Ian A. Trail
Andrew N. M. Fleming
Editors

Disorders of the Hand

Volume 1: Hand Injuries

Springer

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Editors
Ian A. Trail
Wrightington Hospital
Wigan
Lancashire
UK

Andrew N.M. Fleming
St George's Hospital
London
UK

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Preface

In recent years there have been significant advances in the understanding and treatment of disorders of the hand and wrist. This has resulted in a significant improvement in the quality of life for many patients. The authors who have produced this text were chosen as they are hand surgeons who have led many of these exciting developments in the management of both elective and trauma hand surgery. All are internationally respected.

The topics covered are well illustrated with images, radiographs and line drawings and provide practical guidance on surgical procedures. The references at the end of each chapter have been chosen as they are either classic papers or are the most relevant to modern surgical management.

Thus we hope that we have produced a book that will enable improved care for current patients with hand and wrist complaints and inspire surgeons to think in greater detail about treatment options that will provide even better care in the future.

Finally, we would like to thank all the contributors as well as Diane Allmark for her help, but also our families for their patience and support.

Wrightington, Lancashire, UK Ian A. Trail, MBCHB, MD, FRCS (Edin), FRCS (Lon), ECFMG

London, UK Andrew N.M. Fleming, FRCS(Edin), FCS(SA)Plast

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Contributors

Brian D. Adams, MD Department of Orthopaedics, University of Iowa Hospitals and Clinics, Iowa, IA, USA

Sharifah Ahmad Roohi, MD, FRCS, MCh Orth Department of Orthopaedics, Faculty of Medicine and Health Sciences, Universiti Putra Malaysia, Serdang, Selangor, Malaysia

R. Amirfeyz, MD, MSc, FRCS (Trauma and Orth) Department of Orthopaedics, Bristol Royal Infirmary, Bristol, UK

Christopher Armitstead, MBBS (Lond), MRCS (RCSoE), FRCS (Tr & Orth) Department of Hand Surgery, Leighton Hospital, Crewe, Cheshire, UK

Anthony Barabas, BM, BSc, MRCS (Eng), FRCS Department of Plastic Surgery, Hinchingbrooke Hospital, Huntingdon, UK

Richard Barton, MBBS, FRACS (plastics) Plastic and Reconstructive Surgery Unit, The Royal Melbourne Hospital, Melbourne, VIC, Australia

Douglas A. Campbell, ChM, FRCS.Ed, FRCS(Orth), FFSEM(UK) Department of Trauma and Orthopaedic Surgery, Leeds Teaching Hospitals NHS Trust, Leeds, UK

Jeremy Cashman, MB BS, BA, BSc, MD, FRCA, FFPMRCA Anaesthesia and Acute Pain Management, St George's Hospital, London, UK

Louise A. Crawford, FRCS(Tr & Orth) Department of Orthopaedic Surgery, Tameside General Hospital, Ashton Under Lyne, Lancashire, UK

T.R.C. Davis, FRCS Department of Orthopaedics, Nottingham University Hospitals, Queen's Medical Centre, Nottingham, UK

Subodh Deshmukh, MS, MCh (Orth), FRCS, FRCSG, FRCS (Orth) Department of Trauma and Orthopaedic, City Hospital, Dudley Road, Birmingham, UK

David Elliot, MA, FRCS, BM, BCh St Andrews Centre for Plastic Surgery, Broomfield Hospital, Chelmsford, UK

Andrew N.M. Fleming, FRCS(Edin), FCS(SA)Plast Department of Plastic Surgery, St Georges Hospital, London, UK

Grey Giddins, BA, MBBCh, FRCS(Orth) FRCSEd Department of Orthopaedic, The Royal United Hospital, Bath, UK

Henk Giele, MBBS, MS, FRCS, FRACS, AMRACMA Department of Plastic Reconstructive and Hand Surgery, Oxford University Hospitals, Oxford, UK

Michael J. Hayton, BSc (Honours), MBChB, FRCS (Tr&Orth), FFSEM (UK) Consultant Orthopaedic Hand Surgeon, Upper Limb Unit, Wrightington Hospital, Wigan, Lancashire, UK

N.R. Howells, MSc, FRCS (Trauma & Orth) Department of Orthopaedics, Bristol Royal Infirmary, Bristol, UK

Simon P.J. Kay, FRCS, FRCSE(hon) FRCS (Plas Surg) Department of Plastic Surgery, Leeds General Infirmary, Leeds, UK

Richard Kennedy, MB BS, FRCA Anaesthesia and Pain Management, St George's Hospital, London, UK

Simon L. Knight, FRCS Department of Plastic Surgery, Leeds General infirmary, Leeds, West Yorkshire, UK

Caroline Leclercq, MD Consultant, Institut de la Main, Paris, France

Steven Lo, MA, MSc, FRCS Plast Canniesburn Plastic Surgery Unit, Glasgow Royal Infirmary, Glasgow, UK

Carmel Martin, MB BCh Anaesthesia and Acute Pain Management, Wrightington Hospital, Wigan, Lancashire, UK

David McCombe MBBS, MD, FRACS Royal Children's Hospital, Melbourne, Australia

St. Vincent's Hospital, Fitzroy, Melbourne, VIC, Australia

Lawrence Moulton, MBChB(Hons), MRCS(Ed) ST5 Trauma and Orthopaedics, All Wales Rotation, Morriston Hospital, Morriston, Swansea, UK

University Hospital of Wales, ST6 Trauma and Orthopaedics, Heath Park, Cardiff, CF14 4XW, UK

Chye Yew Ng, MBChB (Honours), FRCS (Tr&Orth), DipSEM, BDHS EBHSDip Consultant Hand and Peripheral Nerve Surgeon, Upper Limb Unit, Wrightington Hospital, Hall Lane, Appley Bridge, Wigan, Lancashire, UK

Matthew Nixon, MD, FRCS (Orth) Consultant Hand and Peripheral Nerve Surgeon, Upper Limb Unit, Wrightington Hospital, Hall Lane, Appley Bridge, Wigan, Lancashire, UK

Mark Pickford, FRCS Plast Department of Plastic Surgery, Queen Victoria Hospital, East Grinstead, UK

S. Raja Sabapathy, MS, MCh, DNB, FRCS (Edin), MAMS Department of Plastic Surgery, Hand and Reconstructive Microsurgery and Burns, Ganga Hospital, Coimbatore, India

David J. Shewring MB BCh, FRCS(Orth), Dip Hand Surg(Eur)
Department of Trauma and Orthopaedics, University Hospital of Wales, Cardiff, UK

Ian A. Trail, MBCHB, MD, FRCS (Edin), FRCS (Lon), ECFMG
Department of Orthopaedics, Wrightington Hospital, Wigan, Lancashire, UK

Hari Venkatramani, MS, MCh, DNB Trauma Reconstructive Surgery, Ganga Hospital, Coimbatore, India

Stewart Watson, FRCS, MRCP Plastic Surgery Unit, Wythenshawe Hospital, Manchester, UK

Robert I.S. Winterton, BMedSci, MBBS, MRCS, MPhil FRCS (Plast) Department of Plastic Surgery, Wythenshawe Hospital, Manchester, UK

Blood Vessels

1

Henk Giele and Richard Barton

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Vascular injuries • hand and upper limb • ischemia

Introduction

Vascular disorders of the upper extremity encompass a broad range of pathology with diverse clinical presentations and management options. The consequences of vascular insufficiency may be critical to the point of producing cell death or subcritical events that damage tissue but fall short of generating necrosis. They occur as a result of a structural abnormality (laceration, thrombosis, embolism) or as a consequence of inappropriate physiological control mechanisms or both. Ultimately any symptoms that are a consequence of vascular disorders result from a failure to provide adequate nutritional blood flow to the extremity.

In each case a thorough understanding of the vascular anatomy and an index of suspicion borne out of the knowledge of possible diagnosis is essential to the efficient evaluation and management. In this chapter we discuss the vascular anatomy of the upper limb and the physiological control mechanisms of blood flow. The evaluation, investigation and management of vascular disorders consequent to traumatic, compressive, occlusive, vasospastic, tumour and systemic processes are each outlined separately.

An appropriate level of understanding of the incidence and nature of these anomalies will help to ensure correct interpretation of investigations and define a correct diagnosis in what can occasionally be confusing or even contradictory symptoms and signs.

The upper limb arterial system, via collaterals, anastamosing networks and physiological control mechanisms often has effective compensatory capacity in the face of vascular disorders. It is the hand, which functions as the 'end organ' that is ultimately the source of symptoms.

H. Giele, MBBS, MS, FRCS, FRACS, AMRACMA (✉)
Department of Plastic Reconstructive and Hand Surgery, Oxford University Hospitals, Oxford OX3 9DU, UK
e-mail: Henk.giele@mac.com

R. Barton, MBBS, FRACS (plastics)
Plastic and Reconstructive Surgery Unit, The Royal Melbourne Hospital, Melbourne, VIC, Australia

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Anatomy

A surgeon's understanding of the vascular anatomy of the upper limb, common anatomic variations and the typical pattern of collateral flow are essential in the assessment and management of suspected vascular injury.

Arterial System

The upper limbs are supplied by a right and left subclavian artery that becomes the axillary artery as it passes the outer edge of the first rib and enters the apex of the axilla.

Clinical Pearl – 5 Branches of the Subclavian Artery (Mnemonic VIT*amin* C&D)

V – Vertebral
I – Internal thoracic
T – Thyrocervical trunk (inferior thyroid, suprascapular, transverse cervical)
C – Costocervical trunk (first intercostal, deep cervical)
D – Dorsal scapular artery

The axillary artery extends to the inferior border of the teres major muscle where it enters the periphery and becomes the brachial artery. The axillary artery has three parts according to its relationship to the pectoralis minor (medial, deep, and inferior) and six named branches, the supreme thoracic, thoracoacromial axis, lateral thoracic artery, subscapular trunk and the anterior and posterior circumflex humeral vessels (the mnemonic is Sixties Teens Love Sex And Pot or Screw The Lawyers Save A Patient or She Tastes Like Sweet Apple Pie). Apart from the thoracic vessels these are important for collateral flow around the shoulder.

Clinical Pearl – 6 Branches of the Axillary Artery (Mnemonic)
Sixties – supreme thoracic
Teens – thoraco-acromial axis
Love – lateral thoracic
Sex – subscapular trunk
And – anterior and
Pot – posterior circumflex humeral

The brachial artery enters the flexor compartment in the medial arm and proceeds superficially in this space towards the elbow, gradually spiralling more anterior until it lies midway between the humeral epicondyles in the antecubital fossa. It bifurcates near the neck of the radius into radial and ulnar arteries. Major branches are the profunda brachii and superior and inferior ulnar collaterals. The profunda brachii branches first and follows the radial nerve to run posterior, then lateral to the humerus, and ends as anterior and posterior branches that communicate with the radial recurrent and interosseous recurrent vessels at the cubital anastamosis around the elbow joint. The superior and inferior ulnar collateral branches pass posterior and anterior to the medial epicondyle respectively to join the ulnar recurrent vessels distally. All these branches providing major sources of collateral flow across the elbow.

The radial artery appears as a direct continuation of the brachial artery. It takes a more superficial course than the ulnar artery in the proximal forearm, initially travelling deep to the bicipital aponeurosis and brachioradialis but superficial to pronator teres, flexor digitorum superficialis and flexor pollicis longus, along its path to the wrist. At the proximal wrist, it gives off the superficial palmar artery and a volar carpal branch before proceeding dorsally beneath the first extensor compartment tendons. In the snuffbox it gives rise to the dorsal carpal branch and the first dorsal metacarpal artery before diving between the two heads of the first dorsal interosseous muscle and entering the palm as the deep palmar arch.

The ulnar artery passes beneath pronator teres and the fibrous arch of flexor digitorum superficialis; it joins the ulnar nerve at the junction of the middle and proximal thirds of the forearm, on the surface of the flexor digitorum profundus muscle belly. The ulnar neurovascular bundle proceeds distally to the wrist where it lies immediately deep and radial to the flexor carpi ulnaris tendon. It gives rise to a dorsal cutaneous branch 2–5 cm proximal to the pisiform and a palmar and dorsal carpal branch at the wrist. It enters the hand by crossing superficial to the transverse carpal ligament through Guyon's canal within which it gives a deep palmar branch and continues as the superficial palmar arch.

The common interosseous artery originates from the ulnar within a few centimetres of the elbow and almost immediately divides into anterior and posterior branches. These lie deep on either side of the interosseous membrane enroute to the wrist. They communicate via perforating branches, which pierce the membrane, and then unite distally where branches connect with palmar and dorsal carpal arches, providing a collateral pathway to the hand.

Within the hand and wrist there is a system of arterial arches, which provide multiple interconnecting anastamotic networks and collateralisation. They demonstrate significant anatomic variance, particularly on the radial side of the hand. The most proximal of these arches contains volar and dorsal carpal segments that encircle the wrist. It has contributions from each of the radial, ulnar and interosseous arteries. The volar carpal arch sends branches distally into the hand to anastamose with the deep palmar arch. Dorsal metacarpal arteries two to four arise from the dorsal carpal arch and proceed distally on their respective interossei, communicating via perforating vessels with the palmar circulation at the metacarpal heads. They bifurcate into dorsal digital branches to supply adjacent sides of all four fingers.

The superficial palmar arch is a direct continuation of the ulna artery beyond the flexor retinaculum. It lies in contact with the deep surface of the palmar aponeurosis running transversely at the level of the abducted thumb. From its convexity arise digital branches- a proper digital artery to the ulna side of the little finger and three common digital arteries, to the second, third and fourth web spaces.

The deep palmar arch is a continuation of the radial artery, having entered the palm by passing between the two heads of the first dorsal interosseous muscle and onwards between the oblique and transverse heads of adductor pollicis. The deep palmar arch travels across the palm at a level proximal to the superficial arch, deep to the flexor tendons. In the classic pattern, it gives rise to the palmar blood supply of the thumb via its first branch, the first palmar metacarpal artery or arteria princeps pollicis. This passes distally along the first metacarpal bone and divides into two palmar digital branches of the thumb at the metacarpal head. The radialis indicis supplies the radial aspect of the index finger and variably arises directly from the deep arch or as a common trunk with the arteria princeps pollicis or from the superficial palmar arch. The deep palmar arch also produces three further palmar metacarpal arteries, which pass distally to anastamose with the dorsal metacarpal arteries at the level of the metacarpal heads and the common palmar digital vessels of the superficial arch. All five digits therefore receive arterial inflow from both the radial and ulna arteries via the deep and superficial arches.

The two palmar arterial arches may be incomplete. The superficial arch is most commonly completed by the superficial palmar branch of the radial artery but may also be completed via a persistent median artery or from a branch of the deep palmar arch. The deep palmar arch is less variable and is completed by the deep branch of the ulna artery in 98.5 % of hands [1].

The common digital arteries give rise to two proper digital arteries. These travel along the contiguous sides of all four fingers, dorsal to the digital nerves, between Grayson's and Cleland's ligaments. They have multiple anastamotic connections along their path. These include three transverse palmar arches located at the level of the

necks of the proximal and middle phalanges and just distal to the profundus insertion. The digital artery supplies the metacarpal and interphalangeal joints and each has two dorsal branches, which anastamose with the dorsal digital arteries.

In the thumb there are two constant communicating branches of the palmar digital arteries. The first is at the level of the proximal phalangeal neck, the second lies across the distal part of the oblique pulley of the flexor sheath. Distally, the pulp arcade runs between the insertion of the flexor tendon and the bony tuft of the distal phalanx. Similar to the fingers there are further branches from the digital arteries to the interphalangeal joint, dorsal thumb, nail bed and flexor sheath.

Some arterial anatomical variations have already been discussed; certainly the point has been made about the high incidence of variability on the radial side of the hand. The dominant supply to the thumb, being the ulnar palmar digital vessel will only arise from the first palmar metacarpal artery approximately 60 % of the time, it is otherwise supplied from the first dorsal metacarpal artery, superficial palmar arch or superficial branch of the radial artery. Despite this wide variety of origin, once it has reached the level of the ulnar sesamoid, the ulnar palmar digital artery will follow a constant and superficial course in all thumbs [2].

The most common abnormality above the wrist is a high branching radial artery from the brachial artery. When this occurs it is more likely to be from a high proximal position than from the lower part of the brachial artery, it is common, occurring in approximately 12 % of arms [1]. Peculiarities of the radial artery in the forearm are uncommon, but typically relate to a more superficial position of the vessel, such as lying on the surface of brachioradialis, instead of under its medial border and lying above the first and or third extensor compartments at the wrist. Less commonly the ulnar artery may also vary in its origin, occasionally arising 5–7 cm below the elbow, but more frequently from higher on the brachial artery. With a proximal origin the artery will typically lie in a more superficial position over the flexor muscles in the forearm.

A persistent median artery has a reported incidence between 4.4 and 27 % [3]. During embryogenesis the median artery branches from the interosseous (axis) artery and follows the median nerve in the forearm and into the hand. It provides the dominant blood supply to the distal half of the upper extremity in the first few months of foetal life. In the normal course of development the median artery regresses and usually disappears as the radial and ulnar arteries develop. When present after embryogenesis the persistent median artery accompanies the median nerve through the carpal tunnel on its ventral surface, where it may join the superficial palmar arch or end as one or two palmar digital arteries.

Venous System

The venous system is defined by superficial and deep veins linked by perforating vessels. Valves in each of these systems prevent retrograde flow and the flow of blood from deep to superficial. Deep veins are numerous and accompany arteries in the form of venae commitantes and also lie within muscle bellies. Large superficial veins on the back of the hand form the dorsal venous network. This network contributes significantly to the venous drainage of the palm and this then coalesces on the radial side into the cephalic vein and on the ulnar side into the basilic vein. These two vessels serve as the dominant superficial drainage routes along the lateral and medial aspects of the upper limb.

The cephalic vein gives rise to the median cubital vein distal to the elbow, which receives branches from the deep system and diverges proximo-medially to reach the basilic vein. Above the elbow the cephalic vein runs lateral to biceps, along the deltopectoral groove and perforates the clavipectoral fascia to drain into the axillary vein. The basilic vein runs up the medial border of the limb, perforating the deep fascia halfway up the upper arm and joins the brachial veins to become the axillary vein. The standard pattern of superficial veins in the forearm also includes a median vein that drains the flexor surface of wrist and forearm and joins either the basilic or median cubital vein. There are frequent variations to this pattern [4].

Lymphatic System

The lymphatic glands and vessels of the upper extremity are divided into superficial and deep [5].

The scant superficial glands comprise the supratrochlear and deltopectoral groups; they number only a few in each group. The supratrochlear group are situated above the medial epicondyle of the humerus, medial to the basilic vein. The deltopectoral glands lie adjacent to the cephalic vein in the deltopectoral groove, just inferior to the clavicle. The superficial lymphatic vessels accompany the cephalic, median and basilic veins. This system is in free communication with the deep lymphatics whose glands lie predominantly in the axilla. There may be some scattered deep glands along the course of the arteries in the forearm and the brachial artery in the arm. The axillary glands typically number 20–30 and in surgical terms are described as being in levels one to three. Level I glands lie distal to pectoralis minor, level II glands lie deep to the pectoralis minor and level III glands are in the apex of the axilla, proximal to pectoralis minor [6].

Physiology

The microvascular system of the hand functions to deliver the nutritional requirements of the tissue and to provide flow through the arteriovenous anastamosis that participate in temperature regulation. The nutritional flow required to maintain tissue viability is typically only 10–20 % of the potential blood flow, leaving the remainder to pass through the thermoregulatory beds. This system has considerable capacity and undergoes large fluctuations in volume, under the control of environmental influences, local factors and metabolic demands as well as circulating mediators and centrally controlled sympathetic tone.

Local metabolic demands, mediated through oxygen levels and metabolites, influences microcirculatory blood flow to maintain adequate nutritional requirements.

Endothelial cells are intimately involved in the regulation of vascular tone via the synthesis and release of cytokines, growth factors, prostaglandins and other bioactive macromolecules. Some are mediators of vasodilatation such as prostacyclin and nitric oxide and others, such as endothelin-1 are vasoconstrictors.

The sympathetic nervous system contributes to vaso-regulation via the alpha adrenergic receptors of the vascular smooth muscle which cause vasoconstriction. The nerve fibers travel in perivascular tissue and penetrate the arterial and venous walls of the hand and forearm.

Evaluation of Vascular Disorders of the Upper Limb

History and Presentation

The clinical presentation of upper limb vascular disorders range from significant ischaemic symptoms such as pain, finger tip ulceration or gangrene, to mild symptoms suggestive of inadequate, subcritical blood flow; claudication, peripheral cold intolerance, altered sensation and skin colour changes.

Patients present with symptoms of acute onset or having developed signs and symptoms progressively over time. Acute necrosis or open wounds are relatively easy to assess, but when the complaints are chronic, and mild or intermittent then diagnosis is more difficult and reliant on investigations.

The patient may reveal a history of recent trauma or describe chronic occupational or recreational exposure to repetitive hand injury and vibration. If the condition is non-traumatic a broad past medical history must include the search for atheromatous disease, cardiac ischaemia and arrhythmia, malignancy, diabetes, systemic connective tissue disorders, drug exposure, tobacco use and family history of blood dyscrasias. An element of peripheral vascular disease may be present prior to the injury or indeed render the vessel more susceptible or less tolerant of injury. In chronic or delayed presentations, one should enquire about aggravating and relieving factors, such as activity, arm position and environmental or emotional stressors.

Examination

Examination includes the entire upper limb and neck and aims to determine the adequacy of the vascular system and identify sites of possible vascular compromise. Inspection can reveal open

wounds, joint dislocation, deformity, but also more subtle observations of skin colour change, hair loss, scars, fingertip atrophy, necrosis or ulceration, splinter haemorrhages and fungal infections of the nails.

On palpation one should detect temperature differences, skin texture, hair growth, capillary refill and the quality of pulses. The site of previous injury may reveal the mass of an aneurysm, fistula or haematoma.

There are a number of useful clinical tests that should be carried out during the consultation. Allen's test is used to determine the patency of the dual blood supply and quality of collateral circulation of the hand or digit [7]. When assessing the ulnar and radial arteries the examiner compresses both vessels at the wrist and asks the patient to open and close the hand until it turns pale. The vessels are then released sequentially and reperfusion across the hand is observed. The test is repeated reversing the order of artery release. Delayed perfusion or failure to reperfuse indicates reduced flow in the vessel released.

If the pulses cannot be palpated they may be searched for using the hand held Doppler probe. However presence of the Doppler signal should not reassure one to the extent of avoiding intervention, as a completely occluded artery can still display an audible Doppler signal.

Investigation

The appropriate set of investigations is determined by the clinical presentation, history and examination findings. An open wound, fracture or dislocated joint with loss of pulses and compromised distal vascularity requires no further investigation, other than surgical exploration and reduction.

However in chronic cases, investigations may be required and along with specific upper limb vascular investigations it may be relevant to perform blood tests such as ESR, Rheumatoid factor and antinuclear antibodies. Other considerations, particularly if embolisation is suspected, include ECG and cardiac echocardiogram. A number of patients will benefit from a referral to other disciplines such as rheumatology, cardiology or vascular surgery.

Vascular testing aims to determine the structural configuration of the upper limb vessels and their functional capability to respond to stress. Often a combination of vascular studies is necessary to help differentiate between occlusion and vasospastic disorders and determine their relative importance when both occur together.

In most circumstances plain radiography is the starting point. It is useful in characterising phleboliths, vascular calcification, foreign bodies, and the presence of any osseous abnormalities. Vascular imaging using ultrasound, CT and MRI has greatly increased the diagnostic ability of radiologists [8].

Doppler ultrasound is easily accessible and inexpensive. It is able to differentiate venous from arterial flow, assess flow haemodynamics and vessel lumen morphology. A normal vessel produces a triphasic waveform and progresses to monophasic in a vessel with abnormal flow characteristics. Pulse-echo imaging uses sound to produce a two dimensional representation of the vessel wall. It is, however, operator dependent and is unable to fully evaluate upper limb arterial inflow.

Colour duplex imaging can provide structural and functional information about a vessel. It demonstrates the direction and velocity of flow with varying intensity of either a red or blue colour on the monitor. This non-invasive technique is cost efficient and repeatable. It is useful to differentiate between tumours of the upper extremity such as differentiating ganglia from aneurysms. It can also localise the site of vascular obstruction.

Plethysmography, or digital pulse volume recording, is a technique that quantitates flow by detecting volume change in the limb or digit and can measure the response in blood flow to changes in temperature. It produces characteristic pulse volume recordings that can be used to differentiate a fixed arterial obstruction or narrowing from vasospastic disease. It is further helpful in the evaluation of vasospastic disease by predicting the results of surgical sympathectomy by observing the response of a cold, vasoconstricted digit blocked with local anaesthetic. The anaesthetised digit mimics the physiological conditions achieved following sympathectomy, so an improvement in the signs suffered under environmental stress is a positive predictor of operative success.

Cold stress testing provides an evaluation of the response of the digital vessels to physiologic stress by monitoring cutaneous perfusion and

temperature with exposure to cold. It is most commonly used as an investigation for patients with suspected Raynaud's phenomenon [9].

Magnetic resonance imaging/magnetic resonance angiography (MRI/MRA), computed tomographic angiography (CTA) or intra-vascular contrast angiography can be obtained. Intra-vascular contrast angiography remains the gold standard for evaluation of static structural detail, providing the required information to plan distal hand and digital revascularisation procedures. However, this modality is invasive, requires iodinated contrast agents, exposure to radiation and may induce further vasospasm on top of already compromised vessels. It can also fail to detect significant extra luminal disease without the aid of supplementary investigations.

MRI/MRA does not include ionising radiation or the potential allergic reaction from contrast media, it does not induce vasospasm and has no renal side effects. It is able to provide functional information in the form of velocity, volume and directional data but is limited by the presence of metallic and implantable devises and, compared with CTA and intra-vascular contrast angiography, provides lower spatial resolution and less reliable vessel wall characterisation, which are essential for small vessel imaging of the hand.

CTA does expose the patient to radiation and requires intravenous dye, but unlike intra-vascular angiography, avoids intra-arterial catheterisation. It is a relatively short procedure with wide availability. It offers good vessel wall characterisation with enhanced spatial resolution compared to MRA, and has the advantage of providing comprehensive evaluation of nonvascular structures. Often any decision about the best modality for a specific patient is reached following discussion with a radiologist and is heavily dependent on local expertise and availability [8].

Causes of Injury to Blood Vessels

Traumatic Injury

Trauma causes around half of all vascular disorders of the upper limb. Vascular trauma can be penetrating (open) or blunt (closed). An acute vascular injury from penetrating trauma is usually easily diagnosed and beyond re-establishing adequate blood flow, the complex issues of managing concomitant bony, nerve and soft tissue injury will often determine the clinical outcome. Blunt trauma should arouse suspicion of laceration or obstruction of a significant vessel, and acute ischaemia or subcritical vascular compromise should be looked for, promptly recognised and appropriately managed.

The initial management of a trauma patient is directed by the mechanism and clinical significance of their injuries. Some patients suffering multi-trauma will require stabilisation of their airway, breathing and circulatory control, prior to any formal assessment of the upper limb injury.

Penetrating Trauma

Most patients with penetrating trauma of their upper limb will have an isolated injury. Control of haemorrhage is achieved with direct pressure at the bleeding point and arm elevation. Almost all types of bleeding in the periphery, including a significant partial injury of the brachial artery (often more difficult to control than a complete transection because of the inability of vasoconstriction and thrombus formation to stop flow) can be controlled in this manner.

Prolonged tourniquet application is not justified and serves only to deny the distal limb the benefits of any patent collateral circulation. Another common mistake is misdirected and overly bulky dressings, which rarely prove to be adequate. In this circumstance, under brief tourniquet control, removal of all the dressings and an accurate determination of the bleeding point and subsequent application of direct pressure will control the haemorrhage. It is not appropriate to try and apply potentially damaging surgical instruments, such as artery clamps, blindly into a blood filled wound in an attempt to control blood loss. The risk of collateral damage to nerves is very high as is the likelihood of causing further damage to vessels complicating any reconstruction.

An open injury of the upper limb resulting in arterial damage to the brachial artery or distal vessel will rarely require urgent surgical intervention to control blood loss, the urgent indication is for restoration of blood supply to a

relatively ischaemic limb. The situation is very different for a more proximal vessel injury of axillary or subclavian artery, which can become rapidly life threatening, given their deeper anatomical location and the larger volume of blood loss, and it is much more challenging to achieve control without emergency surgery [10].

Once bleeding is controlled there will be time for a reliable assessment of the limb, particularly the extent of soft tissue injury, nerve and bony damage. The vascular integrity of the limb can only be reliably judged by visual examination of the vessel in the zone of trauma under anaesthesia in the operating theatre. Perfusion of the limb can, however, be assessed clinically by skin colour, temperature, capillary refill and palpation of pulses. The presence of capillary return and pink fingertips is not an indicator of adequate blood supply, merely an indicator of sufficient collateral blood flow for the resting limb, which if ignored can lead to progressive necrosis, or relative ischaemia with activity. The presence of a distal pulse by palpation or Doppler, is not a reliable sign of an intact proximal vessel. The pulse may be the result of retrograde flow through collateral circulation or wave transmission through an injured segment. Other than a plain radiograph, further investigations including angiography are rarely helpful, but can be critically time wasteful [11, 12].

It is obvious that a critical arterial injury in an otherwise salvageable limb requires repair, but we would also advocate repair of isolated non-critical arterial injuries such as a brachial artery injury distal to the origin of profunda brachii or a radial or ulnar artery injury. The aim should be to avoid the development of relative ischaemia in the future, and to decrease the symptoms of inadequate perfusion such as cold intolerance and claudication. Vessel repair will also aid bone and soft tissue repair and nerve recovery. The loss of one of the major vessels supplying the hand places the patient at high risk in subsequent arterial occlusion or vessel injury at a different location [13]. However, many reports on repair of isolated radial or ulnar artery injuries indicate only 50 % patency rates at follow up with no sequelae associated with occlusion of the repaired vessel or from ligating the vessel instead. However, other studies show that 50 % of patients complain of hand weakness, 25 % of paraesthesia and 15 % of cold insensitivity following ligation or occlusion of the traumatised radial or ulnar artery at the wrist [14]. Following radial artery harvest for coronary artery bypass grafting 10 % of patients suffer hand paraesthesia or numbness [15].

Blunt Trauma

Closed arterial injury from blunt trauma is usually associated with joint dislocation or humeral fracture. Shoulder, elbow and scapulothoracic dislocations can cause traction injuries and lead to arterial laceration and avulsion or more insidiously, intimal tears and the risk of subsequent thrombotic occlusion. An anterior dislocation of the glenohumeral joint for example, can result in an axillary artery injury [16]. Blunt subclavian artery trauma is thankfully uncommon but presents a challenging surgical problem. It can result from high-energy trauma that causes fractures of the clavicle or scapula or scapulothoracic dissociation and can produce a life threatening proximal vessel injury [17]. The patient presents with periclavicular haematoma, significant shoulder swelling and neurological deficits as a result of the inevitable brachial plexus injury. Prompt angiographic confirmation of the site of injury and surgical intervention with vessel repair is required. Older patients with atherosclerotic vessels are at risk of arterial injury with a significantly displaced humeral fracture. It is usually a proximal fracture dislocation of the humerus that results in axillary artery injury through the avulsion of circumflex scapular or subscapular arteries or direct axillary artery trauma.

Supracondylar humeral fractures tend to involve young patients and can cause vascular compromise through brachial artery displacement and kinking or direct vessel injury from bony fragments such as entrapment or penetrating lacerations. Median nerve damage (especially anterior interosseous components) are more common than vessel injury [18]. Clinical signs of nerve injury should be sought in all supracondylar fractures especially in cases where vessel

damage has occurred. Closed humeral fracture reduction and pin fixation will often resolve the acute ischaemic symptoms, with blood flow across the elbow re-established via collaterals or a recovered brachial artery. In the persistently ischaemic hand post fracture reduction, prompt recognition and surgical intervention for repair of the arterial injury is required [19], without the delay of further angiographic investigation [20]. If the brachial artery is damaged it will occur at the site of the fracture. The usual mechanism of injury is entrapment within fracture fragments; it may also suffer a laceration through direct injury from bony fragments or occlusive thrombosis subsequent to intimal damage from compression and kinking. Exploration of the vessel will allow a decision to be made about the extent of injury and a suitable method of repair.

A perfused but pulseless hand post supracondylar fracture reduction and fixation, creates a dilemma for the surgeon. The appropriate management of such, particularly indications for surgical exploration, continues to be a source of controversy. Common dogma suggests that the pink, pulseless hand is a benign condition and usually advocates a watchful waiting approach to these injuries [19]. However, we have a very low threshold for surgical exploration of the brachial artery given the high incidence of arterial injury and the severe consequences of persistent vascular compromise (even if only relative), the high risk of associated nerve injury and the relatively low morbidity of microvascular surgery. Certainly, in a limb with worsening pain and deteriorating neurological signs or persistent absence of a radial pulse at 24 h post fracture reduction, surgical exploration is indicated. In a literature review of 331 cases of pulseless supracondylar fractures, 157 remained pulseless after reduction of which 98 extremities were pulseless yet pink following fracture reduction and fixation, and of which on exploration 70 % had a brachial artery injury [21]. Mechanisms of arterial injury found at exploration were; traumatic aneurysm with thrombus formation, complete laceration and partial tear and arterial entrapment at the fracture site. These findings suggest that even though a pink pulseless hand may survive and have no obvious sequelae on superficial examination, the majority are surviving on collaterals with a level of vascularity that must have a bearing on normal physiology and function at extremes of demand.

Surgical Treatment

In penetrating trauma with a clean, sharp, single level injury end-to-end repair will often be achievable. This can be aided by conservative vessel mobilisation, but should not be attempted under undue tension or without adequate debridement of damaged vessel ends. An interposition graft is always a better option than a less than optimal primary repair. Partial lacerations can usually be directly repaired, or with the aid of a vein patch.

In blunt injuries, the vessel may rupture completely, tear (usually at the origin of tethering branches), or suffer an intimal injury maintaining vessel continuity.

The mechanism of injury in blunt trauma or open avulsion injuries always involves some element of longitudinal stretch. This causes more extensive vascular injury than is evident on visual or microscopic examination, making it difficult to judge the length of vessel to resect. In this situation end to end repair will only aggravate longitudinal tension and increase the risk of thrombosis. Vessel injury can be estimated by close observation for linear red streaking indicating separation of the intima. After resecting the estimated length of damaged vessel and demonstrating adequate proximal blood flow, a suitable graft can be harvested. Vein grafts can be harvested from the adjacent large superficial cephalic or basilic veins or their branches. Despite these veins being in the zone of injury their subcutaneous position, separate from the brachial artery, means they often do not bear the same brunt of injury. The vein grafts should be reversed and kept at an appropriate length and tension to avoid kinking particularly with elbow flexion.

Due consideration should be given to the long-term survival of these grafts and the potential of other more suitable donors. The basilic and cephalic veins are both thin walled and theoretically run the risk of developing late vein graft

occlusion from intimal hyperplasia or dilatation and deterioration. The cardiothoracic literature is replete with reports demonstrating improved patency of arterial grafts over vein grafts. Arterial grafts from the thoracodorsal pedicle, deep inferior epigastric artery, descending branch of the lateral circumflex femoral or a vein that best approximates an artery with respect to the thickness of the wall, such as those in the distal lower leg, should be considered. Despite these theoretical concerns the 5 year follow up of lower limb autogenous vein grafts used in 134 injured extremity arteries showed 98 % patency [22].

The use of intra- or post-operative systemic anticoagulation such as unfractionated heparin or dextran is unnecessary in our experience. Close postoperative clinical monitoring of distal perfusion, and pulse, with or without the aid of a hand held Doppler probe or digital pulse oximeter is adequate to confirm the persistence of a patent anastomosis.

Upon achieving good intra-operative flow through the repaired vessel, a prophylactic fasciotomy should be performed in any limb that has suffered prolonged ischaemia, over 3 h, and where reperfusion injury is likely.

Compressive Injury to Blood Vessels

Compartment syndrome is a condition in which the tissue perfusion in an anatomical compartment is compromised by the increase of interstitial tissue pressure within the compartment. In the upper extremity it is most common in the forearm. The intrinsic muscles of the hand may also be involved and rarely the muscles of the upper arm.

There are four compartments in the forearm: superficial palmar, deep palmar, dorsal and a dorsal/proximal compartment containing the mobile wad of brachioradialis and extensor carpi radialis longus and extensor carpi radialis brevis. In the hand each interosseous muscle is its own compartment in addition to the adductor pollicis muscle and the thenar and hypothenar muscles, though some believe these compartments are incompetent at low pressures and hence not true compartments [23].

Compartment syndrome can result from a variety of causes including crush injuries, fractures, haematomas, extravasation injuries, burns and external compression. Basically, anything that causes a decrease in compartment volume, restriction in compartment expansion (such as caused by burns eschar) or an increase in compartment content. Beyond traumatic injury, increased compartment content can be the result of nephrotic syndrome, venous obstruction, infection and exercise. Compartmental compression induced by exercise is some times referred to as recurrent or chronic exertional compartment syndrome. It is much more common in the lower extremity, and is typically transient, resolving with rest.

The symptoms of compartment syndrome are a consequence of the pathologically elevated interstitial tissue pressure within the fixed space compartment, which prevents capillary blood perfusion such that it cannot maintain tissue viability. The end result is muscle and neural ischaemia, necrosis and fibrosis, leading to Volkmann's ischaemic contracture.

Rowland described the relationship between local blood flow (LBF) and the arteriovenous gradient by the following equation:

$$\text{LBF} = \left(\text{Pa} - \text{Pv}\right) / \text{local vascular resistance}$$

The local blood flow in a compartment equals the local arterial pressure (Pa) minus the local venous pressure (Pv) divided by the local vascular resistance [24]. As veins are compressible, the pressure inside them cannot be less than the local tissue pressure; therefore, when the interstitial tissue pressure rises, so does the local venous pressure, which results in a decrease in the arteriovenous gradient and a decrease in the local blood flow. This is combined with complex events at a cellular level involving an accumulation of toxic chemicals in the extracellular environment, increases capillary leakage further contributing to the rise of interstitial tissue pressure. With normal capillary perfusion pressure around 25 mmHg and interstitial pressure around 5 mmHg it does not take much of an increase in interstitial pressure to affect capillary perfusion. If interstitial pressure rises to 30 mmHg, the

patient will suffer pain with activity. At 40 mmHg, pain with passive stretch occurs, and at 50 mmHg severe pain with paraesthesia is experienced. At 60 mmHg ischaemia starts but one does not lose pulses and distal capillary return until the interstitial pressure rises above systolic.

These figures are relative to the patient's pressure, so at interstitial pressures 20 mmHg below systolic blood pressure blood flow reduces and pO2 reduces, whereas at interstitial pressures 10 mmHg below systolic pressures, blood flow stops completely and pH and pO2 drop. In injured muscle such as following crush injuries, the effect occurs at least 10 mmHg lower!

Compartment Syndrome Pearls

Classic Symptoms and Signs of the 5 P's are too late!

Pallor
Pain
Paraesthesia
Paralysis
Pulselessnes

Minor increases in interstitial compartment pressures can have significant detrimental effects.

Symptoms	Interstitial Pressure (maximum normal 25 mmHg)
Pain with activity	−5 mmHg over normal
Pain with passive stretch	−15 mmHg over normal
Pain with paraesthesia	−25 mmHg over normal
Irreversible ischaemia	−35 mmHg over normal
Loss of pulse/ pallor	−120 mmHg (over systolic pressure)

Reduce these pressures by 10 mmHg where muscle injury is already present

Early recognition and diagnosis is essential. In an awake patient without a significant proximal nerve injury, pain is the most important and consistent symptom. The pain is persistent and increasing, it is not relieved with elevation or immobilisation and is exacerbated by muscle stretch with passive extension of the fingers. Sensory nerve fibres are the most susceptible tissues to hypoxia, and as a consequence diminished fingertip sensibility is often a clinical sign. Distal arterial pulses are palpable well after the onset of ischaemic neural and muscle damage, which occurs at tissue pressures below arterial systolic pressure. The loss of pulses and pallor are late signs, with irreversible damage already having occurred.

In patients who are heavily intoxicated, have suffered a head injury or are intubated and ventilated, clinical signs are likely to be limited. In this scenario inter-compartmental pressure monitoring may be necessary. Fasciotomy is recommended when compartment syndrome is suspected or when the compartmental pressures rise above 30–45 mmHg or are showing a rising trend.

As the consequence of a delayed or missed diagnosis is so significant, any patient with a high degree of clinical suspicion for compartment syndrome should undergo emergency fasciotomy. The volar forearm compartments are released through a long curvilinear or straight skin incision that starts at the elbow flexion crease, just radial to the medial epicondyle. It then descends along the ulnar side of the forearm along the radial border of flexor carpi ulnaris to the wrist crease [25]. The incision continues parallel to the wrist crease and ends as a standard open carpal tunnel release. Through this incision decompression of the superficial compartment is performed, and by retraction of the ulnar neuromuscular bundle and flexor digitorum superficialis radially in the middle and distal third of the forearm, the deep flexor compartments of pronator quadratus, flexor pollicis longus and flexor digitorum proudness are easily accessed and decompressed. Finally, release of the transverse carpal ligament is performed. An assessment of the muscle viability can be made and an adequate debridement of any devitalised tissue carried out if necessary.

The dorsal forearm compartment can be released by an incision beginning distal to the lateral epicondyle, between the extensor digitorum communis and extensor carpi radialis brevis, and

extending proximally about two thirds of the way to the wrist. The fascia is released over the dorsal muscles and proximal mobile wad.

In the hand, two longitudinal incisions are made over the second and fourth metacarpals to access both the dorsal and volar interosseous compartments and the adductor pollicis muscle. Separate palmar-radial and ulnar incisions are made to release the thenar and hypothenar compartments. In the acute preventative fasciotomy there is no indication for epineurolysis of the major nerves. This procedure may be of benefit in established compartment syndrome or Volkmann's ischaemic contracture. Decompression of the fingers is carried out via a mid lateral incision, and is only indicated in external compression of the digits as occurs in burns eschar (Figs. 1.1, 1.2 and 1.3).

The fasciotomy wounds should be dressed until the compartment swelling has reduced, following which staged primary skin closure is performed or more commonly the wound is partly closed and mainly split skin grafted or allowed to heal by secondary intention [26].

Acute Occlusive Conditions Affecting Vessels

Acute arterial occlusive disease of the upper extremity results from thrombosis, embolisation and aneurysmal formation. It is rare, and its most common form is iatrogenic following arterial cannulation for monitoring or angiography [27]. Its most common pathological form is due to post-traumatic thrombotic occlusion of the ulnar artery in the hand, known as hypothenar hammer syndrome [28]. Embolisation as a source of occlusion should be considered when

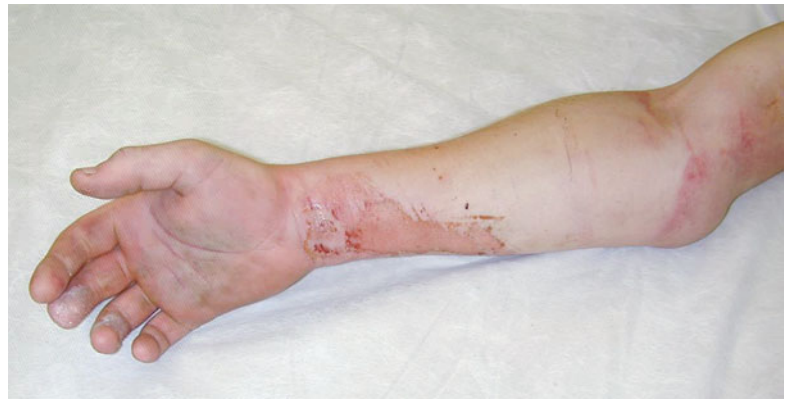

Fig. 1.1 Swollen forearm after crush injury

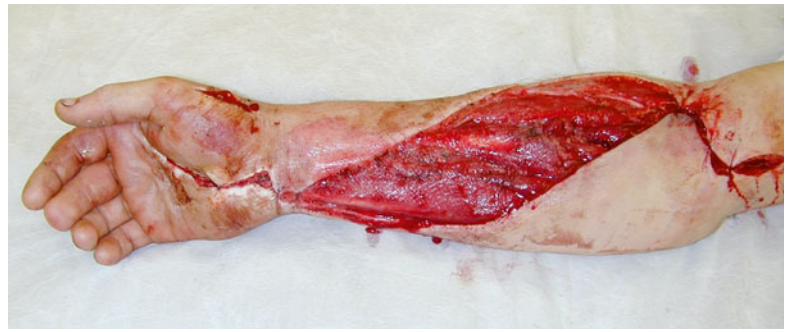

Fig. 1.2 Forearm and palmar fasciotomies

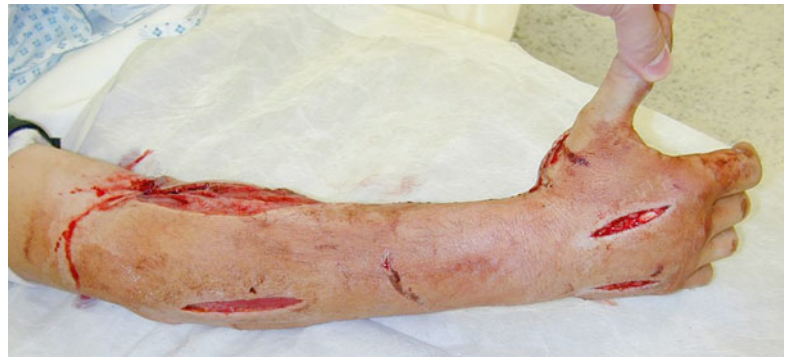

Fig. 1.3 Dorsal fasciotomies

Table 1.1 Rutherford classification of acute lower extremity ischemia [29]

	Viable	Threatened	Non-viable
Sensory deficit	None	Partial	Complete
Arterial Doppler	Audible	Inaudible	Inaudible
Motor deficit	None	Partial	Complete
Pain	Mild	Severe	Variable
Capillary refill	Intact	Delayed	Absent
Venous Doppler	Audible	Audible	Inaudible
Treatment	Urgent workup	Emergency surgery	Amputation

the ischaemia is of sudden onset and is associated with atrial fibrillation or follows a myocardial infarction. Distal micro-emboli however rarely travel all the way from the heart and typically result from thrombotic or aneurysmal disease in upper limb vessels.

Acute limb ischaemia can be classified and categorised according to Rutherford's classification, and this can aid reporting and guide treatment (Table 1.1).

Upper extremity venous occlusive disease most commonly involves the deep system and occurs in association with a hypercoaguable state, venous endothelial injury or arises in otherwise healthy patients because of venous impingement in the thoracic outlet. Patients present with swelling of the arm, pain and skin discolouration. Superficial veins may be dilated.

Paget-Schroetter disease describes upper extremity deep vein thrombosis, typically in the large proximal vessels that occurs spontaneously or in association with thoracic outlet syndrome.

Thrombosis

Ulnar artery thrombosis in the palm in the form of 'hypothenar hammer syndrome' is the most common type of non-idiopathic upper limb arterial occlusion, despite being rare. Repetitive trauma from using the palm of the hand as a 'hammer' causes disruption of the internal elastic lamina of the ulnar artery, with arterial media fibrosis, as it exits from Guyon's canal. This results in aneurysmal dilatation with mural thrombi, leading to ulnar artery thrombotic occlusion and distal embolic events. Symptoms result from microemboli and inadequate collateral circulation to the ulnar digits and are exacerbated by secondary vasospasm [28].

The condition occurs most frequently in male, manual workers who smoke and use vibratory tools. These patients present with vascular compromise to the little, ring and middle fingers, resulting in cold sensitivity, pain, numbness, tingling and ulceration, along with fingertip colour and temperature changes. The severity

of symptoms depends, in part, on the extent to which the radial artery supplies the ulnar side of the hand. Patients also experience downstream embolisation, causing intermittent episodes of digital ischaemia.

An Allen's test will confirm absence of flow through the ulnar artery, with an associated tenderness or palpable mass in the ulnar side of the proximal palm. Angiography or MRA provides detail of the segment of vessel involved and will allow accurate management planning. An early sign of ulnar artery thrombosis seen on angiography provides a 'string of beads' appearance and indicates alternating fibrosis and dilatation but must not be over-interpreted as this appearance may arise from corkscrew dilatation of the post-traumatic or aged ulnar artery [28].

The goal of treatment is the restoration of adequate blood flow and should include conservative measures such as cessation of smoking and avoidance of cold. There can be an indication for thrombolytic therapy in the early stages [30] or vasodilatory interventions, such as cervicothoracic sympathectomy, stellate ganglion blocks and oral sympatholytics, if the symptoms are primarily the result of abnormal physiologic control rather than the underlying structural damage.

Surgical intervention is indicated in acute onset thrombosis or in the presence of inadequate collateral circulation and persistent distal ischaemia or recurrent distal embolisation. It involves resection and reconstruction of the involved vessel with an appropriate graft. The ulnar artery is explored proximal to the wrist and followed distally to the occluded segment and beyond to the superficial palmar arch and its 3 common digital arteries. After assessment of the extent of vascular damage a bypass conduit can be planned using the saphenous vein and its branches from the dorsum of the foot.

The radial artery may also suffer from occlusive thrombosis and be the source of embolisation. It can occur in the anatomical snuffbox and may result from compression from the first dorsal extensor compartment tendons. Far more commonly, brachial or radial artery thrombosis is the result of an iatrogenic injury following cannulation for arterial pressure monitoring or angiography. Temporary thrombosis can occur in 40 % of cannulations but rarely produces major ischaemic problems [31]. Pallor, and occasionally paraesthesia and pain of the index finger and thumb, should prompt rapid removal of the cannula. Surgical resection and reconstruction of the involved segment of vessel yields good symptomatic relief [27].

Embolism

Emboli to the upper limb account for 15–20 % of all peripheral emboli and mostly originate from the heart (70 % of cases) secondary to conditions such as cardiac arrhythmias, ventricular aneurysms, myocardial infarction and bacterial endocarditis. The remaining 30 % result from upper limb vascular abnormalities such as subclavian artery aneurysm secondary to thoracic outlet compression and radial and ulnar artery thrombosis mainly secondary to cannulation [32].

Emboli of cardiac origin tend to be larger and will most commonly occlude the brachial artery bifurcation. Distal emboli to the hand and wrist is usually the result of micro embolic showers, originating from the subclavian artery or peripheral vessel. The defining clinical feature is one of acute onset ischaemia. The limb distal to the embolic occlusion will be painful, cool, pale and pulseless. Evaluation should include a targeted history and examination with complementary investigations including echocardiography and upper limb angiography as indicated. Angiography can be helpful to determine the site of a proximal source of emboli and to differentiate embolism from acute arterial thrombosis [33].

For brachial artery macro emboli, anticoagulation with therapeutic heparin infusion and embolectomy with a Fogarty catheter is indicated. Embolic events in the hand may require anticoagulation and thrombolysis or surgical management of embolic sources with vessel resection and reconstruction [30]. Thrombolysis will not be effective on athero-emboli.

Intra arterial injection injuries, from illicit drug use, occupational injuries involving high pressure solvents and paint products or inadvertent injection of medical therapy, causes arterial occlusion from chemical endarteritis, secondary vasospasm and particulate embolisation. Patients

present with a painful, mottled and cool periphery. The management is aided by colour Doppler evaluation and angiography to define the arterial damage and extent of distal occlusions. Typically, multiple distal embolic events preclude arterial reconstruction and systemic or intra-arterial therapy is the only option. The medical management lacks any universally agreed protocol. Many combinations of vasodilator, thrombolytic therapy, steroids and anticoagulant have been tried. Heparinisation should be initiated to prevent the propagation of thrombus and further therapy considered depending on the relative influence of vasospasm and established thrombosis [34–36].

Aneurysm

Aneurysms comprise the majority of acquired vascular tumours of the upper extremity and typically result from repetitive trauma. The most common site is the ulnar artery, often where it abuts the hook of the hamate [37]. Alternatively, aneurysms can occur in the superficial palmar branch of the radial artery between the abductor pollicis brevis and opponens pollicis, or the subclavian artery, where aneurysms occur as a result of thoracic artery compression (related to thoracic outlet syndrome and cervical ribs) and post stenotic dilatation [38]. At any point on the limb partial vessel injury from penetrating trauma or cannulation can result in the formation of a pseudoaneurysm. Unlike true aneurysms, which involve all three layers of the arterial wall, pseudoaneurysms are haematomas adjacent to the injured artery that become cannulated by blood flow.

Aneurysms usually present as a result of symptoms from distal embolic events, or from the palpable mass. Presentation is only rarely as vascular insufficiency from obstructive thrombosis. On examination aneurysms present as a painless, palpable mass located along the line of a vessel. They often exhibit a thrill or bruit and the diagnosis is confirmed with the aid of colour duplex imaging or angiography.

In the majority of cases, the potential of complications such as rupture and thromboembolism, warrants surgical management by resection and reconstruction of the involved vessel. The artery proximal and distal to the aneurysm should be explored and segmental resection performed with vessel reconstruction.

Iatrogenic

Iatrogenic arterial injuries can occur from cannulation or inadvertent arterial drug injections. Many patients require arterial blood studies or indwelling arterial catheters for blood pressure monitoring and the radial artery at the wrist is the most common target in adults. Repeated vessel injury risks thrombotic occlusion or pseudoaneurysm formation with the possibility of distal embolisation. The brachial artery can also be the site of iatrogenic thrombotic occlusion as it is often catheterised for coronary angiography. This causes local thrombosis in up to 10 % of patients and necessitates percutaneous intra-vascular intervention or acute surgical repair in a little less than 1 % of procedures [39].

Prior to radial artery cannulation an assessment of the hand collateral circulation with an Allen's test is recommended. It is thought that noncritical occlusion occurs in around 25–40 % of cannulated arteries, although most of these will undergo recanalisation over time [31]. In the event of symptomatic occlusion, surgical intervention is often required in the form of exploration and reconstruction of the damaged vessel segment. In adults this is preferred to thrombolytic therapy or simple thrombectomy.

A difficult population at risk of iatrogenic thrombotic arterial occlusion are neonatal and paediatric patients. The survival rates for premature and sick infants has significantly improved and so the incidence of vascular injury from arterial catheterisation has increased. The consequences of arterial thrombosis range from limb or tissue loss, Volkmann's contracture, neural damage, or long-term growth disturbance secondary to ischaemic growth plate insult. These patients are placed at a high risk of thrombotic occlusion due to reduced endothelial tolerance to injury, small vessel diameter, increased tendency to vasospasm and a diminished fibrinolytic system [40].

Due to vessel size, the brachial artery is often the site of catheterisation, this places the limb at

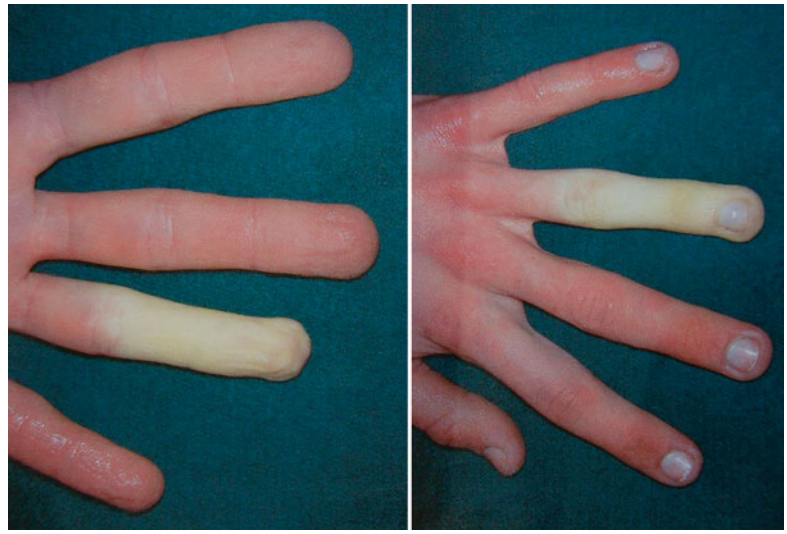

Fig. 1.4 Raynaud's phenomenon

particular risk as it functions as an end artery to the hand. Catheterisation of the radial artery is much preferred as it preserves collateral flow in the event of thrombosis. In the event of symptomatic occlusion all patients should be aggressively anticoagulated with intravenous heparin infusion, in around 50 % of patients further intervention in the form of thrombolysis or surgery will be necessary to restore adequate flow [41].

Vasospastic Injuries

Vasospastic disease is an inappropriate, reversible vascular constriction in the distal extremities in response to a variety of stimuli. It is most commonly a disorder of small arteries, precapillary arteries and cutaneous arteriovenous shunts of the fingers and is characterised by a three-phasic colour change and decreased skin temperature. The vasospastic cessation of digital artery flow produces pallor (white), followed by vasorelaxation and subsequent post capillary venule constriction resulting in desaturated blood-producing cyanosis (blue) and finally post ischaemic hyperaemia (red). The clinical picture is referred to as Raynaud's phenomenon and is a consequence of primary idiopathic disease or an associated symptom of a number of secondary causes, including normal physiological healing (Fig. 1.4).

In primary Raynaud's disease patients have an abnormally strong vasospastic response to cold and emotional stress with anatomically normal blood vessels. It typically has an early age of onset (second or third decade), occurs in young women, is bilateral and is very rarely associated with digital ulceration or digit threatening ischaemia, but can be painful. Secondary Raynaud's phenomenon has been associated with a host of underlying causes, such as collagen vascular disorders and occlusive disease. The classification into primary and secondary is important as it affects prognosis, severity and treatment.

Raynaud's phenomenon typically precedes the clinical onset of any systemic rheumatic conditions and as such its presence often precipitates aggressive assessments for underlying disease. Many non-inflammatory processes and most systemic rheumatic diseases have been linked to Raynaud's, the most frequently seen association is with systemic sclerosis (scleroderma). Raynaud's is thought to occur in more than 90 % of patients with scleroderma, 10–45 % with SLE, 30 % of patient's with Sjogren's, 20 % with dermatomyositis and 10–20 % with rheumatoid arthritis [42]. Furthermore, chronic occupational exposure, in the form of mechanical