications

Bleeding, air embolism, pneumothorax, cardiac perforation, arrhythmias, central vein stenosis.

- Central venous access is obtained via the internal jugular vein, subclavian vein, and femoral veins.
- Subcutaneously tunnelled CVCs travel under the skin away from the point of venous entry and provide long-term intravenous access for haemodialysis. They can remain in place for weeks to months.
- Non-tunnelled catheters should only be placed as the last resort or in emergency situations when more permanent access is not available for dialysis. They need to be exchanged every few days to a week.

BOX 1

Rule of 6's for arteriovenous fistulas

- The rule of 6's is a widely used informal guide to determine when an AVF will support dialysis.
- It states that 6 weeks after creation, an AVF should achieve a blood flow rate of at least 600 mL/min, a diameter of at least 6 mm, an access length of 6 cm for cannulation, and a depth of 6 mm or less from the skin surface.
- It is paramount for clinicians to learn how to access for fistula thrill.

Peritoneal dialysis (PD)

Indications

- Haemodynamically labile patients with poor cardiovascular function who may tolerate haemodialysis poorly.

Contraindications

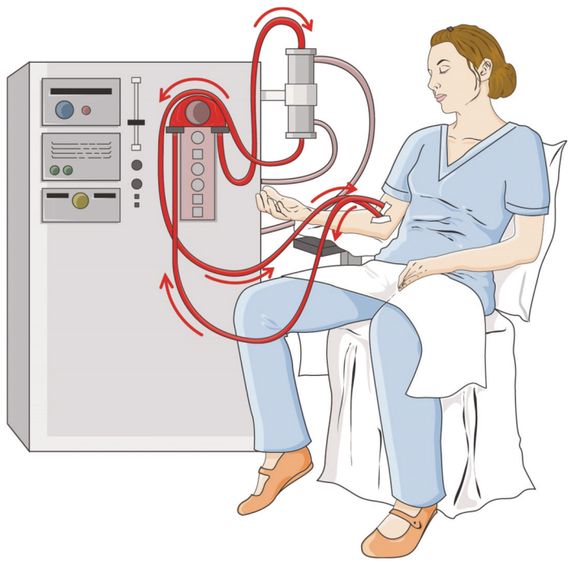
- Inflammatory abdominal conditions, unrepaired hernia.

Complications

Peritonitis, hernia formation, catheter migration and malposition.

- PD catheters can be placed either by an open or laparoscopic surgical technique.
- The borders of the rectus muscle are preferred insertion sites to avoid the superficial and inferior epigastric arteries.
- When using a Tenckhoff catheter with two cuffs, the deeper cuff should rest within the pre-peritoneal space in the rectus sheath, and the superficial cuff should lie 2–3 cm medial to the superficial wound. Placing the deeper cuff outside the rectus muscle may lead to less tissue ingrowth, increasing the likelihood of leakage and herniation. If the superficial cuff placement is too deep, serous fluid may collect in the space outside of the cuff, leading to skin irritation and infection.

DIAGRAMMATIC REPRESENTATION OF SET UP FOR RENAL DIALYSIS USING AN A-V FISTULA



22 ACUTE LIMB ISCHAEMIA

Thomas Lyons, Julien Al Shakarchi

WHAT IS ACUTE LIMB ISCHAEMIA?

- Acute limb ischaemia (ALI) is mostly synonymous with an acute arterial occlusion and is defined as a sudden reduction in limb perfusion that may threaten a limb's viability. It can occur in any peripheral artery of the upper and lower extremities.
- It is a vascular emergency which needs early recognition, prompt diagnosis and intervention. Left untreated it may lead to major limb amputation.
- ALI classically presents with the 6Ps of limb ischaemia: Pain, Perishingly cold, Pallor, Pulselessness, Paraesthesia and Paralysis
- ALI can be managed medically (intravenous heparin), endovascularly (thrombolysis or thrombectomy) or surgically (embolectomy).

BOX 1

Aetiology

Thrombosis

- Atherosclerotic – plaque rupture
- Graft / stent occlusion
- Popliteal aneurysm

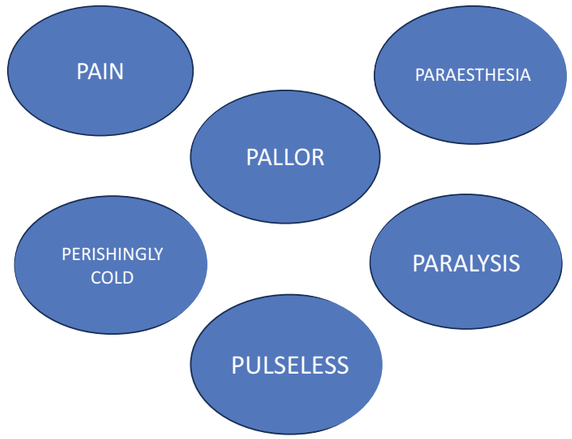
Embolism

- Cardiac (e.g. atrial fibrillation (AF), myocardial infarction (MI), infective endocarditis)
- Non-cardiac (e.g. atherosclerotic disease, aneurysm)
- The embolism classically lodges at an arterial bifurcation

Trauma

Arterial dissection

6 signs of acute limb ischaemia



How to diagnose acute limb ischaemia

History

- The classical clinical presentation of acute limb ischaemia are the 6Ps of limb ischaemia: Pain (severe and resistant to analgesia), Perishingly cold, Pallor, Pulselessness, Paraesthesia and Paralysis.
- Risk factors for vascular disease: history of intermittent claudication, cardiac history (atrial fibrillation, ischaemic heart disease including recent myocardial infarction), diabetes, smoking history and any previous intervention.
- Recent cessation of anticoagulation.

Examination

- Look for obvious signs of ALI – pallor, mottling, fixed-mottling
- Assessment of lower limb pulses (also comparison with pulses of the non-affected limb)
- Palpation (temperature and muscle compartments)
- Assess the neurological status of the limb (sensory and motor)

Imaging

- First line imaging is commonly CT angiography which provides cross sectional imaging and is fast and usually the most readily available imaging modality.
- Prompt imaging is required to allow planning for intervention, however imaging should not delay referral with the vascular on-call team and clinical findings should trigger urgent review.

RUTHERFORD CLASSIFICATION FOR ALI GRADES THE SEVERITY OF ALI AND RISK OF LIMB LOSS

Category	Description	Capillary return	Motor innervation	Sensory innervation	Arterial doppler	Venous doppler
I – Viable	Not immediately threatened	Intact	Intact	Intact	Audible	Audible
IIa – Threatened	Salvageable if promptly treated	Intact/slow	Intact	Partial loss	Inaudible	Audible
IIb – Threatened	Salvageable if immediately treated	Slow/absent	Partial loss	Partial loss	Inaudible	Audible
III - Irreversible	Primary amputation	Absent/staining	Absent/fixed	Complete loss/ insensate	Inaudible	inaudible

Management of acute limb ischaemia

Initial management

- A to E assessment in accordance with CCrISP guidelines.
- Initiate oxygen to optimise physiology unless concerns regarding CO_2 retention
- Bedside tests include: routine bloods including a FBC, UEs, clotting and crossmatch. ECG to look for AF.
- Bolus 5000 IU of IV heparin

Medical

- Anticoagulation, initially with IV heparin bolus (check local guidelines).
- Following the bolus, an infusion is started initially at a rate determined by weight followed by the APTT ratio thereafter.

Endovascular

- Thrombolysis
  - Use of a thrombolytic agent (t-PA or urokinase) to dissolve a thrombus.
  - The thrombolytic agent is given directly to the site of the thrombus (intra-arterial), with percutaneous access to place a catheter within the thrombus to deliver the thrombolytic agent locally. Contraindications:
    - Active internal bleeding
    - Risks associated with bleeding
    - Pregnancy
    - Stroke/TIA within 2 months
    - Vascular or abdominal surgery within 2 weeks
- Thrombectomy
  - Percutaneous thrombectomy
    - Aspiration
    - Mechanical

Surgical

Embolectomy – most commonly performed procedure is a femoral embolectomy with less commonly performed brachial and popliteal embolectomy.

- Expose artery and control inflow and outflow
- Transverse arteriotomy
- Check inflow
  - If poor inflow, proceed to proximal embolectomy, imaging will also inform as to whether any up stream clot
  - If still poor inflow after embolectomy, then may need to consider some alternative vascular surgical reconstruction
  - If there is good inflow, proceed to distal embolectomy with appropriately sized Fogarty balloon catheter
  - If no improvement after embolectomy, can consider operative thrombolysis, more distal embolectomy or distal bypass. On-table angiography is useful to assess the distal outflow in such circumstances.

Fasciotomies

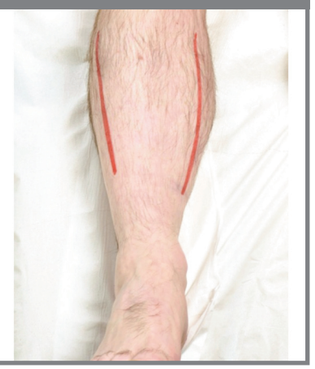
Patients who undergo revascularisation for acute limb ischaemia should be considered for 4 compartment fasciotomies. Two long incisions are made along the lower leg (medial and lateral) to allow access to the compartments where the fascias are incised to release them. It is important to decompress the deep medial compartment.

SURFACE LANDMARKS FOR WHERE TO MAKE THE INCISIONS FOR A 4 COMPARTMENT FASCIOTOMY (RED LINES).

The medial incision is 2cm behind the border of the tibia.

Remember to also decompress the deep medial compartment.

The anterior incision is half way between the tibia and the fibula.



BOX 2

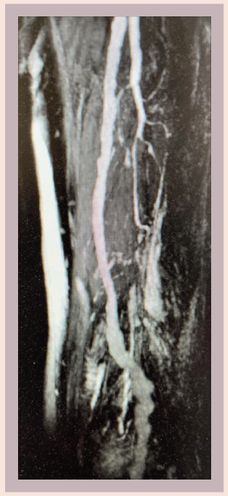
Popliteal artery aneurysms

Popliteal aneurysms are the most common peripheral aneurysm (>80% of peripheral aneurysms) and 50% of popliteal aneurysms are associated with abdominal aortic aneurysms. Risk factors include smoking, atherosclerosis, connective tissue disorder, age, male gender and history of abdominal aortic aneurysm.

Asymptomatic aneurysm of less than 2 cm can be safely monitored with regular duplex imaging. Popliteal aneurysm repair should be considered once the maximal diameter reaches 2.0–2.5 cm.

Acute thrombosis of a popliteal aneurysm poses the highest risk to a limb. Due to the amount of movement at the knee joint, there is increased risk of disintegration of the thrombus leading to microembolism (trash) distally. This can often occur silently hence why there is approximately a 50% limb loss.

In an acutely thrombosed popliteal aneurysm, it is important to ensure there is viable distal run-off. This can be managed with embolectomy +/- thrombolysis to the distal vessels followed by an exclusion bypass of the thrombosed popliteal aneurysm.



MRA showing a left popliteal artery aneurysm

23 MESENTERIC AND OTHER VISCERAL DISEASE

Mary Weisters, Tom Baker

WHAT IS MESENTERIC ISCHAEMIA?

- Mesenteric ischaemia is a condition characterised by reduced or obstructed blood flow to the large and/or small intestine. It can present as an emergency acutely or as a chronic progressive condition.
- The mesenteric arteries supply the gastrointestinal organs

  These include the coeliac trunk, superior mesenteric artery (SMA) and inferior mesenteric artery (IMA), all direct branches of the abdominal aorta.

Primitive Gut Portion	Adult Derivatives	Supplying artery
1. Foregut	Oesophagus, stomach, proximal half of the duodenum, liver and biliary tree	Coeliac trunk
2. Midgut	Distal half of the duodenum, jejunum, ileum, caecum, appendix, ascending colon and most of the transverse colon	SMA
3. Hindgut	Remainder of the transverse colon, descending colon, sigmoid colon, rectum, superior portion of the anal canal	IMA

Acute mesenteric ischaemia

- Acute mesenteric ischaemia occurs due to a sudden reduction in blood supply to the bowel, resulting in bowel ischaemia, bowel necrosis and subsequent death if not treated promptly. Overall mortality from the condition is estimated at 60–80%. The most common cause is an embolic event but there are other rarer causes.
- Aetiology

Embolism	Atrial fibrillation, LV thrombus e.g. after myocardial infarct, aneurysms
Thrombus in situ	Unstable plaque rupture from underlying atherosclerosis
Non-occlusive mesenteric ischaemia "NOMI"	Prolonged hypotension (e.g., hypovolaemic shock / cardiogenic shock), vasoconstriction
Venous occlusion	Mesenteric venous thrombosis

How to diagnose acute mesenteric ischaemia?

History

- Sudden onset severe abdominal pain – diffuse and constant in nature
- +/- Nausea and vomiting
- Often occurs in a setting of an acutely unwell patient e.g. pneumonia, recent myocardial infarction
- May be a history of atrial fibrillation or palpitations/chest pain/shortness of breath or recent cessation of anticoagulation

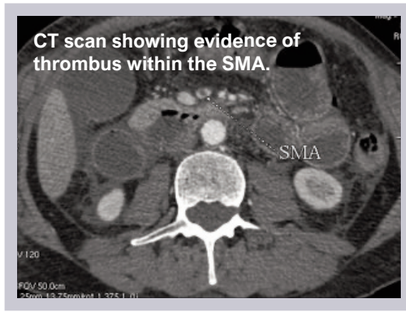
Examination

- Initially abdomen may be soft with generalised tenderness throughout.
- In later stages, where risk of perforation is higher, patients may have signs of generalised peritonism.

Investigations

- Blood gas: metabolic acidosis and raised lactate levels. **NB In late phase with dead bowel lactate can be normal**
- Routine blood tests: FBC, U+Es, coagulation, LFTs.

  Note that if there is coeliac trunk involvement, this may result in hepatic ischaemia and derangement of LFTs and/or INR. The white cell count is usually elevated.



CT scan showing evidence of thrombus within the SMA.

- Imaging:

  CT abdomen and pelvis with arterial, venous and portal venous phase contrast.

  Bowel ischaemia will initially show on CT as oedematous bowel loops.

  This will be progressing in later stages to loss of enhancement in the bowel wall and pneumatosis, with or without evidence of perforation.

  Thrombus in one or more mesenteric arteries.

Management

- Initial management focuses on patient A-E resuscitation, alongside administration of broad-spectrum antibiotics.
- MDT care with involvement of general surgery, vascular surgery and interventional radiology
- Early consideration for involvement of ITU
- Surgical management is the definitive management for acute mesenteric ischaemia, and will focus on two aspects:

  Resection of non-viable / necrotic bowel. Post-operatively this may involve the patient undergoing a re-look laparotomy in 24–48 hours and may require the abdomen to "remain open" after the initial laparotomy.

  Revascularisation of the bowel. This may involve endovascular techniques or open surgical embolectomy.
- **Often this is a 'preterminal event' and management is palliative**

Chronic mesenteric ischaemia

- Chronic mesenteric ischaemia occurs due to reduced blood supply to the bowel, which occurs gradually commonly due to progressive atherosclerosis to the coeliac, SMA and/or IMA. This condition is more common in females. Other risk factors include smoking, diabetes mellitus, hypertension and hypercholesterolaemia.
- Due to the collateral nature of the mesenteric blood supply, at least two of the three mesenteric arteries need to be affected for the patient to become symptomatic. Most often, at least one of these vessels is occluded.

How to diagnose chronic mesenteric ischaemia?

History

- Post-prandial abdominal pain – classically occurs within 4 hours of eating – '**mesenteric angina**'
- Fear of eating
- Significant unintentional weight loss
- Nausea and vomiting sometimes occur
- Loose stools sometimes occur

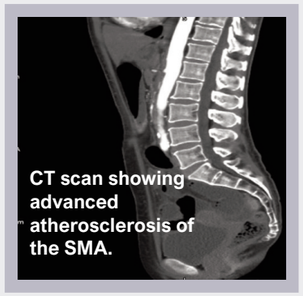
Examination

- Often non-specific, such as mild general tenderness.
- Cachexia / evidence of major weight loss e.g. baggy clothes

Investigations

Comprehensive and often best guided by gastroenterologist. **Chronic mesenteric ischaemia is often a diagnosis of exclusion.**

- Routine blood tests: FBC, U+Es, LFTs may be normal. Electrolytes such as magnesium and phosphate should be checked in presence of malnutrition.
- OGD
- Ultrasound of biliary tree +/- MRCP if deranged LFT or dilated biliary tree
- **CT Angiogram abdominal aorta**


CT scan showing advanced atherosclerosis of the SMA.

Differential diagnosis

- Peptic ulceration/GORD
- Chronic pancreatitis
- Biliary colic
- UGI malignancy

Management

- Management of risk factors for atherosclerosis, to minimise disease progression.

  Lifestyle changes (smoking cessation).

  Medical management with anti-platelet and high-dose statin therapy.
- Revascularisation usually endovascular or occasionally surgical. Intervention is HIGH RISK.

  Endovascular: options are for mesenteric angioplasty and/or stenting.

  Surgical: options are endarterectomy or bypass

Mesenteric aneurysms

True mesenteric aneurysms are rare. The detection of asymptomatic atherosclerotic mesenteric aneurysms has grown in recent years due to increased use of axial imaging (CT scans). They appear as small calcified slow growing aneurysms.

They may occur after a dissection that weakens the artery wall. Occasionally mesenteric aneurysms rupture leading to haemorrhage and death.

Aneurysms of the pancreaticoduodenal and gastroduodenal arcades have a higher risk of rupture and should be treated irrespective of size.

Pseudoaneurysms can occur after severe acute pancreatitis or trauma. They have a high risk of bleeding and also should be treated.

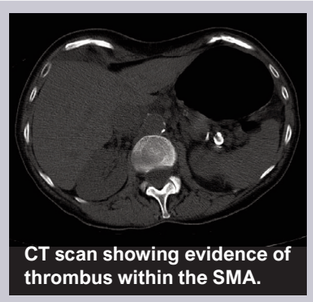
Splenic artery aneurysms should be considered for repair at any size in a woman of childbearing age as the aneurysm can undergo rapid growth and rupture during pregnancy with a risk of maternal and foetal death. Embolism is rare. Thrombosis and thromboembolism often go unnoticed due to the collateral circulation of the gut.

History

- Asymptomatic
- Occasionally abdominal pain

Investigations

- CT angiogram or MRA


CT scan showing evidence of thrombus within the SMA.

Type of aneurysm	Management
True mesenteric aneurysm, symptomatic <2.5 cm	Surveillance
True mesenteric aneurysm >2.5 cm, asymptomatic	Consider endovascular treatment with stent placement or coil embolisation
Symptomatic true mesenteric aneurysm	Endovascular treatment with stent placement or coil embolisation
Mesenteric pseudoaneurysm	Endovascular treatment with stent placement or coil embolisation
Splenic artery aneurysms in a woman of childbearing age	Consider endovascular treatment with stent placement or coil embolisation

ACKNOWLEDGEMENTS OF FIGURES

We would like to acknowledge Smartservier (smart.servier.com) as the source of the following images:

Page 4. The Circle of Willis: https://smart.servier.com/smart_image/circle-of-willis/

Page 4. The extracranial carotid artery: https://smart.servier.com/page/2/?s=Arteries

Page 5. The arterial supply of the upper limb: https://smart.servier.com/page/2/?s=arteries

Page 6. The aorta and the veins of the abdomen: https://smart.servier.com/smart_image/abdominal-aorta/

Page 7. The lower limb arterial and venous suppl: https://smart.servier.com/smart_image/venous-disease-spider-veins/

Page 8. Atherosclerosis (all images): https://smart.servier.com/page/5/?s=atherosclerosis

Page 9. The damaged venous valve: https://smart.servier.com/smart_image/venous-circulation-leg/

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Page 37. The use of open surgical (surgical grafts) in the management of aortic pathology: https://smart.servier.com/page/3/?s=Cardiovascular+system

Page 52. The upper limb: https://smart.servier.com/?s=Arm

Page 53. The venous supply of the upper limb: https://smart.servier.com/?s=veins

Page 53. Diagrammatic representation of renal dialysis: https://smart.servier.com/smart_image/dialysis/

The diagram from chapter 7 is referenced in the text and as follows:

https://www.nice.org.uk/guidance/ng136/resources/visual-summary- pdf-6899919517

We would also like to thank Mid Yorkshire Teaching NHS Trust for the use of the following images:

Page 9. palpation of the doralis pedis pulse

Page 20. palpation of the posterior tibial and dorsalis pedis pulse

Page 21. performing an ABPI

Page 42. Legs that show skin changes due to venous hypertension (2 images)

Page 43. Various methods of applying compression (3 images)

Page 55. Surface markings for the incisions for a 4 compartment fasciotomy

The authors from chapter 20 provided the thorax image from page 5.

The rest of the images have been provided by authors from the book.

NOTE: Links to the images taken from the Servier website are found within the powerpoint presentation

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