Ian A. Trail
Andrew N. M. Fleming
Editors

Disorders of the Hand

Volume 1: Hand Injuries

Springer

Disorders of the Hand

Ian A. Trail • Andrew N.M. Fleming
Editors

Disorders of the Hand

Volume 1: Hand Injuries

Springer

Editors
Ian A. Trail
Wrightington Hospital
Wigan
Lancashire
UK

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

ISBN 978-1-4471-6553-8 ISBN 978-1-4471-6554-5 (eBook)
DOI 10.1007/978-1-4471-6554-5
Springer London Heidelberg New York Dordrecht

Library of Congress Control Number: 2014957713

© Springer-Verlag London 2015
This work is subject to copyright. All rights are reserved by the Publisher, whether the whole or part of the material is concerned, specifically the rights of translation, reprinting, reuse of illustrations, recitation, broadcasting, reproduction on microfilms or in any other physical way, and transmission or information storage and retrieval, electronic adaptation, computer software, or by similar or dissimilar methodology now known or hereafter developed. Exempted from this legal reservation are brief excerpts in connection with reviews or scholarly analysis or material supplied specifically for the purpose of being entered and executed on a computer system, for exclusive use by the purchaser of the work. Duplication of this publication or parts thereof is permitted only under the provisions of the Copyright Law of the Publisher's location, in its current version, and permission for use must always be obtained from Springer. Permissions for use may be obtained through RightsLink at the Copyright Clearance Center. Violations are liable to prosecution under the respective Copyright Law.
The use of general descriptive names, registered names, trademarks, service marks, etc. in this publication does not imply, even in the absence of a specific statement, that such names are exempt from the relevant protective laws and regulations and therefore free for general use.
While the advice and information in this book are believed to be true and accurate at the date of publication, neither the authors nor the editors nor the publisher can accept any legal responsibility for any errors or omissions that may be made. The publisher makes no warranty, express or implied, with respect to the material contained herein.

Printed on acid-free paper

Springer is part of Springer Science+Business Media (www.springer.com)

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

Acknowledgements

We are indebted to all of our co-authors without whom this publication would not have been possible. Despite their busy clinical practices and numerous other commitments, they have produced high quality chapters which we have thoroughly enjoyed reading and hope that you will find helpful in the treatment of your patients.

We are also particularly grateful to Springer for allowing us to pursue this project and would like to especially thank Rachel Glassberg for all her helpful advice and prompting.

Finally we would like to thank our secretaries, particularly Diane Allmark, and respective families who, for longer than we dare think, have put up with us reading and re-reading manuscripts on what they think is only a small part of the body!

Contents

1 Blood Vessels 1
Henk Giele and Richard Barton

2 Nerve Injury 23
Robert I.S. Winterton and Simon P.J. Kay

3 Injury: Tendons – Flexor 45
David Elliot

4 Injuries: Tendon – Extensor 67
Simon L. Knight

5 General Principles of Skin Cover and Flaps to the Elbow, Forearm, Wrist and Hand 89
Stewart Watson

6 Soft Tissue Cover of the Hand 133
Steven Lo and Mark Pickford

7 Distal Interphalangeal Joint and Fractures of the Distal Phalanx 163
Subodh Deshmukh and Christopher Armitstead

8 Proximal Interphalangeal Joint Injuries 175
Grey Giddins and Lawrence Moulton

9 Fractures of the Proximal Phalanx and Those Involving the Metacarpo-Phalangeal Joint 195
David J. Shewring

10 Injury-Fractures-Carpo-Metacarpal Joint and Metacarpal 217
Chye Yew Ng and Michael J. Hayton

11 Fractures of Carpal Bones Other than the Scaphoid 237
Matthew Nixon and Ian A. Trail

12 Acute Scaphoid Fractures 249
Nick R. Howells, Rouin Amirfeyz, and Tim R.C. Davis

13 Fractures of the Distal Radius and Distal Radioulnar Joint 259
Douglas A. Campbell and Louise A. Crawford

14 Hand Injuries in Children 285
David McCombe

15 Acute Thumb and Finger Instability 311
Sharifah Ahmad Roohi and Caroline Leclercq

16 Acute Carpal Instability 367
Ian A. Trail

17 Acute DRUJ Instability 379
Brian D. Adams

18 Massive Upper Limb Trauma 391
S. Raja Sabapathy and Hari Venkatramani

19 Hand Infections 415
Anthony Barabas and Andrew N.M. Fleming

20 Pain Management in Disorders of the Hand 437
Carmel Martin, Richard Kennedy, and Jeremy Cashman

Index 457

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

Keywords
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

I.A. Trail, A.N.M. Fleming (eds.), *Disorders of the Hand: Volume 1: Hand Injuries*,
DOI 10.1007/978-1-4471-6554-5_1, © Springer-Verlag London 2015

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

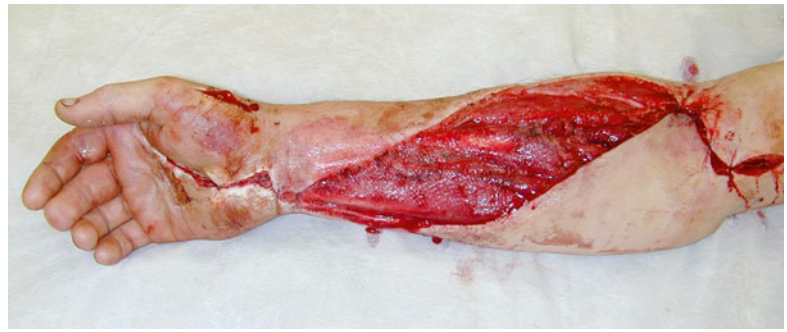


Fig. 1.2 Forearm and palmar fasciotomies

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

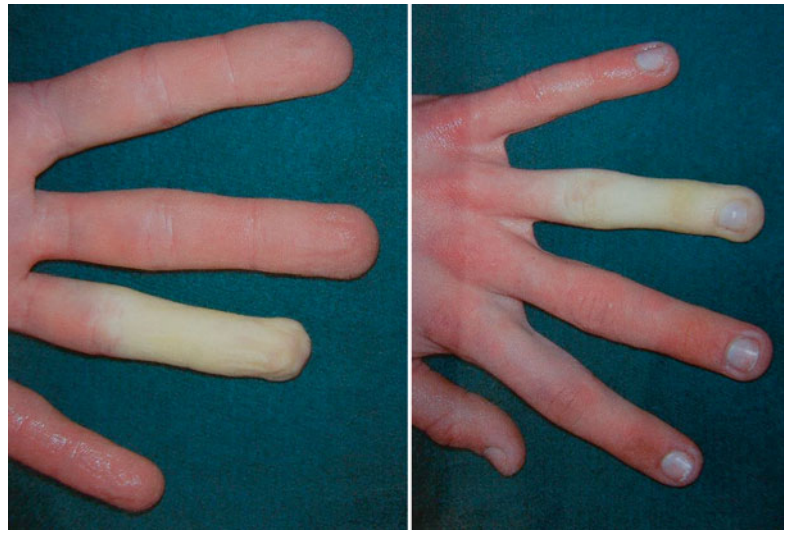


Fig. 1.4 Raynaud's phenomenon

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

vibration caused by pneumatic equipment, can cause secondary Raynaud's phenomenon [43, 44]. As can antineoplastic and chemotherapeutic medications, such as bleomycin and vinblastine, heavy metal intoxication, and a range of vasoactive drugs [45].

The goal of diagnostic testing in this instance is to allow differentiation of Raynaud's phenomenon from other vaso-occlusive conditions and to help distinguish between primary and secondary Raynaud's. In patients with mild disease and normal serology and sedimentation rates, the risk of an associated illness is small, it is likely the overall risk of progression to a systemic illness following the diagnosis of Raynaud's phenomenon is less than 10 % over 10 years [42]. Anticentromere antibodies (ACA) can be important in the early assessment, as ACA-positive patients have a much higher risk of progression to systemic sclerosis and digital ischemia leading to amputation [46].

Physical examination includes an assessment of the quality of pulses, evidence of fingertip necrosis and trophic change. An Allen's test performed at the wrist and digit will reveal any underlying occlusive disease. Bedside evaluation of nail fold capillaries under magnification is useful to distinguish early secondary systemic sclerosis associated Raynaud's. As the presence of characteristic enlarged and tortuous capillaries with 'drop out', has a high specificity for scleroderma [47].

Irrespective of the causes, the symptoms and signs are manifest via vasospasm of the small muscular arteries and arterioles of the digits. Normal vasomotor homoeostasis is maintained by the interaction between endothelium, smooth muscle and autonomic and sensory nerves that innervate the vessels. In primary Raynaud's phenomenon where the underlying cause is unknown, the primary defect is related to aberrant auto regulation of the microvasculature. The pathogenesis is likely in part to be caused by the altered concentrations of endothelial-dependent vasoregulators, specifically, a raised plasma endothelin concentration and reduced endothelial release of nitric oxide.

Endothelium related vasoregulation certainly plays an important role in secondary Raynaud's phenomenon, in which endothelial damage is common. Damaged endothelial cells exacerbate vasospasm and further compromise perfusion by mediating contraction of smooth muscle cells. There is an enhancement of procoagulant activity and reduction of fibrinolysis that promotes the formation of intravascular microthrombi and activates local inflammatory processes. Further debate on the pathogenesis questions the relative contributions of other vasoregulatory influences, such as the sympathetic nervous system, platelet activation and circulating factors.

Management is largely supportive, mild disease can be improved with measures such as cessation of smoking and avoidance of cold. These patients should also avoid substances that promote vasoconstriction such as β (beta) blockers, ergot alkaloids, amphetamines, cocaine, decongestants and caffeine. When pharmacological treatment is necessary it is generally oriented towards promoting vasodilatation [48].

Vasodilator therapy tends to be more effective in primary than secondary Raynaud's. Calcium channel antagonists such as nifedipine, are the mainstay of medical management. This group of drugs has a higher selectivity for vascular smooth muscle and reduced effects on cardiac function. Other pharmacologic modalities used include Angiotensin converting enzyme inhibitors, topical nitrates and iloprost; a synthetic prostacyclin analogue. Each has equivocal efficacy and their own set of unfavourable side effects [42]. Recently the use of Botulinum toxin A has shown promising results. It has been used effectively in primary and secondary Raynaud's, to provide symptomatic relief and heal long standing digital ulceration [49].

Surgery is reserved for refractory severe Raynaud's, with digital ischaemia or non-healing ulcers, options include reconstruction of occluded vessels or modification of sympathetic tone [50]. A sympathectomy can effectively reduce the severity of attacks, though results are poor in scleroderma associated disease [48]. Cervicothoracic sympathectomy has limited use in the treatment of upper extremity vasospastic disease, and digital sympathectomy has become the treatment of choice [51]. It involves separation of the digital arteries from the nerves, division of digital

nerve branches to the vessels and periarterial adventitial stripping. The superficial arterial arch is stripped over its entire distance, the origins of the three common digital vessels are exposed and periarterial sympathectomy is carried out along these vessels for at least 1 cm.

Arteriovenous Fistula

An arteriovenous fistula may be acquired surgically for the benefit of haemodialysis in renal patients, or be the consequence of penetrating trauma. Traumatic fistulas establish a direct arterial-venous communication that can cause vascular shunting sufficiently significant to result in compromise of distal perfusion. Recognition relies on a clinically detectable mass and palpable thrill, aided by colour duplex ultrasonography. Surgical correction requires excision of the fistula and arterial reconstruction.

A distal hypoperfusion ischaemic syndrome occurs occasionally after dialysis access placement of arteriovenous fistulas (AVF). The distal ischaemia in these patients may be a result of elevated venous pressures, occlusive/stenotic arterial lesions or because of steal phenomenon. Steal phenomenon is characterised by retrograde flow in the artery segment distal to the anastamosis and occurs to some degree in most patients with radio-cephalic AVF. Typically the steal phenomenon is subclinical and only rarely leads to distal ischaemia. In the event of ischaemic complications consideration should be given to maximising the collateral circulation, for example by performing an angioplasty of the ulnar artery in a patient with steal phenomenon from a radio-cephalic fistula. Often the solution is to tie off the offending AVF and create a new site for haemodialysis access on the other limb. If this is not possible, a graft that bypasses the fistula to revascularise the distal limb, with ligation of the artery just distal to the fistula, will prevent the retrograde flow and re-establish adequate distal inflow [52, 53].

Systemic Processes Causing Vessel Injury

There are numerous systemic processes that can affect the vascularity of the upper limb.

Connective Tissue Disease

Connective tissue disorders are a heterogeneous group of disease entities with considerable overlap. The associated upper limb vascular disorders are characterised by the reduced calibre of the ulnar, radial and digital arteries, often with superimposed vasospasm and areas of narrowing interspersed with normal segments. The segmental occlusion of vessels is secondary to antibody-antigen complex deposition, which results in endothelial damage, fibrinoid thickening and intimal hyperplasia. This ultimately leads to an obliterative endarteritis and symptoms such as digital ischaemia that can predate the systemic signs of the disease by many years.

Systemic scleroderma refers to a variety of connective tissue disorders characterised by arteriosclerosis and varying degrees of extracellular collagen accumulation, leading to tissue and visceral fibrosis. Several subcategories exist, including systemic sclerosis and CREST syndrome (calcinosis cutis, Raynaud's Phenomenon, esophageal dysfunction, sclerodactyly, telangiectasia). The ulnar artery is occluded in over a third of cases, while the radial artery is invariably spared. As with other vasculitides, the proper digital arteries are the most commonly affected vessels. Patients frequently suffer from recalcitrant digital ulceration because of the impaired cutaneous blood flow. Surgical intervention to fuse joint contractures in better positions of function, and to revascularise digits, reconstruct arteries, and sympathectomize digits may improve the perfusion of digits, heal ulcers, improve pain and improve function [54].

Polyarteritis nodosa is a rare disease with a male predilection, characterised by progressive necrotising inflammation of small to medium sized arteries. The resulting exudations contribute to the formation of palpable nodules and to irregularity throughout the course of the vessel. Immune mediated damage to the vasculature is thought to be the underlying process. Characteristic lesions in the hand include multiple short-segment stenoses of the proper and common digital arteries. Angiography and tissue biopsy are important tools in the diagnosis [55]. Steroids, Infliximab and immunosuppressants are the current therapies rather than surgery.

Fig. 1.5 Ischaemic digit tips after noradrenaline infusion in Intensive Therapy Unit

Buerger's disease also known as "thromboangitis obliterans" is an inflammatory disorder affecting segments of the small arteries of the peripheries of male smokers. Presenting initially with symptoms similar to Raynaud's the intermittent pain, coldness, colour change and sensory symptoms can become constant and progress to ulceration and gangrene. Stopping smoking is the treatment, perhaps allied with attempts at neoangiogenesis [56].

Sepsis

Toxins produced by pneumococcal and meningococcal sepsis result in sluggish peripheral blood flow, vasoconstriction and hypercoagulation. These features are often accentuated in a very ill patient by vasopressive medical therapy such as noradrenalin. The result is bilateral distal ischaemia that is mainly managed expectantly and may include splinting the hand and wrist in a safe position and passive manipulation of joints to ensure optimum potential for functional recovery. There has been a recent discussion regarding the benefit of "prophylactic fasciotomies" or skin and soft tissue releases in order to reduce the risk of amputation [57–60] (Fig. 1.5).

Blood Disorders

Several forms of prothrombotic blood dyscrasias can also be associated with upper extremity distal artery occlusion such as cryoglobulinaemia, myeloproliferative diseases, hyperviscosity syndromes and the presence of cold agglutinins. The pathophysiology is thought to involve small artery thrombosis and diagnosis is confirmed by specific laboratory testing.

Renal Insufficiency

The typical type of vascular disease that occurs in patients with renal insufficiency involves calcinphylaxis, which can produce calcification of the media of the digital arteries demonstrable on plain x-ray. These changes are typically found in patients with diabetes, chronic renal failure or post renal transplant and can result in gangrene or severe ischaemia of the hand. The prognosis is particularly poor [61].

References

1. Gupta C, et al. A morphological study of variations in the branching pattern and termination of the radial artery. Singapore Med J. 2012;53(3):208–11.
2. Ramirez AR, Gonzalez SM. Arteries of the thumb: description of anatomical variations and review of the literature. Plast Reconstr Surg. 2012;129(3):468e–76.
3. Singla RK, Kaur N, Dhiraj GS. Prevalence of the persistant median artery. J Clin Diagn Res. 2012;6(9):1454–7.
4. Imanishi N, Nakajima H, Aiso S. Anatomic study of the venous drainage architecture of the forearm skin

and subcutaneous tissue. Plast Reconstr Surg. 2000;106(6):1287–94.
5. Suami H, Taylor GI, Pan WR. The lymphatic territories of the upper limb: anatomical study and clinical implications. Plast Reconstr Surg. 2007;119(6):1813–22.
6. Suami H, Pan WR, Taylor GI. Changes in the lymph structure of the upper limb after axillary dissection: radiographic and anatomical study in a human cadaver. Plast Reconstr Surg. 2007;120(4):982–91.
7. Puttarajappa C, Rajan DS. Images in clinical medicine. Allen's test. N Engl J Med. 2010;363(14):e20.
8. Reimer P, Landwehr P. Non-invasive vascular imaging of peripheral vessels. Eur Radiol. 1998;8(6):858–72.
9. Zweifler AJ, Trinkaus P. Occlusive digital artery disease in patients with Raynaud's phenomenon. Am J Med. 1984;77(6):995–1001.
10. Sitzmann JV, Ernst CB. Management of arm arterial injuries. Surgery. 1984;96(5):895–901.
11. Hafez HM, Woolgar J, Robbs JV. Lower extremity arterial injury: results of 550 cases and review of risk factors associated with limb loss. J Vasc Surg. 2001;33(6):1212–9.
12. Shanmugam V, et al. Management of upper limb arterial injury without angiography – Chennai experience. Injury. 2004;35(1):61–4.
13. Cikrit DF, et al. An experience with upper-extremity vascular trauma. Am J Surg. 1990;160(2):229–33.
14. Aftabuddin M, et al. Management of isolated radial or ulnar arteries at the forearm. J Trauma. 1995;38(1):149–51.
15. Meharwal ZS, Trehan N. Functional status of the hand after radial artery harvesting: results in 3,977 cases. Ann Thorac Surg. 2001;72(5):1557–61.
16. Gates JD, Knox JB. Axillary artery injuries secondary to anterior dislocation of the shoulder. J Trauma. 1995;39(3):581–3.
17. Ebraheim NA, et al. Scapulothoracic dissociation (closed avulsion of the scapula, subclavian artery, and brachial plexus): a newly recognized variant, a new classification, and a review of the literature and treatment options. J Orthop Trauma. 1987;1(1):18–23.
18. Dormans JP, Squillante R, Sharf H. Acute neurovascular complications with supracondylar humerus fractures in children. J Hand Surg Am. 1995;20(1):1–4.
19. Garbuz DS, Leitch K, Wright JG. The treatment of supracondylar fractures in children with an absent radial pulse. J Pediatr Orthop. 1996;16(5):594–6.
20. Shaw BA, et al. Management of vascular injuries in displaced supracondylar humerus fractures without arteriography. J Orthop Trauma. 1990;4(1):25–9.
21. White L, Mehlman CT, Crawford AH. Perfused, pulseless, and puzzling: a systematic review of vascular injuries in pediatric supracondylar humerus fractures and results of a POSNA questionnaire. J Pediatr Orthop. 2010;30(4):328–35.
22. Keen RR, et al. Autogenous vein graft repair of injured extremity arteries: early and late results with 134 consecutive patients. J Vasc Surg. 1991;13(5):664–8.
23. Guyton GP, Shearman CM, Saltzman CL. Compartmental divisions of the hand revisited. Rethinking the validity of cadaver infusion experiments. J Bone Joint Surg Br. 2001;83(2):241–4.
24. Taylor RM, Sullivan MP, Mehta S. Acute compartment syndrome: obtaining diagnosis, providing treatment, and minimizing medicolegal risk. Curr Rev Musculoskelet Med. 2012;5(3):206–13.
25. Matsen 3rd FA, Winquist RA, Krugmire Jr RB. Diagnosis and management of compartmental syndromes. J Bone Joint Surg Am. 1980;62(2):286–91.
26. Kalyani BS, et al. Compartment syndrome of the forearm: a systematic review. J Hand Surg Am. 2011;36(3):535–43.
27. Garg K, et al. Open surgical management of complications from indwelling radial artery catheters. J Vasc Surg. 2013;58:1325–30.
28. Larsen BT, et al. Surgical pathology of hypothenar hammer syndrome with new pathogenetic insights: a 25-year institutional experience with clinical and pathologic review of 67 cases. Am J Surg Pathol. 2013;37:1700–8.
29. Rutherford RB, et al. Recommended standards for reports dealing with lower extremity ischemia: revised version. J Vasc Surg. 1997;26(3):517–38.
30. Wheatley MJ, Marx MV. The use of intra-arterial urokinase in the management of hand ischemia secondary to palmar and digital arterial occlusion. Ann Plast Surg. 1996;37(4):356–62; discussion 362–3.
31. Bedford RF, Wollman H. Complications of percutaneous radial-artery cannulation: an objective prospective study in man. Anesthesiology. 1973;38(3):228–36.
32. Valentine RJ, Modrall JG, Clagett GP. Hand ischemia after radial artery cannulation. J Am Coll Surg. 2005;201(1):18–22.
33. Maiman MH, Bookstein JJ, Bernstein EF. Digital ischemia: angiographic differentiation of embolism from primary arterial disease. AJR Am J Roentgenol. 1981;137(6):1183–7.
34. Arquilla B, et al. Acute arterial spasm in an extremity caused by inadvertent intra-arterial injection successfully treated in the emergency department. J Emerg Med. 2000;19(2):139–43.
35. Andreev A, et al. Severe acute hand ischemia following an accidental intraarterial drug injection, successfully treated with thrombolysis and intraarterial Iloprost infusion. Case report. Angiology. 1995;46(10):963–7.
36. Sen S, Chini EN, Brown MJ. Complications after unintentional intra-arterial injection of drugs: risks, outcomes, and management strategies. Mayo Clin Proc. 2005;80(6):783–95.
37. Harris Jr EJ, et al. Surgical treatment of distal ulnar artery aneurysm. Am J Surg. 1990;159(5):527–30.
38. Nehler MR, et al. Upper extremity ischemia from subclavian artery aneurysm caused by bony abnormalities of the thoracic outlet. Arch Surg. 1997;132(5):527–32.

39. Siddiqui MU, Khurram D, Elder M. Management of brachial artery thrombosis post catheterization. J Invasive Cardiol. 2013;25(3):E60–2.
40. Arshad A, McCarthy MJ. Management of limb ischaemia in the neonate and infant. Eur J Vasc Endovasc Surg. 2009;38(1):61–5.
41. Coombs CJ, et al. Brachial artery thrombosis in infants: an algorithm for limb salvage. Plast Reconstr Surg. 2006;117(5):1481–8.
42. Herrick AL. Pathogenesis of Raynaud's phenomenon. Rheumatology (Oxford). 2005;44(5):587–96.
43. Pyykko I, Gemne G. Pathophysiological aspects of peripheral circulatory disorders in the vibration syndrome. Scand J Work Environ Health. 1987;13(4):313–6.
44. Gemne G, et al. The Stockholm Workshop scale for the classification of cold-induced Raynaud's phenomenon in the hand-arm vibration syndrome (revision of the Taylor-Pelmear scale). Scand J Work Environ Health. 1987;13(4):275–8.
45. Doll DC, Ringenberg QS, Yarbro JW. Vascular toxicity associated with antineoplastic agents. J Clin Oncol. 1986;4(9):1405–17.
46. Wigley FM, et al. Anticentromere antibody as a predictor of digital ischemic loss in patients with systemic sclerosis. Arthritis Rheum. 1992;35(6):688–93.
47. Anderson ME, et al. Computerized nailfold video capillaroscopy–a new tool for assessment of Raynaud's phenomenon. J Rheumatol. 2005;32(5):841–8.
48. Landry GJ. Current medical and surgical management of Raynaud's syndrome. J Vasc Surg. 2013;57(6):1710–6.
49. Fregene A, Ditmars D, Siddiqui A. Botulinum toxin type A: a treatment option for digital ischemia in patients with Raynaud's phenomenon. J Hand Surg Am. 2009;34(3):446–52.
50. Tomaino MM, Goitz RJ, Medsger TA. Surgery for ischemic pain and Raynaud's phenomenon in scleroderma: a description of treatment protocol and evaluation of results. Microsurgery. 2001;21(3):75–9.
51. Kotsis SV, Chung KC. A systematic review of the outcomes of digital sympathectomy for treatment of chronic digital ischemia. J Rheumatol. 2003;30(8):1788–92.
52. Beathard GA, Spergel LM. Hand ischemia associated with dialysis vascular access: an individualized access flow-based approach to therapy. Semin Dial. 2013;26(3):287–314.
53. Aimaq R, Katz SG. Using distal revascularization with interval ligation as the primary treatment of hand ischemia after dialysis access creation. J Vasc Surg. 2013;57(4):1073–8; discussion 1078.
54. Bogoch ER, Gross DK. Surgery of the hand in patients with systemic sclerosis: outcomes and considerations. J Rheumatol. 2005;32(4):642–8.
55. Stone JH. Polyarteritis nodosa. JAMA. 2002;288(13):1632–9.
56. Dargon PT, Landry GJ. Buerger's disease. Ann Vasc Surg. 2012;26(6):871–80.
57. Warner PM, et al. Current management of purpura fulminans: a multicenter study. J Burn Care Rehabil. 2003;24(3):119–26.
58. Numanoglu A, et al. Meningococcal septicaemia complications involving skin and underlying deeper tissues–management considerations and outcome. S Afr J Surg. 2007;45(4):142–6.
59. Miedema A, et al. Improving outcome in meningococcal disease: don't forget compartment syndrome! Pediatr Crit Care Med. 2008;9(3):e20–2.
60. Penington AJ, Craft RO, Tilkorn DJ. Plastic surgery management of soft tissue loss in meningococcal septicemia: experience of the Melbourne Royal Children's Hospital. Ann Plast Surg. 2007;58(3):308–14.
61. Tzamaloukas AH, et al. Hand gangrene in diabetic patients on chronic dialysis. ASAIO Trans. 1991;37(4):638–43.

Nerve Injury

2

Robert I.S. Winterton and Simon P.J. Kay

Keywords
Nerve injury • Peripheral nerve • Denervation • Reinnervation • Motor neuron • Sensory function • Motor function • Nerve repair • Traction injury • Nerve graft • Nerve conduit • Nerve transfer

Introduction

The first known reference to the central nervous system is found in the Edwin Smith Medical Papyrus, a manuscript originating in approximately 3500BC which contains the word "brain" along with a description of the coverings of the brain [1]. Early physicians, such as Hippocrates, did not distinguish peripheral nerves from tendons, and even when Galen made the distinction in the second century AD, nerve repairs were not attempted. Surgeons worried that manipulating the nerve stumps would cause convulsions, or assumed that function would recover regardless of whether repair was attempted. The development of the science of neurophysiology between 1830 and 1870 led surgeons to attempt to repair nerves, and by World War I primary neurosynthesis was accepted practice.

The management of peripheral nerve injuries advanced significantly as a result of the clinical experience gained during each World War. In the latter twentieth century the evolution of microsurgical techniques, improvements in surgical equipment, and the consistently advancing field of neuroscience also contributed. In the 1970s, work by Millesi [2] and Terzis [3], amongst others, showed both clinically and experimentally that tension across a neurosynthesis inhibits nerve regeneration, and as a result many surgeons adopted the use of nerve grafts to bridge defects.

Nerve injury is common. One study from Canada reported that 2.8 % of trauma patients had an injury to at least one major peripheral nerve [4]. Estimates from the US suggest that 200,000 peripheral nerve lacerations are sustained nationally every year.

This chapter discusses the management of nerve injury, and its basis in neuroscience. Normal nerve anatomy and microanatomy will be considered, as will the science of nerve division and repair. The pathophysiology of nerve

R.I.S. Winterton, BMedSci, MBBS, MRCS, MPhil FRCS (Plast)
Department of Plastic Surgery, Wythenshawe Hospital, Southmoor Road, Manchester M23 9LT, UK
e-mail: wintertonris@hotmail.com

S.P.J. Kay, FRCS, FRCSE(hon) FRCS (Plas Surg) (✉)
Department of Plastic Surgery, Leeds General Infirmary, Great George Street, Leeds LS1 3EX, UK
e-mail: spjkay@gmail.com

I.A. Trail, A.N.M. Fleming (eds.), *Disorders of the Hand: Volume 1: Hand Injuries*,
DOI 10.1007/978-1-4471-6554-5_2, © Springer-Verlag London 2015

crush or traction will also be discussed and developments in the fields of nerve grafting, nerve conduits and neuroprotection will be reviewed.

Nerve Anatomy and Microanatomy

Nerve Micro-Anatomy

The basic cellular component of a nerve is a neuron, consisting of a cell body and an axon. The cell body of a motor neuron lies in the anterior horn of the spinal cord whilst sensory neuron cell bodies reside in the dorsal root ganglion. Axons lie in continuity from the cell body to distal target organs, and may be myelinated or unmyelinated. Myelinated axons are wrapped in the bilayer basement membrane of an accompanying Schwann cell. Manufacturing and energy exchange occur in the cell body and nucleus and include the enzymes required for neurotransmitter synthesis. Some intracellular components may travel along the length of an axon at speeds of 410 mm per day, whilst other components such as structural proteins move at a maximal rate of 1–6 mm per day [5]. It is this slower transport mechanism that limits the rate of nerve regeneration.

An axon and its associated Schwann cell sheath is called a nerve fibre, and each nerve fibre is surrounded by a sheath (the endoneurium). A bundle of nerve fibres (which is surrounded by collagen and elastin) is called a nerve fascicle. A fascicle is invested with perineurium, composed of concentric layers of flat cells with prominent basement membranes that fit together and are linked by "tight junctions". The perineurium serves to resist traction, and also acts as an extension to the blood brain barrier by controlling the intraneural environment via active diffusion control (Fig. 2.1).

A fascicle is the smallest subunit of a nerve that can be surgically manipulated. Fascicles are not separate cables that run in parallel throughout a length of nerve, but instead have numerous interconnections that result in the formation of a complex intraneural plexus. The plexus complexity varies over the length of a nerve. Generally, interconnections are most numerous in the proximal part of major nerves, indeed by the time a nerve had reached the forearm, fascicles may be dissected over long distances before they fuse with adjacent fascicles [6]. Identification and mapping of fascicles and fascicular groups has an impact on the management

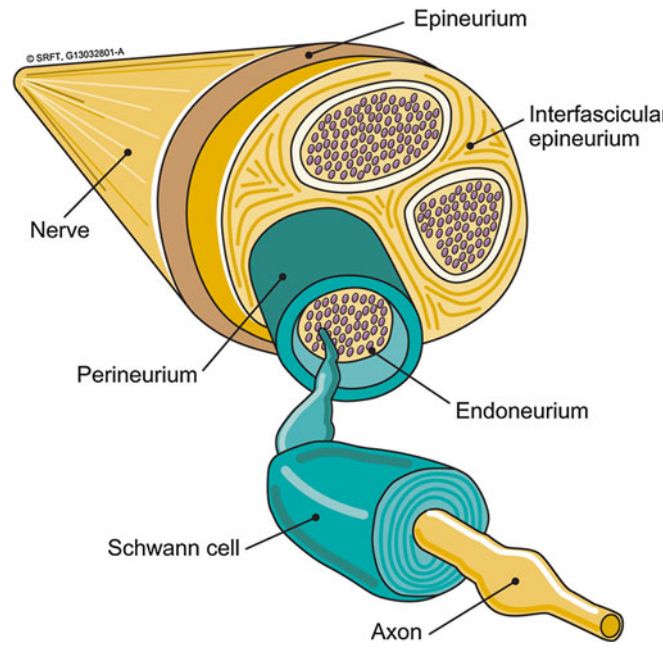


Fig. 2.1 Diagram showing the internal anatomy of a peripheral nerve

of peripheral nerve injuries. Fascicular groups may be isolated over greater distances than individual fascicles and connections are fewer between fascicular groups [6, 7]. Corresponding fascicular groups can be identified in distal and proximal segments, and should be used to orientate the divided nerve before repair, optimising sensory and motor recovery.

A collection of fascicles is a nerve. It is ensheathed by epineurium, a loose connective tissue composed mostly of collagen, which adheres to nutrient vessels as they enter the nerve. This epineurium is usually haemorrhagic after contusion or traction injury, and it is the epineurium which thickens after chronic nerve compression [8]. It is the epineurium that is dissected during intraneural neurolysis. The percentage of nerve cross sectional area that is occupied by epineurium differs along each nerve, from nerve to nerve, and between individuals. Higher amounts of epineurium are often found around joints, and the proportion of epineurium may vary widely from 25 to 75 % of the cross sectional composition of a nerve [9].

Epineurium may reach 2–3 mm thickness in chronic inflammation and much of the scar formed after nerve traction results from proliferation of epineural fibroblasts. It is the epineurium that is sutured in most nerve repairs.

Blood Supply and Surgical Anatomy

Peripheral nerves have a rich segmental blood supply. Nerves are supplied by longitudinal vessels in the epineurium, which in turn supply a segmental plexus that lies within the perineurium. This segmental plexus gives rise to capillary sized vessels that spread uniformly throughout the fascicles. There are no lymphatics within the endoneurium. Because of this longitudinal interconnections between arterioles and venules, an in tact peripheral nerve may survive extensive mobilization from its bed, although this property is proportionally lost if the nerve is transected, as this disrupts the blood supply as well as the axons.

Peripheral nerves rarely cross a joint at its axis of motion. Consequently a nerve moves relative to its surrounding tissue and changes in length as a consequence of joint motion. The mesoneurium is loose areolar tissue around the outside of a nerve that allows for this translation, and the segmental blood supply enters through this layer. The changes in nerve length can be dramatic, for instance the median nerve must elongate by 4.5 % on arm extension and shorten by 4.5 % during full flexion from a resting position [10]. Surgery can create fibrosis within the mesoneurium that leads to tethering and, as a consequence, traction with normal joint motion.

Nerves have viscoelastic properties such as stress relaxation (decreasing tension when pulled to a fixed length) and creep (elongation with constant tension). Normally nerves are elastic and under little tension, although towards the end of joint excursion, increased strain raises intraneural pressure and hence decreases perfusion. An injured nerve has altered biomechanics and therefore is more susceptible to the reduced perfusion caused by joint motion, and also has higher perfusion requirements at the site of repair. Increased neural ischaemia, scar formation and subsequent reduced axon regeneration, may lead to impaired recovery of nerve function. Prevention of this chain of events may require avoidance of tension by nerve grafting, or other manoeuvres such as anterior transposition of the ulnar nerve at the elbow.

Nerve Division

Physiology of Peripheral Nerves Following Injury

Augustus Volney Waller, an English neurophysiologist, originally described the changes that occur in a nerve following a transection or crush injury. Wallerian degeneration (also known as anterograde or orthograde degeneration) is a process in which the part of the axon separated from the neuron's cell body as a result of the injury, degenerates. The length of the proximal segment that degenerates is proportional to the nature of the injury and the amount of force that is absorbed by the nerve. A nerve that is transected by a sharp division has less proximal injury than one that is

avulsed in a heavy machinery accident. Within 24 h after transection, each single axon begins regenerating by sending out multiple axon sprouts. At the tip of each sprout is a "growth cone" that consists of tentacularfilopodia rich in actin. The tips of these filopodia explore the distal environment in search of fibronectin and laminin, which are found in the basal lamina of the distal endoneurial tubes.

In the absence of any mechanical barrier, the growth cones are guided by neurotropic factors that are present in the surrounding environment. Once the regenerating filopodia encounter the appropriate substrate, they adhere to the structure and draw the entire growth cone distally. This process of contact guidance causes the axon to grow further towards the distal nerve segment.

In the distal nerve segment, anterograde Wallerian degeneration occurs along the entire length of the nerve. After transection, Schwann cells proliferate and phagocytose the degenerating myelin and axonal components. Endoneurial tubes collapse, and the proliferating Schwann cells organise themselves along channels. As the axon sprouts from the proximal nerve stump regenerate into the distal nerve remnant, they find the fibronectin and laminin in the basal lamina and are drawn distally.

The preference of a nerve fiber to grow toward a nerve instead of other tissue depends on a critical gap across which the fibre responds to the influences of the distal nerve. Current research suggests that the expression of various Schwann cell and myelin-associated glycoproteins may facilitate or impede the regeneration of damaged axons to their correct targets.

The axon continues to grow until it reaches an end organ or a mechanical barrier. If an appropriate end organ is reached, as when a motor axon that reaches a motor end plate, neurotrophic hormones signal the cell nucleus and the surrounding tissues and the axon is allowed to mature. If an inappropriate connection has been made, such as a motor axon to a Meissner's corpuscle, the errant axon involutes.

The end organ itself, whether a motor end plate or a sensory receptor, also undergoes atrophy. Unlike the motor system in which functional recovery is limited after a period of 12–18 months, recovery of Meissner's corpuscles, Pacinian corpuscles, and Merkel cells is possible years after nerve injury. Functional recovery depends on the number of motor fibers correctly matched with motor endplates and the number of sensory fibers correctly matched with sensory receptors, in a timely fashion.

Morphology of Peripheral Nerves Following Injury

The microscopic appearance of peripheral nerves following injury was described by Terenghi in 1998 [11].

The proximal stump of a divided nerve shows regenerating clusters of unmyelinated and myelinated axons as one moves proximo-distally. This is succeeded by an outgrowth zone of minifascicles of myelinated and unmyelinated axons, and their associated Schwann cells, all lying within a well vascularised collagen-rich matrix, as the neuroma is approached. There is a corresponding change in the perineural sheath over the same length of nerve. The perineurium becomes disorganised at the distal tip of the proximal stump and disappears in the outgrowth zones. However there are perineural-like cells present which may migrate with the regenerating axons and Schwann cells.

The neuroma of the nerve consists of masses of irregular minifascicles, each one enclosed by a layer of perineural-like cells. These axons are associated with Schwann cells positive for the S-100 stain and these in turn are associated with a basal lamina.

In the distal stumps, collagen deposition and endoneurial tube shrinkage are uniformly present. The level to which evidence of re-innervation is seen varies. However, all stumps contain PGP-9.5 positive axons, which are co-localised with S-100 positive Schwann cells, with some containing variable amounts of myelinated axons. There appears to be fewer immunoreactive fibres present in the distal stumps than in the proximal stumps.

Schwann cells were demonstrated to be present within these stumps at a period of 53 months post injury. There was no Wallerian degeneration

seen and no evidence of fragmentation of the basal laminae.

This study by Terenghi et al. [11] confirmed that the morphology following injury is similar in humans to that described in animal models. It was noted that even a neuroma in continuity seemed to provide a rich source of regenerating axons, although few of these entered the distal stump, which conformed to the view that a chronically denervated stump provides a less conducive microenvironment for axon regeneration.

If one accepts that the morphology within injured peripheral nerves is similar in mammalian animal models and humans, then given the findings in rat studies on axon-Schwann cell interactions, one might presume that human axon-Schwann cell interactions in chronically denervated nerves are also inadequate, which may contribute to the poor outcome after nerve reconstruction.

Giannini and Dyck [12] has described the appearance of the basement membrane in Schwann cells following denervation. Electron micrographic studies (in rat peroneal nerves) showed that as time progressed following denervation, the basement membrane became discontinuous and eventually dispersed. It is suggested that this alteration in the scaffold by which axons are able to reinnervate distal nerve stumps, may partially explain why it takes longer for distal nerve stumps to recovery if they have been denervated for a prolonged period.

Furthermore, it has been shown experimentally that chronically denervated Schwann cells down-regulate expression of receptors for axonally derived ligands, specifically c-erbB4 or c-erbB-2 [13], such that axonal growth cone chemotaxis is reduced, as there is a close correlation between levels of receptor expression by denervated Schwann cells and the extent to which distal stumps are reinnervated.

How Does the Response of Denervated Schwann Cells to Reinnervation Relate to Time?

When a peripheral nerve fibre is divided and the two ends retract, the axons within the distal stump degenerate. If the nerve ends can be sutured, or bridged with a short cable graft then regenerating axons typically penetrate the distal stump within a few weeks of injury. However, if the repair is delayed or the graft length is long, few axons will penetrate the distal stump and recovery will be severely compromised [14].

It has been supposed that physical changes in the microenvironment of a chronically denervated distal nerve stump, such as endoneural fibrosis or atrophy, block axonal regeneration. Conversely, we also know that despite this atrophy, they rarely lose all of their Schwann cells, even after 12–18 months [15]. However, despite the presence of Schwann cells, their functionality remains to be defined fully. Any impairment of axon-Schwann cell signaling could contribute significantly to the failure of nerve repair.

Protection of Motor Neurons and Sensory Neurons Following Injury

When considering reconstruction of major nerve function following injury, the principle aim remains motor recovery. However, sensory recovery is also important if a satisfactory functional outcome is to be achieved.

In recent years it has been established that central loss of neurons contributes to poor nerve recovery [16–18], and this is time and site dependent. Approximately 50 % of ventral horn spinal motor neurons die following ventral root transection, and this figure rises to 80–90 % following root avulsion [19, 20]. Neurons can die by passive necrosis, or active cell death (apoptosis). Necrosis is rapid and cannot practically be prevented, although the period of active cellular death lasts some weeks. There is evidence that adjuvant pharmacotherapy with N-acetyl cysteine (NAC) is able to prevent cellular death in sensory neurons following distal axotomy [21] and in motor neurons following ventral rhizotomy [22]. Though surgical intervention may also benefit neuronal survival, it is often less practical in vivo for a variety of reasons.

Protection of the proximal motor neurons from active cell death may in future allow a

potentially delayed and refined intervention to be applied to the injured nerve, which subsequently may allow improved recovery.

Clinical Assessment of the Injured Nerve

History

Trauma to the upper limb that results in a wound, a crush or traction, should raise the suspicion of an injury to a peripheral nerve. The mechanism of injury determines how much degeneration occurs in the proximal nerve segment, and also indicates whether one should anticipate a nerve deficit. It is important to determine the sharpness and width of the cutting object, the degree of traction or crushing, together with an estimate of the force that was involved and its duration of application.

The time elapsed since the injury is also important. Most nerve injuries are seen within the first 24 h, although injuries may not present until much later. Delayed presentation implies scar tissue at the nerve stumps and the possibility of intractable nerve retraction. In addition, following repair, return of motor function may be poor depending on the level of injury and the length of time that the muscle has been denervated, as a result of motor endplate decay.

Symptoms of numbness, paraesthesia or weakness should be elicited. It is also important to determine preoperatively whether there was any history of nerve injury or neuropathy prior to the accident. Specific elements of the patient's general condition, age and occupation will also guide planning to appropriately reconstruct the deficit.

General Assessment

In the emergency setting, where life or limb is at risk, making a diagnosis of peripheral nerve injury and treating the injured nerve may be low on the list of priorities. Once the acute phase is over, however, the loss of function of an injured nerve or the pain related to a nerve injury often dominates the subsequent clinical picture.

If there are wounds, a peripheral nerve in the region of the injury may be injured. If there are no open wounds, a peripheral nerve may be injured in proximity to a closed fracture or dislocation. Detailed, repeated neurological examination, along with the recording of examination findings, is important in identifying and defining the injury.

Assessment of Sensory Function

Sensibility on the hand may be examined with a blunt object such as a paper clip, and testing with a needle is discouraged. It is worth remembering that in the patient already in pain, this stimulus might not be as readily perceived as it would in a non-trauma setting.

The extent of sensory nerve injury is best determined by moving and static two-point discrimination, which are measurements of innervation density and the number of fibers innervating sensory end organs. Light moving touch, for example, evaluates the innervation of large A-β fibers and can be quickly screened with the valid and reliable "ten test" [23] (see below). Vibration instruments and Semmes-Weinstein monofilaments are used as threshold tests to evaluate the performance level of nerve fibers and are more useful in evaluating chronic compressive neuropathies. Testing is also performed after nerve repair to assess the quality of nerve repair, determine the need for revision, and monitor recovery.

Division of the median, ulnar, or radial nerves should not be a diagnostic dilemma in the cooperative patient. In the acute setting, the examiner will elicit the presence or absence of gross sensation in the following areas (Fig. 2.2):

- Volar, proximal third of the distal phalanx of the index or long finger for the median nerve
- Volar, proximal third of the distal phalanx of the little finger for the ulnar nerve
- Dorsum of the thumb index web space for the radial nerve
- Proximal ulnar aspect of the thenar eminence for the palmar cutaneous branch of the median nerve
- Dorsal metacarpal of the little finger for the dorsal cutaneous branch of the ulnar nerve

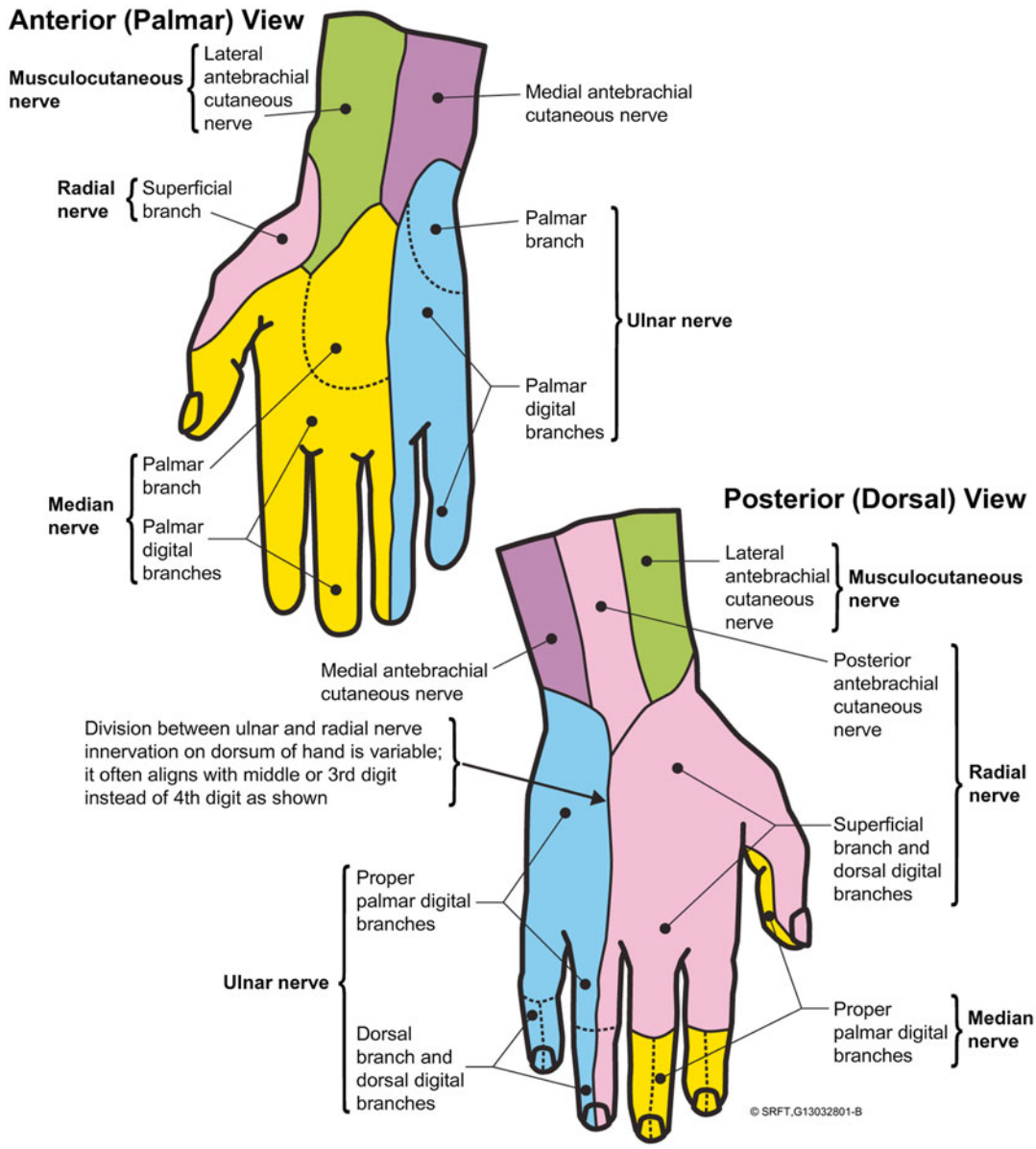


Fig. 2.2 Diagram showing distribution of sensory supply to the hand

When examining digital nerves patients may be asked to grade their appreciation of sensibility from 1 to 10 [23]. By establishing a "normal" score of 10 on an uninjured digit, patients will often record 8–9 out of 10 where no nerve injury is seen but the digit is generally swollen, 6–7 out of ten where the digital nerve is contused and 2–3 out of ten where the nerve is divided. These guides are less reliable in the patient presenting several days after their injury.

More proximal or brachial plexus injuries are better assessed by dermatome assessment. Knowledge regarding the distribution of the sensory dermatomes in the upper limb allows sharp and light touch, together with vibration, to be assessed in each area, thus guiding the examiner

towards the correct identification of the injured nerve root or brachial plexus element.

When assessing hand sensibility following severe proximal injuries it may not be practical to carry out a detailed assessment, and the clinical situation is often best defined by the answer to questions such as:

- Can you feel me touching you?
- Can you tell which side of the hand I am touching?
- Can you tell which finger I am touching?

Assessment of Motor Function

The extent of a motor nerve injury is determined by an evaluation of weakness, loss of function, and muscle atrophy. Evaluation of motor function is generally easier than evaluation of sensibility. If a motor nerve is completely divided then the muscle that is innervated by that nerve does not voluntarily contract. To test a given muscle, the examiner first asks the patient to make active movement. To determine power, the examiner places the patient into the position to which they would be moved if the muscle were functioning, then asks the patient to maintain that position against a force directed in the opposite direction.

For example, to evaluate median nerve motor function at the wrist the abductor pollicisbrevis muscle is tested. The patient's thumb is placed into palmar abduction, while the examiner's finger is placed on the thenar muscles. The patient is then asked to resist as the examiner attempts to push the thumb into adduction. The examiner then notes any contraction or fullness in the thenar eminence. Other examples of muscles that can be tested for nerve injury in the forearm are the flexor pollicis longus and the flexor digitorum superficialis for the median nerve, the abductor digiti minimi and the flexor carpi ulnaris for the ulnar nerve and the extensor pollicislongus, the extensor carpi radialis longus, and extensor carpi radialis brevis for the radial nerve.

The Medical Research Council (MRC) grading of muscle power is widely used to assess muscle function. The patient's effort is graded on a scale 0–5:

- Grade 5: Muscle contracts normally against full resistance
- Grade 4: Muscle strength is reduced but muscle contraction can still move joint against resistance
- Grade 3: Muscle strength is further reduced such that the joint can be moved only against gravity with the examiner's resistance completely removed. As an example, the elbow can be moved from full extension to full flexion starting with the arm hanging down at the side
- Grade 2: Muscle can move only if the resistance of gravity is removed. As an example, the elbow can be fully flexed only if the arm is maintained in a horizontal plane
- Grade 1: Only a trace or flicker of movement is seen or felt in the muscle or fasciculations are observed in the muscle
- Grade 0: No movement is observed

The value of this grading system in the upper limb is, however, limited to the large proximal joints. Gravity has little influence over the posture and movement of the intrinsic muscles of the hand or the supinators/pronators of the wrist. When recording the power of these movements it is more useful to use descriptive terms such as:

- Flicker of movement
- Movement, but not against resistance
- Limited ability to overcome resistance
- Strong movement against resistance
- Normal power

In cases of delayed presentation, muscle fibers lose trophic stimulation and decrease in bulk. Clinically this is apparent as muscle wasting which implies prolonged loss of innervation.

Clinical Pearl

An accurate knowledge of dermatome and sensory distribution is essential to accurately assess nerve injury.

Similarly, a thorough knowledge of muscle innervation and action is important to detect motor deficit.

Other Techniques

In the child or unconscious patient, a rarely used but helpful test for nerve division is the water immersion test. A normally innervated fingertip wrinkles within 4 min when placed in 40 °C water [24]. The absence of wrinkling of a finger when it is immersed indicates that the peripheral nerve that innervates that fingertip is not in continuity.

A more straightforward assessment may be made of sweating within the dermatome of the nerve in question. If the nerve is not functioning, the skin will feel characteristically dry to touch as it loses the ability to excrete sweat. If nerve function returns, the skin will once again be able to excrete sweat and the normal tactile qualities of the skin will be restored.

Nerve Recovery After Direct Repair Following Division

Terms

Although individual nerve fibres support axoplasmic flow, nerves are not hollow tubes. Therefore joining two ends of nerves is best termed a nerve repair or neurosynthesis, not an anastomosis. When sutures are placed through the epineurium, any neurosynthesis is an epineurial neurosynthesis. When the sutures are placed through the epineurium and perineurium, joining individual fascicles, the repair is called a fascicular neurosynthesis or epineural repair. When several groups of fascicles are joined, the repair is termed a grouped-fascicular neurosynthesis. A nerve may be fixed by a direct nerve repair, which is also called an end-to-end, abutment, or coaptation neurosynthesis. The term "nerve reconstruction" is used more commonly when referring to the use of a conduit or nerve graft.

When a nerve has a segment of neural tissue missing, there is a nerve defect. However, simple transection of a nerve can result in a gap between the two ends, even if no neural tissue is missing. The difference between a 1 cm gap and a 1 cm defect is that the repair of a gap is not usually under any excess tension. In addition, in a gap, the fascicular anatomy is mirrored at the proximal and distal faces. In the case of a nerve defect, increased tension is required to bring the nerve ends together, occasionally making end-to-end repair impossible. Furthermore, because of the ever changing internal topography of the nerve trunks, axons at the proximal and distal faces of the nerve are usually arranged differently. This can have an adverse effect on regeneration.

Principles of Nerve Repair

We know that once a nerve has been divided it cannot be repaired in such a manner that sensory and motor end organ function will be restored to the pre-injury state [25].

The basic principles of nerve repair include the use of meticulous microsurgical techniques using adequate magnification, instruments, and sutures. Ideally a primary nerve repair is performed in a tension-free manner. To facilitate repair the injured ends of the nerve may be mobilized (or, in the case of the ulnar nerve at the elbow, transposed) to obtain length. Peripheral nerves intrinsically afford a limited degree of excursion, on account of the nerve fibres following an undulating or zig-zag course at a microscopic level. This property gives peripheral nerves a banded appearance, known as the bands of Fontana, which disappear when the nerve is compressed or stretched. If a tension-free repair cannot be achieved, an interposition nerve graft with the limb held in a neutral position is preferred to a primary repair with the limb in an extreme position for a prolonged period of time.

Clinical studies have not shown fascicular repair to be superior to epineural repair. If the internal topography of the nerve is known to be segregated into discrete motor/sensory groups, as is more likely the closer to a branch of the nerve one travels (as the fascicles become designated to end organ territories), a grouped fascicular repair should be better than an epineural repair. If there is not such an arrangement, then the additional

manipulation and increased suture material may hinder functional recovery. For this reason epineural repair is standard.

Bleeding from epineural vessels should be controlled with gentle pressure or fine bipolar coagulation under microscopic guidance. After transection of a nerve the individual fascicles tend to mushroom out from the epineural sheath because of the endoneurial fluid pressure. At the time of epineural repair fascicles may not lie smoothly, causing a misdirection of the regenerating fibers. Appropriate trimming of the fascicles allows them to lie end to end within the epineural sheath. The epineural sutures should be placed so as not to cause additional bunching of fascicles so the nerve can be realigned appropriately.

Postoperative motor and sensory re-education maximizes the surgical result.

Timing of Nerve Repair

Optimum results are obtained after immediate repair of a sharply divided nerve. Fascicular patterning and vascular landmarks are present to guide the proper orientation of the nerve ends. Retraction and neuroma formation, which may result in the need for grafting, are avoided. Within the first 72 h following nerve transection, motor nerves in the distal nerve segment still respond to direct electrical stimulation because of the presence of residual neurotransmitters within the nerve terminals.

When a nerve is injured by crush, avulsion or blast, the surgeon must consider nerve injury proximal and distal to the site of transection. In the acute setting the extent of injury may be difficult to determine even using the operating microscope. In this situation, the nerve ends should be tacked together to prevent retraction and repair delayed until the local area permits. At re-exploration the extent of injury will be defined by neuroma and scar formation. The neuroma must be excised until a healthy fascicular pattern is seen proximally and distally and the resultant defect usually requires nerve grafting.

Clinical Pearl

If a repair without tension cannot be achieved, an interposition nerve graft with the limb in a neutral position is preferred to a primary repair with the limb held in an extreme position for a prolonged period of time.

Clinical studies have shown no difference in outcome between a fascicular or epineural repair.

Surgical Technique for Nerve Repair

General Considerations

Surgical nerve repair may be classified as immediate or delayed based on the time between injury and repair. An immediate repair is one that is performed within the first 24 h after injury. A primary repair is one that is performed within the first week after injury. A delayed primary repair is performed after 1 week following the original injury.

In most instances, transected nerves should be repaired at the time of exploration. An alternative, however, particularly if there is inadequate surgical expertise or equipment, or where the patient condition precludes a long surgical procedure, it is appropriate to simply tag the nerve ends with a marker suture, returning later to do a formal nerve repair. Nerve repair may also be delayed in cases of gross contamination, or where a nerve graft is required and soft tissue cover is inadequate.

Delayed exploration and repair of a nerve may be indicated where a nerve is injured by traction or crush. In these cases, surgical exploration may reveal the nerve to be in continuity. In such circumstances (especially with the common peroneal nerve at the knee) it may be apparent that there is a considerable redundancy in the stretched nerve, although it is still in continuity. In such instances, the redundant nerve should be excised back to healthy nerve ends and a graft used to reconstruct the defect.

Such clinical findings are most easily evaluated in the first few days following trauma,

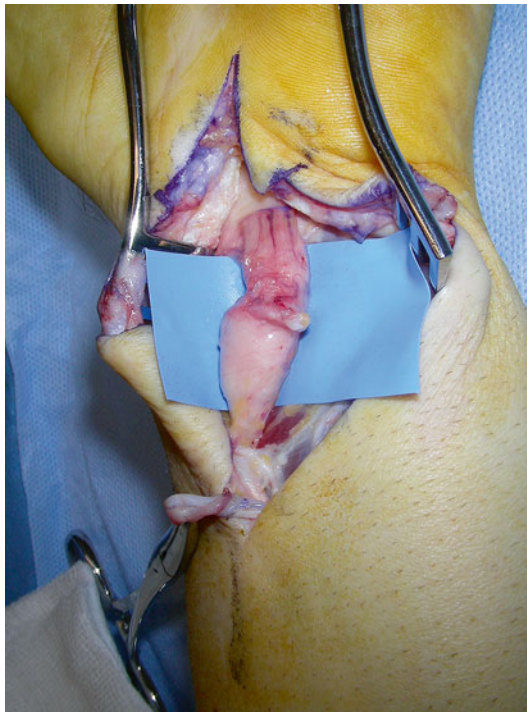


Fig. 2.3 "Mushrooming" seen at the transected end of the median nerve in the forearm after excision of a neuroma (Image kindly provided by Norbert Kang (Royal Free Hospital))

although even then doubt can exist and a "wait and see" policy adopted. If the wound is closed over a lesion in continuity, meticulous attention to the anticipated signs of recovery is needed, and early re-operation is indicated if this recovery fails to appear.

Prior to immediate or primary repair of a peripheral nerve, the cut ends of the nerve should be trimmed, under magnification, until axonal mushrooming is seen (Fig. 2.3). In delayed or secondary repair, the nerve should be trimmed so that there is no intra-fascicular scarring present and fresh, viable nerve fascicles are visible. Some surgeons employ frozen section microscopy to decide the extent of resection, although this requires a skilled specialist histopathologist to be present during the operation. If this preparatory procedure results in a nerve defect then strategies to reconstruct the gap should be employed.

Suitable anaesthesia and (usually) a pneumatic tourniquet are used as appropriate.

Epineural Repair

The aim of nerve repair is to direct regenerating axons in such a way as to allow sensory and motor axons make optimal end organ connections. The technique most commonly used is end to end epineural repair.

Longitudinal vessels on the extrinsic epineurium may be used as landmarks to align fascicles. Along with the varying cross-sectional areas of fascicles, these landmarks can be used to line up the nerve prior to repair. The first suture will provide rotational realignment and the stitch perforates only the epineurium. Most neurosyntheses are performed with a 9-0 (or smaller) non-absorbable suture. The epineurium should be closed loosely. As a guide, if the nerve ends cannot be held in approximation with a single 8.0 suture, then the nerve is under too much tension for a satisfactory repair.

Subsequent sutures are placed to align other anatomic landmarks, each time ensuring that the internal fascicular alignment is correct. The sutures should not violate the fascicles, and the nerve should not gap. It is worth remembering that this technique provides a physiologic tube for nerve regeneration, although the axons themselves are not mechanically reattached. Of the available techniques this is the simplest and quickest, and is most applicable to pure motor or sensory nerves or nerve repairs with indeterminate internal topography.

One suture is usually required for each peripheral fascicle, and the closure should be loose, rather than watertight, to avoid the development of a haematoma. The use of tissue glue around the neurosynthesis is a useful adjunct to prevent movement around the repair, which may otherwise dislodge or disorientate the aligned fascicles (Fig. 2.4).

Fascicular Repair

A Group of fascicles may on occasion be repaired in a manner similar to an epineural repair. The external epineurium is incised, while protecting the internal epineurium and perineurium. The

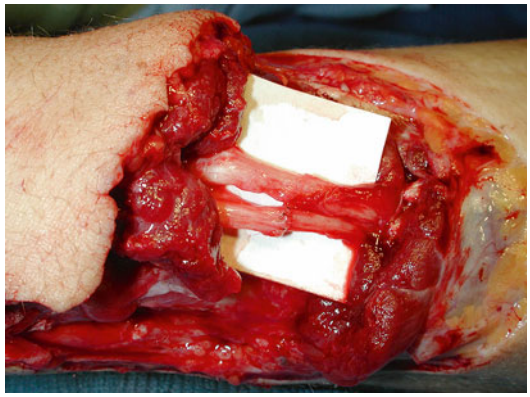


Fig. 2.4 Direct neurosynthesis performed in divided ulnar nerve and dorsal sensory branch of the ulnar nerve in the forearm (Image kindly provided by Norbert Kang (Mount Vernon Hospital))

largest identifiable group is repaired first, using two or three 9.0 or 10.0 non-absorbable sutures for each group.

This technique is most appropriate for repairing nerves with easily identifiable fascicles, such as the common digital nerve in the palm or the ulnar and median nerves at the wrist. It requires considerable effort and manipulation of the nerve compared to an epineural repair. It is especially important that this technique is not performed under tension because tension at this level is transmitted directly to individual fascicles, thus leading to ischemia at the repair site. For this reason, further sutures may be placed in the epineurium to take the tension away from the internal epineurium.

Single fascicles can also be repaired, but indications are limited. The external and internal epineurium must be incised, while the perineurium is preserved. The suture material should be 10-0 or 11-0 material, with few sutures per fascicle. This type of repair is usually only indicated in partially transected nerves.

Many *in vitro* and *in vivo* studies have compared whether fascicular repairs or epineural repairs result in better outcomes [26–28]. To date, evidence is insufficient to suggest one technique is superior to the other. If it were possible to align the fascicles perfectly with minimum surgical disruption, it is theoretically likely that fascicular repair would produce better clinical results than an epineural repair. However as fascicular repairs almost certainly (but unintentionally) result in misalignment of some fascicles, and cause additional intraneural damage to an already traumatised nerve, this theoretical improvement is not seen. Despite an epineural repair being inexact, it does allow neurotropic factors to exert their influence on the direction of nerve fiber growth and this is probably why this less exact technique produces the same clinical results as fascicular repair.

Results of End to End Neurosynthesis

The results of end to end neurosynthesis of acute nerve transection varies widely from patient to patient. Motor and sensory return have been found to correlate closely with four factors:

- Age (the single most important factor): young patients have better results than older patients.
- Level of transection: the more distal the transection, the more functional return can be expected. This is due to the shorter time to reinnervate end organs, and the more discrete fascicular anatomy leading to better fascicular alignment.
- Mechanism of injury: sharp transection has a better outcome than crush, traction or avulsion injuries. Blunt injuries have a longer section of injured nerve so precise axonal matching is more difficult.
- Pre-surgical delay to exploration.

Usually, none of these factors is within the control of the surgeon. Sensory end organs may be reinnervated several years after injury but motor end plates appear to become refractory to reinnervation after approximately 15–18 months. Since axons only regenerate at a rate of 1.0–1.5 mm per day, in brachial plexus injuries, even where primary repair or grafting is carried out promptly, there is such a delay before reinnervation reaches the hand, that good motor recovery of the intrinsic muscles rarely occurs.

Post-operative Care and Complications

Postoperative care is usually straightforward. The soft tissue envelope is closed and the limb is

splinted in a manner that is appropriate for the nature of the surgery. Because the neurosynthesis is not under tension, prolonged protection of range of motion is not necessary for the nerve itself, but may be indicated following repair of other injured structures. For isolated nerve injuries, movement should be started within 3 weeks of surgery to prevent joint contractures and to promote sliding of the nerve relative to the surrounding tissue.

In patients with a nerve injury that results in a motor deficit, the limb should be splinted in a safe position. Concurrently the strength of the functioning muscle is maintained with an exercise program, which also helps to prevent contracture of the denervated muscle.

Oral antibiotics may be given, depending on the amount of wound contamination. Unlike bone or cartilage, nerve is well vascularised and so is not at a greater risk of infection than other soft tissues. Pain control is important, as decreased pain leads to decreased inflammation and swelling. The patient should take enough oral analgesia to eliminate sharp or burning pain and to permit participation in postoperative therapy.

The most common complications of nerve repair or reconstruction are failure to achieve the desired functional outcome, painful neuroma in continuity, or both. Some patients present with adequate distal nerve recovery, but complain of having painful scars. This may be due to scarring between the nerve and the skin (or surrounding tissues), which produces traction. Scar massage and desensitisation therapy are indicated in these patients.

Occasionally nerve repair or reconstruction may result in complex regional pain syndrome. The detailed treatment of this disorder is beyond scope of this chapter, but early recognition and intervention are essential. Hand therapy, TENS, medications including corticosteroids, and regional indwelling anaesthesia all have a role to play.

Clinical Pearl

A blunt or traction injury and delay to surgery all have negative effects on the clinical outcome.

Complex Injury

Traction Injury

Peripheral nerves may be stretched approximately 10 % without losing function and approximately 15–20 % with temporary loss of function (neurapraxia) [29, 30]. Stretch of greater than 20 % results in the elastic limits of the perineurium being exceeded and therefore creates at least an axonotmesis and sometimes a neurotmesis or complete rupture.

The most common causes of traction injuries in the UK are motor vehicle collisions, sports and birth related trauma. Such injuries may recover over a period of months if a neurapraxia has been sustained, or may result in permanent loss of function if there has been extensive disruption of axons with secondary intraneural fibrosis.

The diagnosis of traction injury requires clinical suspicion based on the mechanism of injury and knowledge of other injuries sustained. If the injury has resulted in an open wound, the relevant nerves should be explored at the same time as any vascular/bony/soft tissue repair. If the wound is closed, immediate surgical exploration is not usually carried out solely for the purpose of evaluation of the nerve. With a closed wound diagnosis is based on repeated physical examinations, and electrophysiology studies.

Classification

The classification of nerve injuries was first described by Seddon in 1947 (three classes of injury) and expanded by Sunderland in 1951 (five classes of injury). This classification system remained unchallenged until Mackinnon added a sixth category representing a mixed injury pattern.

- *First-degree injury* (*neurapraxia*) – a localised conduction block in a nerve segment that remains structurally in tact, with normal conduction proximal and distal to the segment, and trophic activity maintained. Axons are not

injured therefore regeneration is not required and complete recovery should occur.

- *Second-degree injury* (*axonotmesis*) – axonal disruption in which the distal segment undergoes Wallerian degeneration. By definition connective tissue layers are uninjured. Recovery is complete unless the distance of the injury from the motor endplate results in such prolonged denervation of the receptor muscle that motor recovery is adversely affected.
- *Third-degree injury* – Wallerian degeneration is combined with endoneurial fibrosis. Recovery is incomplete because scar within the endoneurium may block axonal regeneration or cause mismatching of regenerating fibers with the appropriate end organs.
- *Fourth-degree injury* – the nerve is in continuity, but only the epineurium remains in continuity. Thus a complete scar block (neuroma in continuity) means regeneration will not occur unless the block is removed and the nerve is repaired or grafted.
- *Fifth-degree injury* (*neurotmesis*) – nerve is completely divided and must be repaired before any regeneration can occur.
- *Sixth-degree injury* – a combination of any of the previous five levels of injury. Because of the longitudinal nature of crush injuries, different levels of nerve injury can be seen at various locations along an injured nerve. This is the most challenging nerve injury for the surgeon, as some fascicles will need to be protected and not "downgraded," whereas others will require surgical reconstruction.

The degree of injury is important in determining treatment. First, second, and third degree injuries have the potential for recovery and generally do not require surgical intervention. A first degree injury recovers complete function within 3 months. A second degree injury recovers completely but slowly (somewhat less than 1 mm per day from proximal to distal), whereas recovery after third degree injuries is slow and incomplete. Fourth and fifth degree injuries will not recover without surgical intervention. A sixth degree injury shows a variable recovery.

Neurapraxia

Neurapraxia is a localised conduction block in a nerve segment that remains structurally in tact, with normal conduction proximal and distal to the segment, and trophic activity maintained. Axons are not injured, regeneration is not required and complete recovery should occur. It is therefore very important to be able to recognize neurapraxia, as surgery is not indicated in such cases.

Factors within the history and examination suggest when a neurapraxia has occurred:

- History of minor or low energy trauma, or prolonger low pressure crush
- Often not all function modalities are lost (motor and proprioception is lost preferentially, while vibration, pain and hot versus cold discrimination are often preserved)
- No Tinel's sign on examination, and no muscular fibrillation
- No trophic changes are seen, and minimal muscle atrophy occurs
- Sensory recovery occurs ahead of motor recovery (sensorimotor dissociation)
- Sudden, sporadic, non-sequential recovery of motor function occurs

If a neurapraxia is suspected, then regular and repeated examinations should be undertaken to monitor recovery and confirm the diagnosis. EMG studies may also be of value to demonstrate preservation of distal action potentials which lends weight to the diagnosis.

Gun Shot Wound

Gunshot wounds are severe, high energy and unique. They are capable of dividing a nerve (grade V injury) but most often their damage is due to blast effect (grades I-IV injury). The former requires exploration and direct repair, the latter should be observed and managed as a traction injury.

In general it is unlikely that a gunshot wound would cause a transection of a major peripheral nerve without damaging the nearby artery. However, as almost all gunshot wounds (even

without major vascular injury) require debridement, then exploration of the peripheral nerves is usually indicated. A delayed primary repair of a complete or partial nerve laceration caused by a bullet is easier than management of a neuroma in continuity, and this can safely be undertaken when the wound is stable.

Management of a Nerve gap

Principles of Nerve Grafting

When end to end neurosynthesis cannot be carried out without tension, a nerve graft (or other solution) is necessary, as even minimum tension on the nerve coaptation may compromise the final result. Repairing a peripheral nerve with the extremity flexed to approximate the two nerve ends is inappropriate, as ultimately the nerve must glide as the extremity moves not only to a neutral position, but also into full extension.

There remains contention as to the critical length of nerve gap that necessitates a nerve graft. In the digit, a 1 cm gap in a digital nerve cannot be overcome. In the arm and forearm Millesi [31] supports mobilisation and end to end repair for defects of 6 cm or less, if they are easily approximated without flexion of the adjacent joints. Our experience is that a 6 cm nerve gap would be difficult to overcome without nerve graft, with the exception of the ulnar nerve where anterior transposition gains some length.

Placement of the nerve graft into a healthy bed is important for graft survival. The grafted nerve, much like a skin graft, requires ingrowth of blood vessels from the surrounding tissue to survive. Thin cutaneous nerve grafts are more easily vascularised than trunk or cable grafts [32]. If neovascularisation of the nerve graft has not occurred by the third day, the special components of the nerve, such as the endoneurial Schwann cells, involute and are replaced by fibrous tissue [32].

Nerve grafts heal by ingrowth of new vessels, and therefore require a vascularised bed capable of angioneogenesis (not seen in post irradiation tissue). They also require no barriers to ingrowth and inosculation such as infection, haematoma, fat on the nerve graft or metalwork. The nerve graft should be under no tension, repaired with sutures and minimal fibrin glue, and have a maximal surface area to volume ratio. Surgical technique is crucial in that nerve grafts should not be clumped together to form a trunk. All fat should be removed from the graft, haemostasis should be obtained and antibiotics used to minimize infection. Generally, a slightly longer graft is preferred to avoid tension. On occasion, a vascularised would bed may need to be imported (with a muscle flap), or a dry wound bed created with the use of a silicone spacer, the latter left in situ for a few weeks before grafting.

One of the technical challenges of nerve grafting is maintaining the proper fascicular alignment between the proximal and distal nerve ends. Often the internal topography of nerve will change as it traverses a gap, or the proximal stump will contain a different number of fascicles to the distal stump. When nerves contain mixed sensory and motor fascicles the problem is compounded. Correct orientation is assisted by knowledge of the internal anatomy of the nerve, distal dissection, and careful inspection of the cut ends of the nerve. When resecting a nerve associated with a tumour, if the nerve is functioning before resection then individual fascicle stimulation and marking may help orientation of the graft during reconstruction.

A further challenge is to maximize the number of axons crossing a nerve graft through both the proximal and distal neurosynthesis. Reversal of the direction of the nerve graft reduces leakage of axons through side branches, and effectively funnels regenerating axons distally. Making use of the maximum possible number of cables of nerve graft across a gap also increases the number of axons which may potentially regenerate (Fig. 2.5).

Clinical Pearl

The key to success of a nerve graft is surgical precision. Any graft should be under little or no tension and sit in a good vascular bed. It is also important that the fascicles are aligned as accurately as possible.

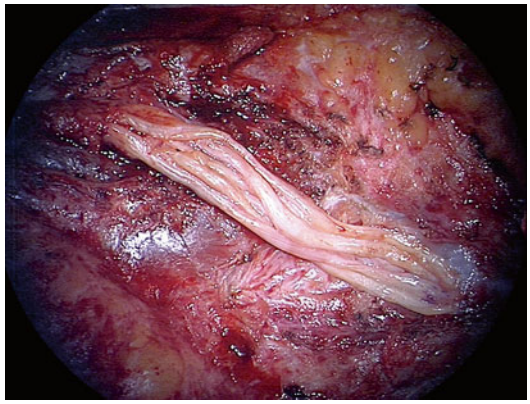


Fig. 2.5 Multiple cables of sural nerve graft crossing a 7 cm nerve gap in the common peroneal nerve, following resection of injured nerve after posterior knee dislocation

Donor Nerves

The sural nerve in adults can provide 30–40 cm of graft, with 11–12 fascicles being reliably present [33]. In 80 % of dissections it is formed by the medial sural cutaneous nerve and the peroneal communicating branch. The communicating branch can contribute an additional 10–20 cm if required. The nerve is adjacent to the lesser saphenous vein posterior to the lateral malleolus, and is usually harvested through a longitudinal incision (so as to avoid compromising the quality of the nerve graft), though some surgeons prefer multiple transverse incisions, or to harvest the nerve endoscopically. The consequent area of numbness on the lateral foot is well tolerated. The disadvantages of this donor site are the separate distal donor site, and the relatively high proportion of connective tissue to axons when compared to upper limb nerves.

When a small amount of nerve graft is required, the medial or lateral antebrachial cutaneous nerves may be harvested from an injured upper limb. The lateral antebrachial cutaneous nerve (LACN) lies adjacent to the cephalic vein along the ulnar border of brachioradialis, and 8 cm of graft can be harvested in adults. The medial antebrachial cutaneous nerve (MACN) lies in the groove between triceps and biceps alongside the basilic vein, and has an anterior and posterior division. Twenty centimeter of nerve graft may be harvested if both divisions are taken, although sensibility is lost over the elbow and resting part of the forearm if the posterior division is harvested.

For short grafts, to reconstruct digital nerve defects for instance, the posterior interosseus nerve may be used. It is most accessible where it lies in the radial part of the fourth dorsal extensor compartment at the wrist, and a short incision allows easy harvest of a 2.5 cm graft. Loss of wrist joint sensibility and proprioception is well tolerated if the anterior interosseus nerve is left undisturbed.

Surgical Technique of Nerve Grafting

The surgical technique of nerve grafting that was developed by Millesi [34–36] is similar in many ways to primary nerve repair. The surgeon prepares the proximal and distal ends of the nerve by evaluating and excising the damaged or scarred nerve. The harvested nerve is then placed across the gap between proximal and distal stumps without tension. The involved extremity is moved through a passive range of motion before setting the length of the graft. One end of the graft is sutured, the graft is positioned in the gap, and the other end trimmed to fit the defect. Two or three fine sutures are all that are required to secure each graft to the nerve stumps. It is our practice to reinforce the neurosynthesis with fibrin glue. Major nerves may require several cables of nerve graft, while a digital nerve will only require a single cable in most instances.

When insetting the nerve graft, it is important to try to achieve an appropriate sensory and motor fascicle match at the proximal and distal repair sites. Techniques to assist in the orientation of the nerves have been discussed previously. In proximal extremity grafts, the fascicles at the proximal stump are frequently mixed and so the fascicular alignment of the grafts cannot be specific. At the distal nerve stump, the alignment can usually be more specific. Occasionally it may be useful to direct all reinnervation into the restoration of a critical motor function. For example in radial nerve injuries, the proximal level of the graft is frequently a point at which the fascicles are mixed

motor and sensory. Distally, however, the sensory fibers of the superficial radial nerve can be completely excluded from the graft so that all regenerating fibers are directed into the distal motor fascicles and none into the sensory fascicles.

Vascularised Nerve Grafts and Allografts

In conventional nerve grafting, success is inversely proportional to the length of the graft. This is because the longer the graft is, the more likely it is that part of the graft will not revascularise. Vascularised nerve grafts aim to circumvent this problem, particularly where the wound bed is poor or the graft is long.

Potential donor nerves include anterior tibial, saphenous, superficial peroneal, deep peroneal, superficial radial, ulnar, and sural nerves. Although clinical series have proven the feasibility of performing vascularised nerve grafts in humans, a definite clinical role in peripheral nerve repair is yet to be defined. Currently there is little experimental evidence to demonstrate their superiority to justify the additional surgical morbidity and effort.

Rarely, a situation arises where an otherwise salvageable extremity has a peripheral nerve injury that is unreconstructable with autogenous nerve grafts because of insufficient donor sites. Mackinnon and Hudson [37] have reported obtaining protective sensation in the foot by using a 23 cm, ten cable allograft to reconstruct a sciatic nerve and Bain has used this technique in seven patients who needed major nerve reconstruction [38]. Although still experimental, this technique may provide an excellent alternative source of graft material.

Nerve Conduits

A conduit is a tubular structure used to connect the two transected ends of nerve across a gap. The potential role of conduits in the repair and reconstruction of peripheral nerves has interested surgeons for many years. Ever since Büngner reported

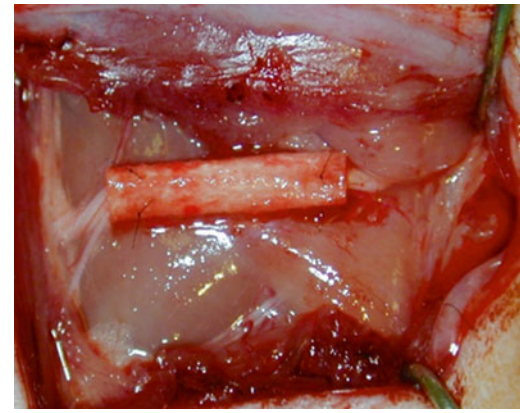


Fig. 2.6 Short segment bio-absorbable nerve conduit placed experimentally in the hind leg of a rat model

successful nerve regeneration through an arterial graft in 1891 [39], we have known that severed nerves can grow across a gap through a conduit.

The theory underpinning conduit repair of peripheral nerves is that the enclosed space allows neurotropic agents released by the distal stump to establish a uniform concentration through the conduit, thereby inducing chemotactic attraction to the proximal sprouting axons, which results in more numerous axonal reconnections.

However, there is a limit to how far a bare axon sprout can grow and various growth factors are required by the growth cone to survive. Some are provided through internal axonal transport, others are obtained from the surrounding millieu. Schwann cells begin to surround the new growth cone and provide metabolic support, and at the same time, fibroblasts begin to lay down collagen to provide structural support.

Nerve regeneration across (short defect) bio-absorbable polyglycogen tubes (Fig. 2.6) has been shown in both animal and human models, and has been comparable to a standard nerve grafts [40]. More recent work on introducing neurotrophic factors to the surface of these conduits has lead to encouraging improvements in the regeneration seen. Micro-engineering of the internal surface of conduits has also allowed the potential length of the conduit to be extended in animal models.

Commercial conduits are available for reconstruction of short defects in small and large diameter nerves, although their uptake is not

widespread. As confidence in the efficacy of these devices grows, and use increases, it may eliminate the morbidity associated with harvesting donor nerves. Currently, however, they are best considered experimental rather than clinically efficacious.

Finally, vein grafts have been used to reconstruct short, distal, sensory nerve defects, but results are not as satisfactory as using conventional nerve grafts [41].

Nerve Transfer

Indications

Nerve transfers involve using nerves with relatively less important roles, to restore function to a more useful nerve that has been injured. The use of nerve transfers has expanded as a consequence of the improved knowledge of the internal topography of peripheral nerves in the limbs.

Nerve transfers are indicated in very proximal peripheral nerve injuries or root avulsions where a proximal stump is unavailable for primary repair or grafting. Even when grafting is possible the injury may be so proximal that a nerve transfer facilitates better re-innervation of distal motor endplates than does a nerve graft. Nerve transfers are also indicated to avoid operating in regions of severe scarring, when nerve injuries present in a delayed fashion, when partial nerve injuries present with a well-defined functional deficit, or when the level of injury is unclear such as in idiopathic neuritides or radiation-induced nerve injury.

Principles

Nerve transfers are based on the same principle as tendon transfers, namely, the sacrifice of a less important function to restore a function of greater importance. An example is the attachment of the superficial branch of the radial nerve to the ulnar most palmar digital nerve to restore sensibility to that side of the hand in a patient who has an unreconstructable ulnar nerve injury.

The characteristics of an ideal donor nerve to transfer for motor function are:

- Expendable donor motor nerve with a large number of pure motor axons.
- Located in close proximity to target motor endplates, thus minimising the distance and time regenerating axons need to travel to re-innervate their target.
- Donor nerve innervates a muscle that is synergistic with its target.

Clinical Application

The most common application of motor nerve transfers is after brachial plexus injuries and includes the restoration of elbow flexion, shoulder abduction, ulnar-innervated intrinsic hand function, forearm pronation, and radial nerve function.

To restore elbow flexion, the medial pectoral, thoracodorsal, or intercostal nerves can be transferred to the musculocutaneous nerve. The flexor carpi ulnaris branch of the ulnar nerve [42] and the flexor carpi radialis branch of the median nerve can also be transferred to the biceps and brachialis branches of the musculocutaneous nerve to more specifically restore elbow flexion and limit donor nerve morbidity.

To restore shoulder abduction, the distal accessory nerve can be transferred to the suprascapular nerve, or the triceps branch of the radial nerve can be transferred to the axillary nerve. To restore intrinsic hand function, the distal anterior interosseous nerve can be transferred to the ulnar nerve. Transferring redundant fascicles of the flexor carpi ulnaris branches of the ulnar nerve to the median nerve-innervated pronator teres can restore forearm pronation. Alternatively, the flexor digitorum superficialis, or palmaris longus branches of the median nerve, can be transferred to its pronator branch. The radial nerve is most commonly reconstructed by transferring a portion of the ulnar nerve supplying flexor carpi ulnaris.

After dissecting out the functional proximal and nonfunctional distal nerves, the surgical

technique is essentially the same as a standard nerve repair. Care must be taken to ensure that, like a tendon transfer, the nerve transfer is surrounded by adequate soft tissue to provide protection from external trauma and is not obstructed or placed under compression by the anatomic structures along its course.

End to Side Neurosynthesis

End to side neurosynthesis is a technique in which the distal end of a transected nerve is coapted to the side of an intact nerve. Axons from the intact nerve theoretically sprout from the interrupted side and grow down the attached nerve to the distal end organ. End to side nerve repair was reported in patients at the turn of the century [43] after experimental work undertaken in the early 1990s [44].

Since then, a great deal of research has been published that supports the idea and refines it for clinical use. It was initially unclear where the "sprouting" axons were originating from, but double-labeling experiments demonstrated that true collateral (nodal) sprouting from the nodes of Ranvier really occurs [45]. Sensory and motor nerves are capable of collateral sprouting, and there appears to be only minimal loss of donor nerve function [46].

The technique involves mobilising the distal end of the transected nerve and aligning it next to an appropriate donor nerve. The donor nerve should be of a synergistic muscle group if it is used for motor reinnervation, or from a neighboring dermatome if it is used to reconstruct a sensory defect. Under magnification, a window that is the size of the transected nerve end is created in the epineurium, and fine sutures are used to secure the coaptation.

End to side neurosynthesis has been used in cases of facial palsy, brachial plexus injury and median, ulnar, and digital nerve injuries, all with mixed results. It appears to be more useful for sensory reconstruction when nerve grafting is not feasible, although more research is needed for this to become a reliable replacement for nerve transfers.

Investigations After Nerve Injury

Investigation

With the exception of injured nerve roots, which maybe investigated by contrast myelography or high resolution MRI, radiologic imaging is of little practical value in the diagnosis of a peripheral nerve injury. Ultrasound may be useful in a few cases to establish nerve continuity or the presence of a neuroma, although these instances are rare.

Electrophysiological diagnosis has many pitfalls. Its major limitation is that even a completely divided motor nerve may have normal findings for the first 3 weeks after the injury.

In the case of suspected peripheral motor nerve injury, serial electromyography (EMG) can demonstrate whether a muscle is innervated and beginning to undergo progressive reinnervation (nascent or polyphasic potentials) or remains denervated (spontaneous spike potentials at rest and denervation potentials). This may be useful to guide whether surgical exploration of the nerve is indicated. Measurement at 4 weeks post injury is the first occasion where the information becomes useful, and combined with clinical assessment a diagnosis of neurapraxia may be tentatively made (Fig. 2.7).

In certain circumstances, somatosensory evoked potential measurements may be a useful clinical test in the unconscious patient, as somatosensory evoked potentials can determine the integrity of the neural pathways from the fingertip to the postcentralgyrus.

Exploration

If there is still clinical doubt after a thorough history and physical examination, the diagnosis of a potential nerve injury can best be made by surgical exploration of the wound.

Summary

Nerve repair, transfer and grafting have benefited from the development of microsurgical techniques and advances in the field of

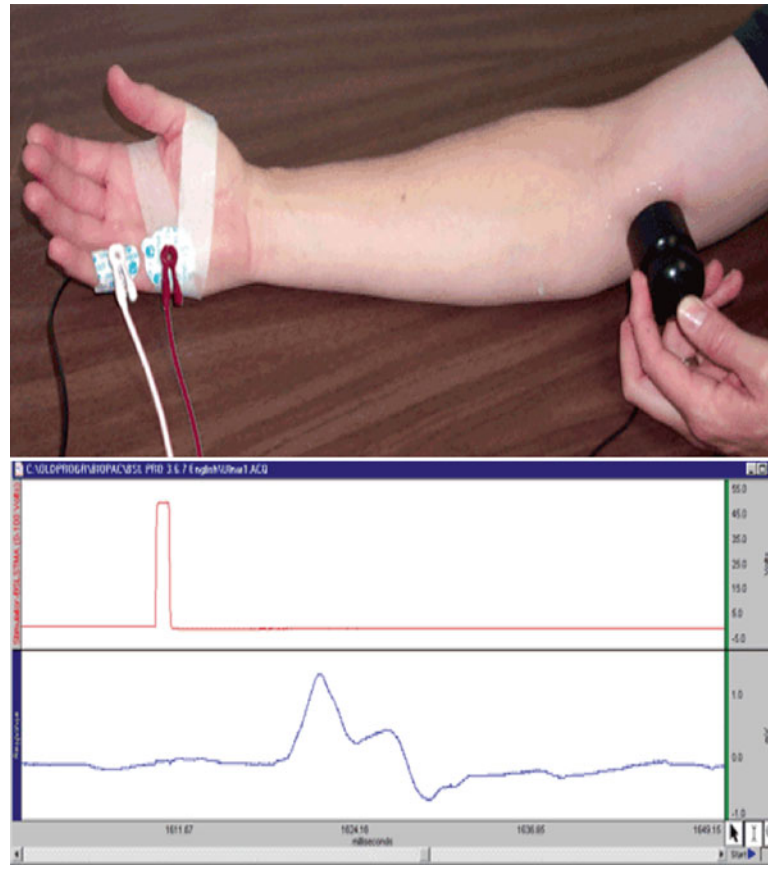


Fig. 2.7 Electromyography 4 weeks after injury may assist in decision making when combined with serial clinical assessment

neuroscience. Optimum outcomes from nerve repair not only demand precise surgical techniques, but also additional measures to direct nerve regeneration to its original function.

Although nerve grafting remains the gold standard for reconstruction of a nerve gap, synthetic conduits now play a limited but important role in the peripheral nerve surgeon's armamentarium, and this role is likely to increase over the coming decades.

References

1. Elsberg C. The Edwin Smith surgical papyrus. Ann Med Hist. 1931;3:271–9.
2. Millesi H. Reappraisal of nerve repair. Surg Clin North Am. 1981;61:321–40.
3. Terzis J, Faibisoff B, Williams B. The nerve gap: suture under tension vs. graft. Plast Reconstr Surg. 1975;56:166–70.
4. Noble J, Munro CA, Prasad VS, Midha R. Analysis of upper and lower extremity peripheral nerve injuries in a population of patients with multiple injuries. J Trauma. 1998;45:116–22.
5. Dellon AL, Mackinnon SE. Basic scientific and clinical applications of peripheral nerve regeneration. Surg Annu. 1988;20:59–100.
6. Jabaley ME, Wallace WH, Heckler FR. Internal topography of major nerves of the forearm and hand: a current view. J Hand Surg. 1980;5:1–18.
7. Williams HB, Jabaley ME. The importance of internal anatomy of the peripheral nerves to nerve repair in the forearm and hand. Hand Clin. 1986;2:689–707.
8. Mackinnon SE, Dellon AL. Experimental study of chronic nerve compression. Clinical implications. Hand Clin. 1986;2:639–50.
9. Sunderland S, Bradley KC. The cross-sectional area of peripheral nerve trunks devoted to nerve fibers. Brain. 1949;72:428–49.
10. Millesi H. The nerve gap. Theory and clinical practice. Hand Clin. 1986;2:651–63.
11. Terenghi G, Calder JS, Birch R, Hall SM. A morphological study of Schwann cells and axonal regeneration in chronically transected human peripheral nerves. J Hand Surg. 1998;23:583–7.

12. Giannini C, Dyck PJ. The fate of Schwann cell basement membranes in permanently transected nerves. J Neuropathol Exp Neurol. 1990;49:550–63.
13. Li H, Terenghi G, Hall SM. Effects of delayed reinnervation on the expression of c-erbB receptors by chronically denervated rat Schwann cells in vivo. Glia. 1997;20:333–47.
14. Hems TEJ, Glasby MA. The limit of graft length in the experimental use of muscle grafts for nerve repair. J Hand Surg Br. 1993;18:165–70.
15. Calder JS, Norris RW. Repair of mixed peripheral nerves using muscle autografts: a preliminary communication. Br J Plast Surg. 1993;46:557–64.
16. Ma J, Novikov LN, Wiberg M, Kellerth JO. Delayed loss of spinal motoneurons after peripheral nerve injury in adult rats: a quantitative morphological study. Exp Brain Res. 2001;139:216–23.
17. Fu SY, Gordon T. The cellular and molecular basis of peripheral nerve regeneration. Mol Neurobiol. 1997;14:67–116.
18. McKay Hart A, Brannstrom T, Wiberg M, Terenghi G. Primary sensory neurons and satellite cells after peripheral axotomy in the adult rat: timecourse of cell death and elimination. Exp Brain Res. 2002;142:308–18.
19. Gu Y, Spasic Z, Wu W. The effects of remaining axons on motoneuron survival and NOS expression following axotomy in the adult rat. Dev Neurosci. 1997;19:255–9.
20. Novikov L, Novikova L, Kellerth JO. Brain-derived neurotrophic factor promotes axonal regeneration and long-term survival of adult rat spinal motoneurons in vivo. Neuroscience. 1997;79:765–74.
21. Hart AM, Terenghi G, Kellerth JO, Wiberg M. Sensory neuroprotection, mitochondrial preservation, and therapeutic potential of N-acetyl-cysteine after nerve injury. Neuroscience. 2004;125:91–101.
22. Zhang CG, et al. Motorneuron protection by N-acetylcysteine after ventral root avulsion and ventral rhizotomy. Br J Plast Surg. 2005;58:765–73.
23. Strauch B, et al. The ten test. Plast Reconstr Surg. 1997;99:1074–8.
24. Cales L, Weber RA. Effect of water temperature on skin wrinkling. J Hand Surg. 1997;22:747–9.
25. Lundborg G, Dahlin L, Danielsen N, Zhao Q. Trophism, tropism, and specificity in nerve regeneration. J Reconstr Microsurg. 1994;10:345–54.
26. Grabb WC, Bement SL, Koepke GH, Green RA. Comparison of methods of peripheral nerve suturing in monkeys. Plast Reconstr Surg. 1970;46:31–8.
27. Bora Jr FW, Pleasure DE, Didizian NA. A study of nerve regeneration and neuroma formation after nerve suture by various techniques. J Hand Surg. 1976;1:138–43.
28. Cabaud HE, Rodkey WG, McCarroll Jr HR, Mutz SB, Niebauer JJ. Epineurial and perineurial fascicular nerve repairs: a critical comparison. J Hand Surg. 1976;1:131–7.
29. Lundborg G, Rydevik B. Effects of stretching the tibial nerve of the rabbit. A preliminary study of the intraneural circulation and the barrier function of the perineurium. J Bone Joint Surg. 1973;55:390–401.
30. Haftek J. Stretch injury of peripheral nerve. Acute effects of stretching on rabbit nerve. J Bone Joint Surg. 1970;52:354–65.
31. Millesi H. Peripheral nerve injuries. Nerve sutures and nerve grafting. Scand J Plast Reconstr Surg Suppl. 1982;19:25–37.
32. Millesi H. Techniques for nerve grafting. Hand Clin. 2000;16:73–91, viii.
33. Brammer JP, Epker BN. Anatomic-histologic survey of the sural nerve: implications for inferior alveolar nerve grafting. J Oral Maxillofac Surg. 1988;46:111–7.
34. Millesi H, Meissl G, Berger A. Further experience with interfascicular grafting of the median, ulnar, and radial nerves. J Bone Joint Surg Am. 1976;58:209–18.
35. Millesi H, Meissl G, Berger A. The interfascicular nerve-grafting of the median and ulnar nerves. J Bone Joint Surg Am. 1972;54:727–50.
36. Millesi H. Nerve grafting. Clin Plast Surg. 1984;11:105–13.
37. Mackinnon SE, Hudson AR. Clinical application of peripheral nerve transplantation. Plast Reconstr Surg. 1992;90:695–9.
38. Bain JR. Peripheral nerve and neuromuscular allotransplantation: current status. Microsurgery. 2000;20:384–8.
39. Bungner O. Die degenerations-und regenerationvorgange am nerven verletzungen. Beitr Pathol Anal. 1891;10:321–93.
40. Lundborg G, Rosen B, Dahlin L, Danielsen N, Holmberg J. Tubular versus conventional repair of median and ulnar nerves in the human forearm: early results from a prospective, randomized, clinical study. J Hand Surg. 1997;22:99–106. doi:10.1016/S0363-5023(05)80188-1.
41. Chiu DT, Janecka I, Krizek TJ, Wolff M, Lovelace RE. Autogenous vein graft as a conduit for nerve regeneration. Surgery. 1982;91:226–33.
42. Oberlin C, et al. Nerve transfer to biceps muscle using a part of ulnar nerve for C5-C6 avulsion of the brachial plexus: anatomical study and report of four cases. J Hand Surg. 1994;19:232–7. doi:10.1016/0363-5023(94)90011-6.
43. Al-Qattan MM. Terminolateral neurorrhaphy: review of experimental and clinical studies. J Reconstr Microsurg. 2001;17:99–108.
44. Viterbo F, Trindade JC, Hoshino K, Mazzoni Neto A. End-to-side neurorrhaphy with removal of the epineurial sheath: an experimental study in rats. Plast Reconstr Surg. 1994;94:1038–47.
45. Zhang Z, Soucacos PN, Bo J, Beris AE. Evaluation of collateral sprouting after end-to-side nerve coaptation using a fluorescent double-labeling technique. Microsurgery. 1999;19:281–6.
46. Zhang Z, et al. Long-term evaluation of rat peripheral nerve repair with end-to-side neurorrhaphy. J Reconstr Microsurg.2000;16:303–11.doi:10.1055/s-2000-7338.

Injury: Tendons – Flexor

3

David Elliot

Keywords
Tendon • Flexor Tendon • Flexor Tendon Primary Repair • Flexor Tendon Rehabilitation • Flexor Tendon Rupture • Flexor Tendon Adhesion

Introduction

We are all guilty of complacency in our belief that all is well with flexor tendon surgery and that our knowledge of this subject is complete. Like many other parts of hand surgery, when one looks a little more closely, one discovers that much that seemed fully understood is far from understood and what we have assumed to be based on hard fact often rests on opinion. Although, currently, there is debate about many details of technique, the central tenet of modern flexor tendon surgery is to repair and move divided flexor tendons within a few days of injury.

Repair of the divided flexor tendon to achieve normal, or near normal, function is a problem which we have not yet solved and primary flexor tendon surgery remains difficult, with each result still being uncertain. Over and above the actual technical difficulties of repairing tendons, we face the complications of rupture and adherence of repairs during healing and these two problems have dominated thought on primary flexor tendon surgery for a century. Healing the flexor tendon takes about 3 months and, for much of that time, tendon continuity depends largely on the strength of our sutures. Unfortunately, this period is sometimes longer than that for which the hand can be kept free of activities, or accidents, liable to snap the repair. In any healing area, a glue of fibrin-loaded oedema is formed which later converts to scar tissue. Unfortunately, the body does not limit this healing process to those structures which are injured. Everything in the vicinity becomes involved in the healing process with the result that all the tissues become 'spot-welded' together. Although the body then remodels the scar tissue, this is generally too little and too late to allow a return to normal function for structures, such as those in our hands, which must move to function. The devastation this can cause, not only to the flexors but also to the nerves, extensor tendons and joints of the hand, is the cause of a great deal of the morbidity of hand injury and the source of much of our secondary surgery. This 'spot-welding' by scar adhesions can occur anywhere along the length of a flexor tendon, but is a particular problem in the fingers themselves, where the flexors are confined within the tendon sheath

D. Elliot, MA, FRCS, BM, BCh
St Andrews Centre for Plastic Surgery,
Broomfield Hospital, Chelmsford, UK
e-mail: info@david-elliot.co.uk

I.A. Trail, A.N.M. Fleming (eds.), *Disorders of the Hand: Volume 1: Hand Injuries*,
DOI 10.1007/978-1-4471-6554-5_3, © Springer-Verlag London 2015

in a system as finely bored as the pistons in an engine.

Historical Background

In 1913, Lexer introduced early mobilisation of repaired flexor tendons to avoid the effects of scar adhesions, by mobilising the hand 6 days after flexor graft surgery [1]. In 1917, Harmer adopted this approach following primary flexor tendon surgery in Boston in the United States of America [2]. He introduced a new and special suture which he claimed was strong enough to allow early active motion within hours of primary tendon repair, using no splint. In the same year in Vienna, Kirchmayr described the same, using his new special suture, which we now know as the Kessler suture after its re-inventor 30 years ago [3, 4]. Lahey, writing in 1923, rather cleverly managed to upstage everyone by backdating his experience of early mobilisation of primary flexor tendon repairs to 1907 [5]. Even Bunnell, writing in 1918, agreed that this approach could be taken in selected patients [6]. How successful these early surgeons actually were and how many cases ruptured the repairs is not known because all of these papers describe only a technique of management and none of the authors reported their results. However, in their works we see an earlier realisation of the problems of rupture and adherence which we still face. We also see the beginnings of the 'surgeons' solution, namely stronger sutures and early mobilisation, which remains the predominant direction of thought in this field today.

In 1920's, Bunnell decided that primary flexor tendon surgery was unsafe in most hands. He advised that these injuries should not be repaired as a primary treatment, but that the skin should simply be closed and secondary tendon grafting carried out at some later date when the patient was in the hands of an experienced tendon surgeon. This attitude was to prevail until the 1950s when three groups of surgeons – Kleinert's team in Louisville, Kentucky, Young and Harman, also in the United States, and Verdan in Switzerland – started repairing flexors immediately in zones 1 and 2 again and also moving them very early after repair [7–10]. To do this with what they considered reasonable safety, they introduced protective splinting regimes intended to reduce the force on the repair during early mobilisation. This was the beginning of primary flexor tendon surgery in our era.

An alternative approach to reducing the effect of adhesions on movement of the repaired tendon which has been considered periodically is to try to minimise the formation of adhesions by chemical means. This has been attempted with a variety of drugs, including cytotoxics, hyaluronidase, Adcon and, most recently, Hyaloglide. The last of these drugs has been show to be effective in limited clinical trials [11] but its use has not yet caught on and it remains to be proven whether this, or any other, drug reducing adhesions should be used routinely following flexor tendon repair.

Early mobilisation does not, of course, prevent adhesions entirely, but it does seem to create a form of scarring which allows us to regain much of the range of movement and, sometimes, even return function to normal. Because rupture defeats this aim, there is a need to create sutures and suture techniques strong enough to allow this movement. In our time, surgeons have almost all felt a need also to protect the sutures from the full brunt of normal activity by use of some system of protective splinting. Consequently, research drive over the last 50 years has been largely two-pronged and has been dominated by attempts to modify the suturing techniques and to modify the rehabilitation regimes, with different individuals and different generations of surgeons moving one line of advance, or the other, or, occasionally, both, forwards in fits and starts through the twentieth and early twenty-first centuries.

Clinical Evaluation

Because of the close proximity of the flexor tendons to the skin surface along most of their length, the need for exploration of the palmar/flexor surface of the hand, wrist and

forearm to determine whether there has been injury to the flexor system arises under almost all circumstances in which this surface of the distal part of the upper limb has been cut through the full thickness of the skin to a significant degree.

Fingers, and occasionally the thumb, lying in a position of extension relative to the other digits or to the normal resting hand position may make flexor tendon division(s) obvious. Where pain does not preclude active movement by the patient or passive wrist tenodesis and forearm squeezing tests, these means may also be used to help confirm tendon division. However, a high index of suspicion should be present, even in the absence of positive clinical findings, when the skin has been breached over the flexor tendons.

Clinical Pearls – Suspect open flexor tendon injury if:

Any full-thickness skin breach over flexor surface of finger

Finger lies extended relative to normal cascade

Positive wrist tenodesis test

Positive forearm squeeze test

Closed rupture of the flexor tendons in a healthy hand and forearm, without previous injury, is unusual and largely confined to detachment of the profundus flexor tendons of the ring and little fingers from their insertions onto the distal phalanx. A history of sudden forced extension of the finger while the flexor tendons are active, as occurs in the typical rugby jersey incident, which is the commonest cause of this injury, and clinical testing for profundus function in the involved finger will make this diagnosis. Although radiological investigations, in particular ultrasound, have been used and recommended in the diagnosis of both detachment of the profundus tendon and rupture of flexor tendon repairs, these are used little in the primary diagnosis of open flexor tendon division(s). Diagnosis remains, primarily, clinical.

Surgical Anatomy

It is essential both to evaluation and to treatment of the flexor tendons to understand their anatomy and that of the pulley system of the tendon sheaths (Doyle and Blythe, 1975; Idler, 1985) (Fig. 3.1) [12, 13]. It is also necessary to understand the classification of the digital flexor tendons into the zones described by Verdan and Michon in 1961, and later modified by Kleinert and Weiland (1976), as this is the basis of discussion of current treatments (Fig. 3.2) [14, 15]. Zone 1 is that part of the finger profundus flexor tendon between its insertion into the distal phalanx and the distal edge of the insertion of the FDS tendon into the middle phalanx. Zone 2 is that part of the contents of the flexor sheath from the distal edge of the FDS insertion to the proximal edge of the Al pulley. Zone 3 is that part between the proximal edge of the Al pulley and the distal edge of the carpal tunnel and Zone 4 is the part within the carpal tunnel. Zone 5 is that part proximal to the carpal tunnel. In the thumb, the zones are less specifically defined. Zone 1 is that part of the FPL tendon from its insertion to the proximal edge of the A2 pulley and Zone 2 is that part between the proximal edge of the A2 pulley and the proximal edge of the A1 pulley. Zone 3 is that part in which the tendon passes through the thenar eminence. The proximal zones (4–6) correspond to those of the fingers.

More recently, Tang has divided zone 2 into four sub-zones (Fig. 3.3) [16]: zone 2A is the

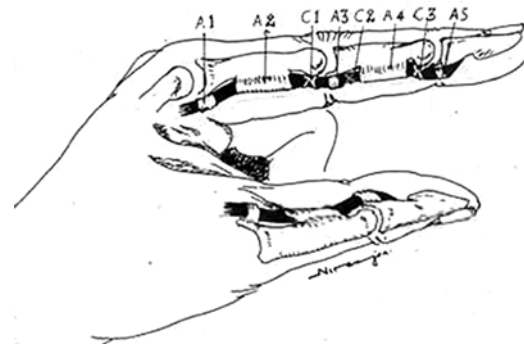


Fig. 3.1 The flexor pulley system (Picture courtesy of Mr N Nirinjan FRCS, Consultant Plastic Surgeon)

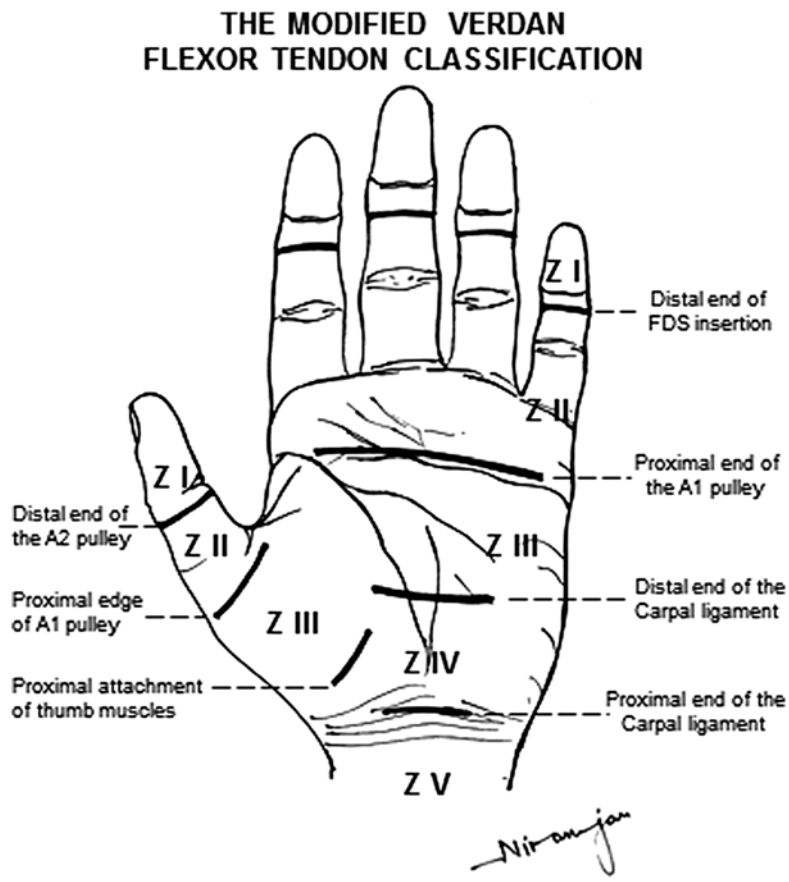


Fig. 3.2 The modified Verdan flexor tendon classification (Picture courtesy of Mr N Nirinjan FRCS, Consultant Plastic Surgeon)

insertion of the FDS tendon into the middle phalanx, Zone 2B includes those parts of the tendons between the proximal edge of the FDS insertion and the distal edge of the A2 pulley, Zone 2C includes those parts of the tendons under the A2 pulley and Zone 2D those parts proximal to the A2 pulley but still within the flexor sheath. The author and his colleagues have also subdivided zone 1 injuries into three subdivisions (Fig. 3.3) [17]. Zone lA is that part of the profundus tendon adjacent to its insertion into which it is impossible to insert the distal half of a Kessler suture, necessitating re-attachment of the tendon to the bone of the distal phalanx. Zone IB is that part of the profundus tendon between zone lA and the distal edge of the A4 pulley, while Zone 1 C is that part of Zone 1 which lies beneath, or proximal to, the A4 pulley. These subdivisions of zones 1 and 2 have clinical implications and represent attempts to analyse these important injuries more usefully and in greater detail than has been possible by the original Verdan classification. Another useful addition to the zoning system would be to sub-divide zone 5 into (a) tendinous distal injuries and (b) those within the muscle bellies, as it is easier to feel happy with early mobilisation of the former than repairs actually within the flexor muscles, unless it is possible to find the intramuscular continuations of the tendons to ensure strong suturing.

By convention, a flexor tendon injury is classified according to the point of penetration

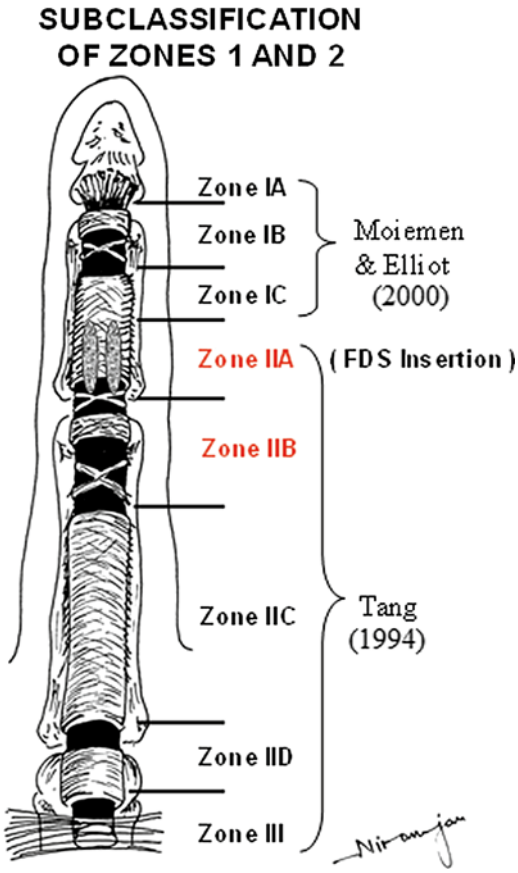


Fig. 3.3 Subclassification of Zones 1 and 2 (Picture courtesy of Mr N Nirinjan FRCS, Consultant Plastic Surgeon)

of its sheath for zones 1 and 2 or, in the more proximal zones, its immediate surrounding soft tissue.

Timing of Repair

Primary repair of the flexor tendons should be as early as possible after the injury. However, there is a body of evidence to show that delay of 24–72 h is not followed by poorer results and it is likely that delayed primary repair by an experienced surgeon will achieve a better result than immediate surgery by an inexperienced surgeon. Transfer of patients to specialist units and delay to investigate and treat more pressing problems is acceptable practice. Although primary treatment is necessary within 72 h, this surgery need not be considered an emergency, or treated as such.

Technique of Repair (See Appendix)

Primary flexor tendon surgery should be performed under tourniquet and full arm or general anaesthesia and should rarely be attempted under local or ring block anaesthesia as the surgery is often more lengthy than predicted and the tendons may have to be handled, or retrieved, in more proximal zones than that in which they were injured. More recently, the practice of 'wide-awake- flexor surgery, or use of local anaesthetic with adrenaline injection into the hand has been introduced to avoid the need for a tourniquet and allow the patient to help evaluate the effectiveness of the surgery by active mobilization of the flexor tendons on surgical request on the table. This has followed the increasing claim that use of local anaesthetic with adrenaline is entirely safe. The author has no experience of wide-awake surgery.

The flexor tendons are generally approached through zig-zag (modified Bruner) skin incisions which are deepened through the subcutaneous tissue, although some surgeons prefer to use mid-lateral incisions in the digits. In zones 1 and 2, the tendon sheath has to be circumvented and this is done with as little disruption as possible while providing sufficient exposure to examine, then repair, the injury to the tendon(s). A window of 2–4 mm in length is created by enlarging the primary wound in the sheath to a transverse opening across its full width then splitting the lateral attachments of the sheath proximally, distally, or in both directions, to allow it to be folded back, exposing the tendons. Where the tendons have been cut with the finger in extension it is often unnecessary to open the sheath more than this to repair the divided tendons as the tendon injury lies directly below the original breach of the sheath. Where the tendons were cut with the finger in flexion, the tendon injury will lie distal to the original cut in the sheath, except when the

finger is held in the same degree of flexion as it was at the time of injury. In such cases, the tendons can only be repaired with the finger in a greater degree of extension that that at the time of injury and greater distal exposure is necessary. The skin incision is extended and the window in the sheath is either enlarged or, if this would involve undue division of the pulley system, the sheath is opened through a second window overlying the distal end of the tendon when the finger is extended sufficiently to allow repair. Because the A2 and A4 pulleys are generally considered to be the most important to function [12, 18], the sheath is opened in such a way as to preserve as much of these pulleys as possible. It is often necessary to divide one lateral attachment of the A2 or A4 pulley along part of the pulley length either to effect repair of the tendons or to allow the repair to glide freely through a full range of motion without snagging on the edge of a pulley, because a flexor tendon repair is inevitably of greater diameter than the original tendon and the uninjured tendons already fit very tightly within the sheath. Fortunately the A2 pulley is of sufficient length that one third, or slightly more, of its length can be released laterally when necessary without resulting dysfunction. The whole of the A4 pulley can be released laterally, although complete release is seldom necessary, without bow-stringing across the distal finger provided sufficient of the A3 and C pulleys have been retained. Distal bow-stringing will occur if all of the sheath is absent between the distal edge of the A2 pulley and the A5 pulley.

Methods of Repair

The method of repair of the flexor tendons has been a matter of considerable debate for many years. The first recognition of the advantage of a primary repair of sufficient strength to allow immediate mobilisation was in 1917 [2, 3]. Because of Bunnell's teaching, it was not until the late 1950s that early mobilisation after primary repair appeared again. Since that time, there have been very many papers written recommending different suture types and materials and a variety of post-operative immediate mobilization regimes for early mobilization of tendons, albeit now in splints and more cautiously than advocated by the first pioneers. A balance of sufficient early mobilisation to achieve good finger mobility and avoid scar adherence of the tendons, but with the minimum number of ruptured tendons is the goal of this endeavour. At the time of writing, there is no 'best' suture material or 'best' suture technique and the choice of each in anyone unit, country or area of the world is more often determined by opinion, historical precedence and availability of particular materials than by science. A landmark paper in the assessment of the strength requisites of these repairs was that of [19]. This paper remains the gold standard for our efforts, although there is a need to repeat this work in the light of the many changes of suture materials and techniques since 1975.

At present, most surgeons feel that flexor tendon repair should include a suture within the tendon, the 'core' suture, and a continuous 'circumferential', or 'epitendinous', suture around the edge of the repair. The principle of the core sutures in common use is that the suture grips the tendon at a distance from the cut ends to prevent the suture pulling through the tendon fibres when subject to longitudinal tension during mobilisation in the first few weeks after repair, at which time the tendon ends soften [20]. This softening of the tendon ends eliminates any advantage of strength of different suture materials [19]. Until the beginning of this century, the commonest core suture used in the United Kingdom was the two-strand Tajima modification of the suture originally described in 1917 by Kirchmayr [3] and re-described in 1969 by Kessler and Nissim [4]. The Tajima modification buries a single knot in the centre of the tendon. The Strickland modification of the Kirchmayr/Kessler core suture in which two knots are buried in the centre of the tendon was more popular in the United States [21]. The Kirchmayr/Kessler suture is generally made with a 3/0 or 4/0 monofilament

polypropylene (Prolene) or braided polyester (Ticron) suture material, with both materials being of adequate strength and each having relative benefits. We use the polypropylene sutures, partly out of habit, partly because we find it easier to pull them through the tendon and partly because they are less bulky to knot. Following the work of Savage in 1985, showing that a six strand Kirchmayr/Kessler-type of core suture was very much stronger than the original two-strand repairs, there has followed two decades of intense activity to find a four, or six, strand suture which achieves a similar strength but is more easily placed within the cut tendons than the Savage suture [22, 23]. Many four and six strand core sutures have been described and tested, mostly in vitro, and the 'best' of these still remains to be identified. Currently most repairs are done with four-strand core sutures.

The tendon repair has been commonly completed using a continuous circumferential over-and-over suture, usually of 5/0 or 6/0 monofilament nylon (Ethilon) or polypropylene (Prolene). This was originally introduced to tuck in ragged parts of the tendon edges to allow easier gliding. However, it became evident that the circumferential suture has considerable strength and is much more significant than the tidying role originally ascribed to it [24, 25]. Several papers then described elaborations which increased the strength of this part of the repair to a point where it could be greater than that of the core suture and many times greater than that required to prevent disruption of the repair during early mobilisation. All of these new circumferential sutures act by gripping the tendon in each throw of the suture in much the same way as the core sutures have done, so that the circumferential suture will grip the tendon on either side of the division with eight, ten or more 'bites'. Like the Savage core suture, these elaborated circumferential sutures are not so easy to insert neatly and with the degree of precision suggested in line drawings by authors of research papers. A further problem of increasing the elaboration of this suture on the surface of the tendon is increasing resistance to free gliding of the tendon within the tendon sheath [26] and it was never established which of the new circumferential sutures provided the most useful balance between additional strength and increased resistance to movement. Although elaboration of the circumferential suture raised the possibility of dispensing with the core suture entirely, the pendulum of research activity has swung back more recently to increasing the core suture strength, with simple circumferential over-and-over suturing being the commonest finishing suture in current use. This may reflect the difficulties of inserting the more complex circumferential sutures, particularly along the deep surface of the tendon repair after completing the core suture and, particularly, in the tight confines adjacent to the A4 pulley. Placing these sutures along the 'back wall' of the repair prior to completing the core suture, a technique commonly used with simple circumferential suturing to avoid bunching of the repair, is very much more difficult with the complex circumferential sutures.

Critical analysis of the rupture rates in the clinical papers written during the last 10 years fails to show a consistently significant reduction in rupture of repairs, despite the laboratory evidence that these sutures are stronger than two-strand repairs. Most reported series of primary flexor tendon repairs in zone 2 of the fingers, which has been the testing ground of flexor tendon surgery for 50 years, include a rupture rate of approximately 5 %, whatever method of core and circumferential suturing is used. As yet, it remains to be seen whether any of the new core or circumferential sutures have affected these figures, although many have been shown in the laboratory to have considerably more strength than the conventional core and circumferential sutures. In 1994, and again in 1999, we presented results with a 4–5 % rupture rate of 17 of 397 (4 %) fingers with tendon injuries in zones 1 and 2 following use of a conventional two-strand Kirchmayr/Kessler core suture of 3/0 or 4/0 polypropylene (prolene) and a running circumferential over-and-over suture of 5/0 or 6/0 monofilament nylon (Ethilon) or polypropylene (Prolene) [27, 28]. These results still stand nearest to Savage's rupture rate of 1 of 31 (3 %) of finger tendon repairs

in Zone 2. There is also some limited evidence that suture material has a deleterious effect on tenocyte activity and, hence, a possibility that increasing amounts of suture material increase this effect [29].

While researching repair of the flexor pollicis longus (FPL) tendon, we experienced a much higher rupture rate of repairs of this tendon than the 5 % occurring in our zone 2 finger flexor repairs [27]. Difficulty carrying out primary repair of the FPL tendon, probably because of the particular tendency of this muscle to retract more than the finger flexor muscles, has been well recognised in the literature from as long ago as 1937 [30], although this particular flexor has received little attention since the 1950s and 60s. The higher rupture rate of this repair makes it particularly suited to testing the adequacy of the newer sutures clinically, in preference to the time-honoured zone 2 model. We found that addition of one of the newer circumferential sutures, described in 1993 by Silfverskiold and Anderson [31], to a conventional Kirchmayr/Kessler suture reduced the rupture rate of the FPL repair considerably [32]. In a more recent series of FPL repairs, in which a second Kirchmayr/Kessler suture, inserted at right angles to the first (which is one of many new methods of creating a four-strand core suture) with the Silfverskiöld circumferential suture, has reduced the mechanical rupture rate to zero [33]. This work would suggest we increase the complexity of the core sutures, the circumferential sutures or both.

However, inserting these more complicated sutures may be at a cost. They are more difficult to insert and make an already complicated procedure even more so. Bearing in mind that most primary flexor tendon surgery is carried out by trainee hand surgeons world-wide, this may prove a serious disadvantage to their use. An alternative approach has become increasingly popular in the Far East, where Tsuge described a single suture repair with a looped double strand nylon suture which acts to grip the tendon on either side of the division in a manner which is akin to a single, large epitendinous suture (Fig. 3.4B) [34, 35]. This was elaborated by Tang, who suggested using three Tsuge sutures spaced evenly around the circumference of the tendon [36]. Tang and his colleagues examined the strength of a number of suturing techniques, including both their own ‘triple-Tsuge’ technique and the combination of a two-strand Kessler suture and a Silfverskiöld circumferential suture which we had used in FPL repairs [37].

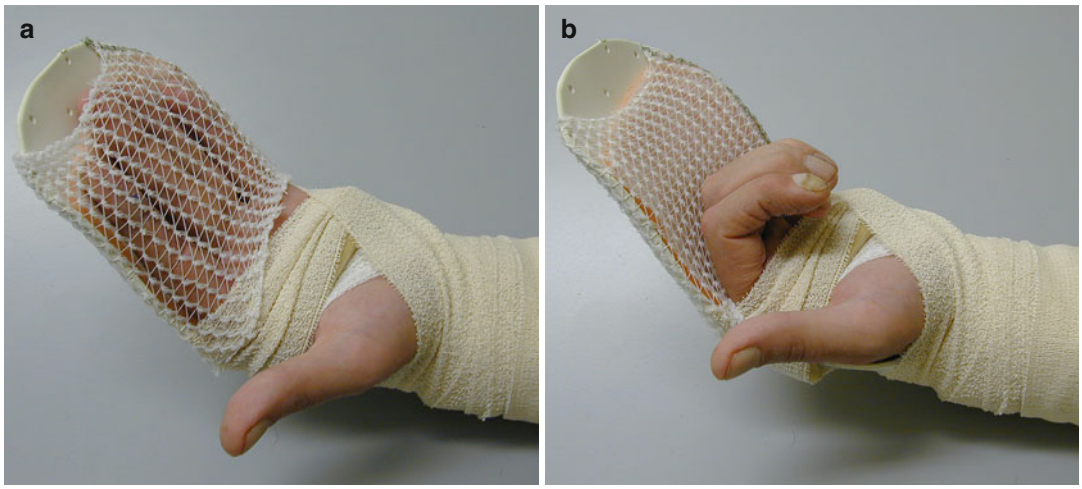


Fig. 3.4 (**a**) The current splint for early active motion used by the author’s unit, incorporating the sleeve to encourage interphalangeal extension. (**b**) The hand is shown in the sleeve between exercises and with the fingers out of the sleeve to allow exercises

While all of the more elaborate sutures used in this study showed greater strength than a simple two-strand Kessler suture, and all were sufficiently strong to resist early mobilization, the Tang technique proved the strongest. However, it is not particularly the strength of the Tsuge-Tang approach which is most appealing, as Tang and his colleagues still could not avoid the inevitable small rupture rate in their earlier clinical study, but its simplicity at a time when the Western approach may be becoming too complicated. The most recent of our studies of FPL repair included fifty FPL divisions repaired by the Tang suturing technique and mobilized actively with no ruptures during mobilization [38].

Management of the Tendon Sheath

The period of closing the tendon sheath completely has passed. The repaired tendons, which are inevitably greater in diameter than the original tendon, are more likely to suffer restriction of their free movement if the sheath has been closed. Results in studies 30 years ago in which the tendon sheath was only laid back, but not sutured, were as good as in those with complete sheath closure. However, even with a policy of simply laying the sheath back, catching of some repairs on the main pulleys remains a problem. It had become recognized that one had to preserve, or reproduce, the A2 and A4 pulleys, as a minimum when carrying out secondary tenolysis surgery if the mechanical function of the flexor system was to be preserved [12, 18]. This became translated into an absolute need to maintain the A2 and A4 pulleys in their entirety during primary tendon repair, despite their being the main cause of repairs catching. Complete preservation of these pulleys had become almost sacrosanct, although work by Savage (1990) had shown that parts of these pulleys, and even the whole A4 pulley, could be removed without significant loss of mechanical function, provided the remainder of the sheath was mostly intact [39]. This work has been confirmed by others [40–42].

In 1998, we examined one hundred zone 2 repairs between the distal edge of the A2 pulley and the zone 1/ zone 2 interface, at the distal end of the flexor digitorum superficialis insertion into the middle phalanx (Tang's zones 2A and 2B) [43]. The repairs had been done by senior registrars in Plastic Surgery with considerable experience of operative hand surgery. This study shows that 64 % of these repairs require some lateral venting of the A2 or, more commonly, the A4 pulley. Sometimes, partial pulley venting of the A4 pulley was necessary at an early stage to place the core suture into the distal tendon end, as the transverse pass of this suture has to be placed 0.5–0.75 cm distal to the tendon division, and this is sometimes under the A4 pulley in these particular injuries. Sometimes, either the A2 or A4 pulley had to be partially vented to get a full free range of passive motion of the tendon repair without catching on the pulleys, before closing the finger. The degree of venting of the A4 pulley in our study varied from 10 to 100 %. Complete division of the A4 was necessary in 14 cases. When we analysed these 14 cases carefully, we realised that it was inevitable that some flexor tendon injuries would be placed such that the A4 pulley either had to divided completely for one of the two reasons described above, or for a combination of both. It has yet to be proven that venting, partial or complete, has no effect on the long-term results of primary repair. However, over and above these observations of the necessities of clinical practice, venting the pulleys would seem, intuitively, correct unless perfect repair of the tendons, exactly reproducing the original diameter of the undivided tendons, is routinely being achieved. Repairs snagging on pulleys treated by early mobilisation will either restrict movement of the finger or cause the repair to snap. In reality, this is only a problem of the A4 pulley, as the A2 pulley is of sufficient length that one can excise any third of it to allow repair and free movement of the repair and still have a pulley which is one, or more, centimetres in length and, so, functional. In that there appears to be no indication in secondary flexor tendon surgery to reconstruct an A4 pulley alone for bow-stringing at the DIP joint level, it is unlikely that the A4 pulley, in itself, is vital to flexor tendon function when most of the remainder of the sheath is

intact. Distal bow-stringing will only occur if all of the sheath is absent between the distal edge of the A2 pulley and the A5 pulley. It is, therefore, beholden on the trauma surgeon, when the profundus tendon is cut in the proximal part of the finger with the PIP joint flexed, to open a separate window beyond the PIP joint to repair this tendon distally, so preserving most, or all, of the A3 and C pulleys.

It is now our practice to simply lay the sheath back over the tendons after adequate lateral venting of the pulleys to allow free running of the repairs. Free movement of the repair is then tested for a final time by passive movement of the finger through a full range of motion before closing the skin of the finger. Haemostasis of the wound is then carried out and the skin closed. The hand is placed on a padded POP dorsal splint which prevents finger extension beyond mid flexion and the hand elevated overnight.

Post-operative Management

The fundamental need to mobilise repaired flexor tendons early to avoid adhesion and loss of tendon gliding is now generally accepted, as is the fact that healing of the flexor tendon, at least in the digits, does not necessitate the formation of adhesions. Experimental evidence would also suggest that this early movement encourages more rapid tendon healing under the influence of longitudinal forces [44–47]. At the present time, flexor tendon repairs are mobilised by most surgeons in a dorsal blocking splint as an additional safeguard against tendon rupture. Early mobilisation of repaired flexor tendons without protection was first advocated nearly a century ago but we have not yet returned to this freedom of post-operative activity since the re-introduction of early mobilisation 50 years ago. A definitive dorsal thermoplastic splint is generally applied 24–72 h after surgery, whatever the technique of rehabilitation. While the interphalangeal joints are invariably allowed to fully extend, the precise angles to which the wrist and MCP joints may extend in these splints vary from unit to unit. The degree of standardisation of splint construction possible in clinical practice probably belies such precision in print and the variability of the statements in the literature would suggest that the precise degree to which these joints are allowed to extend may not be of great significance. This is discussed further later.

The first of the early mobilisation regimes has come to be known as “Kleinert traction”; after one of its pioneers, but is more accurately described as “active extension -passive flexion” mobilisation. The original intention of this regimen was to move the fingers in a dorsal splint preventing full extension of the wrist and metacarpophalangeal joints, with extension of the fingers by active use of the extensor tendons but flexion of the fingers passively by rubber bands attached between the fingertips and the flexor aspect of the mid-forearm,. This was intended to achieve flexor tendon gliding without subjecting the tendon repairs to direct tension during finger flexion. Although it has undergone several modifications, the technique remains unchanged in principle. Probably the most significant of the modifications has been the change of direction of the line of pull of the rubber bands to encourage interphalangeal movements by the addition of a palmar bar [48, 49]. All early mobilisation regimes suffer the problem of how diligently the patient actually carries out the finger movements at home, but this one has the additional problem of whether the patient actually uses the rubber bands to passively flex the fingers, or uses the repaired flexor tendons and actively flexes the fingers, despite the presence of the rubber band driven alternative. Another criticism has been the fact that the fingers rest in the flexed position between exercise sessions. In those less able and/or co-operative patients who move the fingers seldom, the fingers spend long periods flexed and may develop flexion contractures of the proximal interphalangeal joints, which are difficult to correct later. Although this has been identified as a particular problem of Kleinert traction in the past, modifications, such as suggested by Sifverskiöld and May, in 1994, which prevent prolonged resting with the PIP joints acutely flexed, can prevent this [50]. Tensioning the bands is also a problem as the forearm attachment of the bands often moves and the bands slacken,

sometimes within a few minutes of being tensioned by the therapists. Many more recent papers describing use of this technique have incorporated features of the alternate techniques described below, to try to overcome the problems of the original method [50, 51]. Variations of this regimen remains the most commonly used of the early mobilisation techniques worldwide today although the popularity of the 'Kleinert' regimen is declining.

Throughout the 40 years since Kleinert traction was introduced, two alternatives have been developed. In one, the repaired flexor tendons are mobilised completely passively in both directions, either by a therapist or by the patient with his/her other hand. This technique was introduced by Duran and Houser in 1975 and embraced by Strickland and his colleagues in Indianapolis [52, 53]. Although the original authors had a 15 % rupture rate and only 53 % of the digits achieved a good or excellent range of motion in the second paper, this technique has been used widely, particularly in the United States. However, it is very expensive in therapy time which militates against its use in most countries. More recently, features of this regimen have been added to the basic Kleinert regimen by many surgeons in North America to try to achieve fuller ranges of extension and flexion.

Through-out the period since the introduction of the Kleinert regimen, a number of surgeons have described techniques of mobilisation by both active flexion as well as active extension of the fingers, as proposed by the pioneers at the beginning of this century, but with protective dorsal blocking splints. Most of these active regimes have involved variations of suturing to cope with the perceived increase in tensions on the repair during active flexion. In 1989, the Belfast surgeons actively moved flexor tendon repairs in zone 2 without any addition to the suture technique, effectively a Kleinert regimen without the rubber bands [54]. The original name of "early active motion" has been changed in some subsequent publications to "controlled active motion" and the detail of this technique of mobilisation varies slightly between units, but the principle is unchanged. Subsequent reports from units using variants of the Belfast regimen through the early 1990s achieved results similar to those reported using the Kleinert technique [27, 55–57]. The advocates of the "Belfast" regimen believe it to be simpler, cheaper and easier to manage for both patients and therapists than the "Kleinert" regimen and its variants.

Of the ongoing controversies in flexor surgery, possibly the one most often discussed is which is the best of the mobilization techniques today: Kleinert, now often amalgamated with Duran-Houser, or Early Active Mobilization. This discussion is an unproductive exercise. If one looks at both techniques closely, one realizes that both are moving towards freer movement and both are pushing repairs ever harder during the early postoperative period. The series reported by Sifverskiold and May, in 1994, from Gothenburg in Sweden, has the best results reported from a civilian unit, to date, in the world [50]. It actually combines features of Kleinert, Duran-Houser and Belfast mobilizations. As we all get more aggressive in our mobilization of repaired flexor tendons, the problem is not which regimen of mobilization to use, but how far we can go along this track without increasing the rate of tendon rupture.

We examined our own rupture patients to try to identify a soluble common cause for the problem [28]. Unfortunately, a 5 % rupture rate means only 5 people in every 100 flexor repairs are available to study, so it takes a long time to collect a meaningful series of rupture patients. We also added zone 1 to the zone 2 cases to increase the numbers and still only managed to collect 23 digits with ruptures in 23 individuals out of approximately 500 digits with flexor tendon repairs in 6 years. Our study shows a rupture rate of 5 % and 4 % in zone 1 and zone 2, respectively. Forty-seven percent of these patients ruptured their repair doing something stupid, with one patient having ruptured his repair while lifting furniture. Some had taken off their splint and some ruptured in their splint, although our splint is intended to stop active hand use. Several years ago, we felt that one of the advantages of Kleinert traction over the Belfast regimen, in this respect, was the fact that the rubber bands prevented the

patients placing objects in the palm and gripping them, so we added what the patients referred to as 'anti-beer can bars' to our splint to achieve the same effect. Most ruptures in both sexes occur in young patients who, as an age group, are, perhaps, more inclined not to listen and do inadvisable things with their injured hands. However, there was a small group of older men, in their forties, who ruptured their tendon repairs despite, mostly, doing exactly as instructed by our therapists. So, increasing age in the male, or, possibly, in either sex, may be another factor making repairs more liable to rupture. Unfortunately, we would have had to analyse another two to three thousand patients for this observation to achieve statistical significance. This study does suggest that the only practical way to reduce the rupture rate, at present, is to strengthen the sutures, so that moving furniture during the early post-operative period can be accommodated – even if not recommended!

For the last 20 years, we have used a variant of the regimen of early active mobilisation described in Belfast in 1989 (Table 3.1). Our own preference at present is to have the wrist in approximately the neutral position and the MCP joints at 40° of flexion, which angles represent a slight modification of the wrist position of 30° of flexion and MCP position of 30° of flexion which we advocated in 1994. While the precision with which these angles are described may not be important, the reasons for the modifications provide a useful vehicle for consideration of some of the finer points about rehabilitation. The degree of flexion of the wrist is probably less significant than previously believed. In fact, the extreme flexion advocated in early papers is almost a Phalen test position and can have the same effect on the Median nerve as this test, particularly when associated with the considerable local oedema of a zone 5 injury. Savage (1988) suggested that flexion of the wrist did not achieve less tension on flexor tendon repairs distal to the wrist because any relaxation of the flexor muscles was countered by increased tension on, and spontaneous firing of, the extensor muscles, applying force to the repairs in the opposite direction [58]. He suggested that the position achieving least tension on repairs in the fingers and palm was the 'resting position' in which we commonly splint hands, with the wrist in slight extension. Several units now splint the wrist in this position. We examined fifty patients mobilised in this wrist position and found no increase in tendon ruptures with the same percentage of good and excellent results as we had reported previously with the wrist in the flexed position (Elliot, 1999). Loss of full extension of the interphalangeal joints remain a problem of both current active mobilizing regimes, and achieving full extension of the PIP joints, particularly that of the little finger, is still difficult. In fact, it is documented that the results of repair in the little finger are worse than in the other fingers, with rupture

Table 3.1 The St Andrews early active mobilisation regimen (2014)

Week 1	Discharge from hospital when pain is controlled by simple oral analgesics, the patient is able to do any necessary dressings and is achieving full extension to the splint and active flexion to 25 % of full flexion. Patient instructed to carry out ten active flexion and extension exercises per hour.
Week 2	Seen by surgeons and therapists. Full extension to splint and active flexion to 50 % of full flexion. Ten exercise repetitions per hour. Passive flexion exercises started.
Weeks 3 and 4	Seen weekly by therapists only. Full extension to the splint and progression to full range of active flexion as soon as possible (usually achieved by the end of week 3). Ten exercise repetitions per hour. Passive exercises. Ultrasound started if necessary in week 3.
Week 5	Seen by surgeons and therapists. Splint removed, except at night and when there is risk to the hand (e.g. in crowds). Wrist extension started, at first with the fingers relaxed.
Weeks 6 and 7	Seen weekly by therapists. Progression to full range of movements of wrist and fingers.
Week 8	Splint discarded completely. Passive extension exercises and dynamic extension splinting started, if necessary. All but heavy activities allowed, including driving. Return to work (except heavy manual workers)
Weeks 10–12	Progressive return to heavy work by week 12.

of the primary repair more likely and re-rupture of any re-repair also more likely [59].

As early as the first week, when necessary, our therapists place a pencil, or one of their own fingers, behind the proximal phalanx during active extension to lift the fingers away from the splint, so increasing the flexion of the MCP joints temporarily in order to encourage full PIP joint extension. Passive extension exercises and dynamic extension splinting are also now started slightly earlier, during the eighth post-operative week. Our therapists feel that the recent small increase in MCP joint flexion of our current splint helps increase the action of the intrinsic extensor tendons on the PIP joints, so reducing this problem. Unfortunately, these methods of increasing MCP flexion are ineffective in zone 5 injuries to the tendons of the little finger which include division of the ulnar nerve, and, so, paralysis of the intrinsic extensor muscles. Failure to extend the PIP with secondary contracture of the PIP palmar plate ligament of the little finger continues to be a problem of this particular injury. A few years ago, we introduced a modification of the original splint, to further encourage PIP joint extension. The protective palmar bars added to the dorsal splint in the nineteen-nineties have been replaced by a sleeve of elasticated open weave material for the period of continuous splinting (Fig. 3.4). This is worn at all times and exerts a slight extension force on the inter-phalangeal joints. The sleeve is rolled proximally into the palm when doing exercises. Ultrasound is introduced at an earlier stage of healing when finger movements are more sluggish than expected, previous fears about ultrasound having a deleterious effect on the early stages of healing now being believed to be incorrect.

Whatever the method of rehabilitation, there appears to be relative consensus of opinion about the length of rehabilitation, although the source of the timing of the various stages of this assisted recovery is obscure. Whichever technique of early mobilisation is used, flexor tendon repairs are mobilised in dorsal splints with no active grasping with the fingers for 4–5 weeks. There follows a period of 3–4 weeks of gradual increase of activity, with the splint only being worn at night and in public places, where the fingers might be accidentally pulled into extension. Full use of the hand for light activities and therapy to correct failures of finger extension begins only after 8 weeks, with heavy grasping activities being avoided for 12 weeks. Patients return to sedentary manual activities at 8–10 weeks and to heavy manual labour at 12 weeks after surgery. Although suggestions of shortening of the period of splinting are sometimes aired in meetings, they have not yet appeared in print. Our finding that all but one patient who ruptured a primary flexor repair in the fingers did so in the first five weeks, with three of twenty-three ruptures (13 %) occurring in week five and none in week six (Harris et al., 1999) would suggest that the current period of splinting is close to correct [28].

Adequate analgesia is necessary and the use of anti-inflammatory drugs during the early post-operative period is of particular value not only for their general analgesic effect but also in allowing the therapists to encourage early movement in nervous patients and those with a low pain threshold and/or concomitant painful nerve injuries.

Most units publishing in this field report only about 70–80 % of patients achieving good or excellent finger mobility in exchange for a rupture rate of about 5 % and a similar incidence of tendon adhesion, whatever the method of rehabilitation. Clinical testing of cases with incomplete ranges of movement very frequently identifies a passive loss of flexion of the involved digit, indicating that extensor tendon tethering by fibrin and, later, scar to its surrounds is a more common reason for this problem than flexor tendon adherence to the sheath [60]. However, both may occur concurrently.

Hand surgeons currently report their results using systems of assessment in which ‘excellent’ may be a result less than normal. For example, in the Strickland I Assessment, ‘excellent’, as defined, may only be 85 % of normal function [53]. Bearing this in mind, an international audit from units which are probably achieving results which are better than their national average in which only 70–80 % of results are reported as good or excellent, with one in every ten repairs either rupturing or adhering, is not impressive and concern to make improvement is justifiable.

Repair in Other Zones

Almost all of the research on flexor tendon surgery has been carried out in zone 2 injuries, which early researchers perceived to be the most difficult zone in which to get good results. Indicative of this concentration on zone 2 is the finding that there have been nearly twenty methods of assessment of flexor tendon repairs described since 1950 and all of them had been designed for assessment of zone 2 injuries [61]. As a result, Zone 2 methodology has been extrapolated to the treatment of the other zones, despite the enormous differences anatomically in the different zones, both in the tendons themselves and in their surrounds. As a pure flexor tendon injury, the zone 2 injury is, arguably, the most difficult technically to repair, although the space in which one is operating is much more confined in the less frequent zone 1 injury. However, as a total injury, injuries of zones 3, 4 and 5 are often more devastating to hand function and more difficult to rehabilitate, not so much because of the tendon injury per se but because of the associated damage done to the other structures in the zone. There is a need to analyse injuries in the other zones in more detail and to examine whether our zone 2 surgical and mobilisation techniques are directly applicable. Our current ways of assessing the results may also not be entirely appropriate in the other zones, as we discovered when analysing our zone 1 results [17].

In the 1970s, Kleinert wrote that he could see no reason not to apply the lessons of zone 2 surgery to zone 5, so early mobilization and repair of both flexor tendons in zone 5 became the norm. As a consequence, every divided structure in flexor wrist wounds has been repaired at the first operation for the last 40 years, although there had been a long held belief before Kleinert that only the FDP tendons should be repaired in these injuries, as also repairing the FDS tendons caused tethering problems. A small study paper published in 1992 by Stefanich and his colleagues reported only 37 % of 19 patients to have achieved independent FDS action after zone 5 immediate repair and early mobilization in a Kleinert regimen [62]. We were intrigued and started a prospective study of zone 5 [63]. We examined 50 patients with zone 5 injuries mobilized in the Belfast manner. Our results showed independent FDS action in only 66 % of 161 fingers with one, or both, flexors cut at the wrist but good, or excellent, ranges of motion in 90 % of the fingers. For the first time, this paper attempted to assess the interaction between the fingers when more than one set of flexor tendons had been injured. These results were rather better than Stefanich's paper had suggested, but indicated far from perfect FDS function. Independence of FDS action in the fingers, while not of great importance to power gripping, is a significant factor in the more controlled and individualised finger movements we use in finer hand function, so a more effective mobilization regimen for the zone 5 injury is needed.

In 1991, a paper by Gerbino and his colleagues in the United States, reported a series of 20 patients with zone 1 injuries which found very significant difficulties in this zone, with a complication rate of poor ranges of motion, sticking repairs and rupturing repairs of 35 % [64]. This was a very small study but it was one of only two studies which we could find in the English literature at that time in which zone 1 injuries had been treated in approximately the same surgical manner, with two-strand Kessler core sutures and circumferential sutures, which we were using. Gerbino and his colleagues reported results of zone 1 injuries mobilised using Kleinert traction while we were using an early active mobilisation regimen. In 1990, a paper reporting the results of an American physiotherapist, Evans, who had treated the patients of many different American surgeons local to her practice using a variant of the Duran-Houser passive mobilisation regimen recorded an even higher complication rate [65]. We reviewed our results of zone 1 injuries over a period of 8 years [17]. The study included 93 zone I tendon divisions in 89 patients, repaired with a Kessler core suture and a simple circumferential suture, then mobilized in the same early active mobilisation regimen as used for zone 2 injuries.

In respect of the good and excellent results in this study, our results were similar to those of Gerbino et al. in their US Army study, with good

or excellent results in 60 % of fingers using the original Strickland assessment and 47 % of fingers on DIP only assessment, and both studies had a rupture rate of 5 %. However, only 4 % of our repairs required tenolysis while Gerbino et al. reported a complete loss of DIP movement in 30 % of their cases, which they attributed to gapping of the repairs from over-zealous mobilization. We believe the problem in these cases is catching of the repairs on the A4 pulley after completely closing the sheaths following tendon repair. Our policy is to vent, or cut, the A4 pulley as much as necessary at the time of zone 1 repair to allow free movement of the repair and, then, to leave the sheath completely open. We think this is more realistic in the very closed confines of zone 1.

Severe Injuries

We all use the same mobilising regimen for the flexors in severe finger and palmar trauma that we use for simple tendon divisions but this is, yet again, an extrapolation of the study of simple injuries of zone 2 and the author is not aware of any work having been done to examine whether we should be doing anything different either surgically or post-operatively for more severe injuries. We simply expect poorer results for worse injuries and we tend to get them! The poor results in bad injuries may also be a result, at least in part, of applying the same surgical techniques which we use in simple injuries to a much more complex situation. In the 1960s, surgeons were uncertain whether one could repair both tendons in the tendon sheath without causing problems of tendon adhesion. Both Kleinert and Verdan showed that this was not only possible but, in fact, preferable in terms of achieving maximum strength and better independent finger function. It also led to fewer complications. Recently, this has been questioned by Tang for those injuries occurring in what he has defined as zone 2C, which is the part of zone 2 under the A2 pulley at the base of the finger [36]. This is the tightest part of the sheath, where the FDS also wraps itself around the FDP. Tang examined 37 fingers in 33 patients with zone 2C injuries and showed better results in those fingers in which he repaired the FDP alone. This problem area of zone 2 has been recognized before: Boyes and Stark, in 1971, identified this same trouble spot under the A2 pulley as that where tendon adhesion was most likely to occur, although they did not go so far as to propose single tendon repair as a solution [66]. In a study of zone 2 injuries (Kwai Ben and Elliot, 1998), we looked at zone 2C with Tang's idea in mind but could find no particular problems in our 31 zone 2C injuries, when compared with the injuries elsewhere in zone 2, to substantiate his idea of single tendon repair in zone 2C [43]. The author believes there is a role for this idea in more complex injuries and for repair of simpler injuries in Zone 2C and D if being carried out by inexperienced surgeons. Over the last 20 years, the author has treated eleven patients who had had severe injuries of the distal palm and bases of the fingers in whom both flexor tendons were repaired by training surgeons in zones 2C or 2D routinely at primary surgery. The tendons subsequently became swollen and became stuck under the A2 pulley. At secondary surgery, both tendons immediately proximal to the A2 pulley were swollen to a degree that each was the diameter of both together in their normal state. The FDS tendons were removed and the FDP tendons alone only just moved freely under the A2 pulleys. Like many surgeons, I have repaired only the FDP in replants for a long time. Tang's single tendon repair seems logical for other severe injuries in this part of the hand, such as tendon divisions of all four fingers, bad crush or lacerating injuries, and replantations and revascularisations at the bases of the fingers or in the distal palm, to try to avoid secondary surgery. The same problem arises if all of the flexors are repaired in the carpal canal after severe injuries at this level, such as the typical machete wound to the ulnar side of the wrists when an unarmed person raises his, or her, hands to protect the face during such attacks.

The Zone 2 'Black-Box'

While zone 2 has become the testing ground for mobilization techniques and the means whereby

individual units announce their presence on the flexor tendon stage, most of the well- known clinical papers on this subject report only 50 or 60 cases, at most. So, it is obviously difficult to collect more than about 50 cases of zone 2 injuries in one unit in a reasonable period of time. As a result of this logistic dilemma, three problems arise. The first is that the total number of clinical studies in the world literature remains small because few units can accumulate a respectable number of cases. The second is that most studies are small in themselves and, in many cases, too small to examine scientifically and the third is that the available numbers are too small to allow analysis of what is actually inside the 'black box' which we have chosen to call 'ZONE 2', as everything has to be grouped together to get enough patients to achieve a publishable series. Zone 2 is far from homogeneous in a number of ways. There are eight permutations of tendon injury within this part of the tendon sheath. We simply ignore all partial injuries and group the others together, although these include five different tendon injuries. Recently, we have examined this and, although this study is not yet finished, it appears that there may be differences in the results of treatment of the different injuries, with certain combinations of tendon lacerations having worse results than others after mobilisation using our current rehabilitation regime. Another possible problem of treating zone 2 as a 'black box' may arise in fingers in which both tendons have been divided. There will be two tendon suture lines. If the tendons were cut with the finger in a partially flexed position, these will be a long way apart when the finger is extended and only come together in flexion. By contrast, when the finger is cut in extension, the tendon repairs remain close together all through flexion and extension. Consequently, one might expect the results to differ between these two injuries and it might be advantageous to rehabilitate these two injuries differently. Another fact about zone 2 which we ignore is that the sheath is not a cylinder of unchanging topography along the length of the zone. This is also true of Zone 1. So the tendon environment is varying quite dramatically along both zones. As mentioned earlier, Tang has tried to split zone 2 into 4 subzones and we have split zone 1 into 3 subzones, but most authors do not have sufficient numbers to subdivide the zones in this way. Larger series are needed to make more meaningful comments about these unanswered questions locked in the zone 2 black box.

Classification of Injuries

We continue to describe our flexor tendon injuries using a system of classification which may have outlived its usefulness, at least in part. Although the current zoning was a modification by Kleinert and Weiland in 1976 [15], the zones were actually defined by Verdan in the 1950s before the problems of the pulleys had been studied in any detail [9, 10, 14]. Although all the other zone interfaces were defined by recognizable extra-tendinous anatomical structures, Verdan defined the interface of zones 1 and 2 as the distal end of the attachment of the FDS tendon to the middle phalanx. This has come to be meaningless: in a recent study, we found that this interface lies under the A4 pulley in 70 % of fingers while the FDS insertion is completely proximal to the A4 pulley in 30 % of fingers [43]. Once the pulleys had been identified, this particular zone interface should have been redefined, or abandoned. Perhaps, we should be recording those finger lacerations which divide the flexor tendons with greater attention to the detail of the tendon injuries themselves and their relationship to the pulley system during flexion and extension of the finger than to the place of penetration of the tendon sheath, according to Verdan's classification, as we do currently. The zoning system also causes confusion as a result of the movements of the tendon injuries with the movements of the finger. Consider a mid-palmar laceration, cutting the flexor tendons when the fingers happened to be in mid- flexion. By definition, this will be a zone 3 injury, but, when the fingers are straightened, the cut tendons will move distally into zone 2 and this injury will have the problems of a proximal zone 2 (zone 2D) injury of sticking under the Al, or A2, pul-

leys. The same applies to tendons cut in flexion in the distal part of zone 2, moving into zone 1 on extension of the finger. The time may have come to rethink some of the ground rules for discussion of flexor tendon surgery. We appear to be trying to apply a zoning system which has become too crude for the finer analysis necessary to scrutinise this subject more closely. Just as the term 'No- Man's Land' eventually had to be replaced, perhaps the time has come to reconsider the zone system for discussion of these injuries.

Specialisation in Flexor Tendon Surgery and Rehabilitation

The last 50 years is notable for the reversal of Bunnell's policy of universal secondary tendon surgery and recognition that results after primary or delayed primary flexor tendon repair, that is within a few days of tendon division, can be better than after delayed tendon grafting. However, this is only true if surgery is carried out by adequately trained surgeons and followed by early mobilisation of the repaired tendons. The growth of specialist hand therapy during this period has been an essential factor in this change. For the future, emergency services should be arranged to relocate these injuries to appropriately trained surgeons and hand therapists. Where this is possible, training of hand surgeons should be organised to allow maximum exposure to acute flexor tendon surgery as this treatment will deal with 80–90 % of all injuries to the flexor tendon system.

Appendix: Fine Detail of the Technique of Primary Flexor Tendon Repair

1. **This surgery should be performed under tourniquet control**, so, requires adequate anaesthesia proximal to a tourniquet which will maintain the patient's limb comfortable for a minimum of one hour for a single digit. This is longer than it takes to suture two flexor tendons but the exact extent of the injuries are not known before exploration.
2. **Draw a conversion of the skin wound into a modified Bruner incision** to allow access. At the level of the injury, the Bruner incision may need to allow access to the neurovascular bundles to repair them. Proximally and/or distally, the incision only needs to allow access to the tendon sheath so the width of the points of the Bruner incision need be less and never extending out to the mid-lateral line.
3. **Open this skin marking adjacent to the skin wound only**, extending it as necessary as the operation progresses rather than opening the whole length of the finger at the beginning.
   (i) ascertain if the neurovascular bundles are cut.
   (ii) expose the wound of the tendon sheath. Widen the sheath wound slightly by excising 2–3 mm of the sheath to identify the precise injuries to the two tendons and whether the tendons have been cut in finger flexion or extension. The exact order of events to effect repair will depend on both of these factors. For the purpose of describing the various 'tricks' which are commonly used to facilitate repair, a division of both tendons at the commonest injury site, viz. just proximal to the PIP joint crease, is considered below.
4. **If the tendons have been cut in finger extension**, the tendon ends will be visible, at, or in the vicinity of, the tendon sheath opening. The Bruner incision is opened by a small amount proximal and distal to the initial skin wound. Then, sufficient of the sheath is opened by longitudinal incision laterally on one, or two sides if necessary, from the sheath wound to allow tendon repair. Repair is carried out partly by delivering the cut tendon ends from the sheath and partly by opening the sheath, with the former increasing and the latter decreasing as the experience of the operator increases. The proximal tendon ends are first delivered into the opening in the sheath. The ends may appear in the sheath opening or sufficiently near to this opening to be held and pulled into the operative field with a small

toothed forceps or arterial clamp gripping the centre (but never the sides) of the proximal tendon end. In other cases, the proximal tendons can be exposed by flexing the joints proximal to the tendon end and 'milking' the forearm flexor compartment from proximal to distal. If the tendon ends have still not been retrieved, it is reasonable to pass a small, curved artery clamp into the proximal sheath for 1–1.5 cm and try to grip the tendon end centrally. The confines of the sheath will not allow the clamp to open sufficiently to grip the circumference of the tendon. Repeating this manoeuvre more than once, or twice, with the proximal joints flexed and the forearm milked is rarely successful. If unsuccessful, open the palm with a 2 cm C-shaped incision at the level of the distal palmar crease in the line of the involved digit and identify the two flexor tendons just proximal to the beginning of the tendon sheath. Without pulling the tendons proximally and delivering their cut ends into the palmar wound, pass a fine sterile plastic medical tube up the sheath alongside the tendons to the wound in the finger, then suture the tube to the profundus tendon in the palm with a side-to-side suture and use the tube to pull the proximal tendon ends into the injury wound in the finger. When both tendons have been divided, if attention is directed at one of the two tendons, the other will also move distally because of their anatomical arrangement. As they are moved distally by pulling on the plastic tubing from the finger wound, the profundus tendon will also remain in its correct position between the two halves of the superficialis tendon. If only the profundus tendon has been divided, then care has to taken that the plastic tube and the profundus tendon pass through the chiasma of the superficialis, and not around it, during this manoeuvre. This can be done by visualising the chiasma from the finger wound as the tubing is passed distally. Deliver just more than 1 cm of the proximal tendon ends into the injury wound, and insert a core suture into the deepest tendon and then use this suture to maintain the distal hold on the tendon ends. As the superficialis tendon will lie behind the profundus at this level of injury, it is repaired first. Allow the tendons to drop back to the original position and remove the tube and suture used to deliver them from under the A2 pulley through the palmar wound. Occasionally, for proximal digital and palmar injuries, this same manoeuvre is necessary at the wrist to achieve retrieval of the proximal tendon ends from the carpal tunnel. Pulling the core sutures distally again from the injury wound, move the proximal ends until they are just within the operating field at the injury level but not fully delivered and occupying all of this, very limited space. Secure this proximal tendon position by passing a fine hypodermic needle transversely through the sheath and the tendons 1–1.5 cm proximal to the working area, making sure to lodge the sharp end of this needle safely in fat without injuring a neurovascular bundle, but so neither operator nor assistant suffers a needle-prick injury during the tendon repair. The distal tendon ends are then delivered into the wound by flexion of the interphalangeal joints and the second half of the superficialis core suture passed through the distal tendon end. This is easier if the proximal tendon ends have not been fully delivered into the wound. At this stage, the hypodermic needle holding the fixed position of the proximal tendon ends can be removed as the proximal tendons are now linked to the distal tendons and cannot drop back into the palm. Having removed the needle, the proximal end of the superficialis can now be moved distally to meet the distal end. A common problem of flexor tendon suture which makes the repair bulkier than the remainder of the tendon and which may impede gliding later is 'bunching' as a result of the core suture being too tight. This can be eliminated almost completely in every case by suture of the back wall of the circumferential suture at this stage, before knotting the core suture. The core suture is then tightened and knotted using the back wall suture line as an indicator of adequate core suture tension to prevent bunching. The front part of the superficialis circumferential suture is then com-

pleted. When the superficialis tendon has been divided slightly closer to its insertion, it is in two parts and each needs be sutured independently. Occasionally, each has sufficient substance to hold a core and a circumferential suture, inserted as described above, but often the tendon has thinned to a pair of thin flat ribbons which will not take a core suture. Horizontal mattress sutures of the gauge used for the circumferential suture, or one size larger, with the knots tied laterally outside the sheath are a practical alternative. The suturing procedure is then carried out for the profundus tendon. At this stage, it is necessary to ensure that the repairs, inevitably wider than the original tendon, will move through a full range of flexion without catching on edges of the adjacent pulleys. This is tested by passive movement of the finger. If the tendons have been cut in extension, the repair will only have to move proximally. It may be necessary to release, or 'vent', the sheath laterally at the distal edge of the pulley immediately proximal to the repair. With the injury just proximal to the PIP joint crease, this 'venting' involves loss of a small part of the total length of the A2 pulley. When the injury is in zone 1, this may require partial, or complete, venting of the A4 pulley. The sheath is then laid back without suturing, the tourniquet released and haemostasis achieved. It is generally quicker to close the skin in a bloodless field so the tourniquet is re-applied and the skin sutured.

5. **If the tendons have been cut in finger flexion**, the cut ends will be distal to the wound of the sheath with the finger in extension and will only become apparent at this wound on flexion of the finger. As the practicalities of performing repair require that the finger be straight, or nearly straight, the sheath needs be opened at the level of the distal tendon ends in the extended finger position. Therefore, the Bruner skin incision is extended distally until the distal tendon ends are seen inside the sheath. The sheath is opened transversely, then windowed further by lateral longitudinal incisions, as described above. Because the repairs of the two tendons will be at different levels, this sheath opening is often longer than is necessary to repair tendons divided in finger extension. This mostly involves defunctioning of part of the C1 – A3 – C2 part of the pulley system. It is advisable to try to retain as much of this as possible in case the A4 pulley has to be fully vented to achieve full passive mobilization of the repair (see below): distal bowstringing of the profundus tendon will occur if there is no sheath between the distal edge of the A2 pulley and the proximal edge of the A5 pulley. The proximal tendon ends are delivered in the manner described above into the original wound of the tendon sheath. The core sutures are now placed in the proximal tendon ends by passing the sutures through the sheath from the opening at the level of the distal ends so that these sutures can be used to pull the proximal tendon ends distally through the sheath between the original wound and the more distal opening. When the superficialis tendon repair will be deeper than the profundus repair, the superficialis is pulled through the sheath between the two sheath wounds and repaired first. The distal end of the superficialis tendon lies in the distal opening of the sheath and requires no further exposure. To repair the profundus tendon requires exposure of 0.75–1.0 cm of the distal tendon to achieve the lateral passes of the core suture. Where the repair is just proximal to the A4 pulley, this is difficult but can often be achieved by an assistant holding the finger with the PIP joint completely straight and the DIP joint in extreme flexion to deliver the distal end of the tendon from under the A4 pulley. However, sometimes part of the proximal part of the A4 pulley has to be vented laterally to achieve a good bite of the distal profundus tendon end. Because repair is often done with the finger in slight flexion, it is essential in such cases to test that the repair does not snag on the proximal edge of the A4 pulley after completion of the repair, when the DIP joint is fully straightened passively. If need be, further A4 venting laterally is carried out and, occasionally, the whole A4 pulley will have to be divided if the patient is to achieve full active DIP extension.

6. **Suture of the flexor tendons** is carried out by a variety of methods. Currently, we use two two-strand Kirchmayr/Kessler core sutures of 3/0 Prolene, or 4/0 for smaller tendons, in planes at right angles to each other [67], with the sutures tied using a single knot between the tendon ends, as described by Tajima, and a simple circumferential suture of 6/0 or 5/0 Prolene or Nylon for finger tendons. For all core sutures, we prefer to use Prolene, as opposed to braided, sutures as it can be pulled through the tendon more easily when inserted in the complicated manner of a Kessler suture. The memory of Prolene is not a particular problem at the gauges used for core sutures but is more of a nuisance at the finer gauges used for circumferential sutures and some of us prefer to use nylon for these for this reason. Our suture uses and configurations for repair of the FPL tendon have varied considerably because this tendon has been our tool for research into flexor tendon rupture during early mobilisation. Currently, the FPL tendon is repaired using the same suture materials but using two Kessler sutures inserted into the tendon ends at right angles to each other and with both knotted with a single knot between the tendon ends. The simpler of the two circumferential sutures described and illustrated in 1993 by Silfverskiöld and Andersson is then used to complete these repairs. In all digits, when the tendon division is so close to the distal phalanx that it is impossible to insert a Kessler suture into the distal tendon end, we attach the proximal tendon, through the distal stump and without excising this, to the bone of the distal phalanx. This is carried out by a technique which we described several years ago in which the two strands of the Kessler repair are passed up the lateral sides of the distal phalanx, then one strand is passed through a drill-hole in the tuft of the distal phalanx and knotted to the other strand, after exposure of the distal phalangeal tuft through a fish-mouth incision close to the nail at the tip of the digit. This technique of tendon to bone suture avoids the need for a button on the nail and is considerably cheaper than using bone tags.
7. **Immediate post-operative management** is by dorsal splinting the hand and the mid-forearm to just beyond the finger-tips. This splint is set with the wrist at 20° of flexion, the MCP joints at 40° of flexion and the interphalangeal joints straight. The hand is elevated overnight. The following morning, dressings on the palmar aspect of the hand and fingers are removed and mobilisation started. Where the luxury of thermoplastic splints is possible, this is more comfortable for 5 weeks than plaster of Paris and the thermoplastic splint is fitted as soon as possible. However, a thermoplastic splint is not essential. A plaster of Paris slab is more likely to last for 5 weeks if it is made with fifteen or more layers of plaster and is ribbed along its dorsal surface to achieve the same strengthening as corrugation of a roof. If the distal end of the slab, lying free beyond the palm and unbandaged to the fingers (to allow finger flexion) is wrapped in bandage, this will also help preserve the plaster for the full period of early active mobilisation.

References

1. Lexer E. Die Verwerthung der freien Sehnentransplantation. Arch Klin Chir. 1912;98:818–25.
2. Harmer TW. Tendon suture. Boston Med Surg J. 1917;177:808–10.
3. Kirchmayr L. Zur Technik der Sehnennaht. ZBL Chir. 1917;40:906–7.
4. Kessler I, Nissim F. Primary repair without immobilisation of flexor tendon division within the digital sheath. Acta Orthop Scand. 1969;40:587–601.
5. Lahey FH. A tendon suture which permits immediate motion. Boston Med Surg J. 1923;22:851–2.
6. Bunnell S. Repair of tendons in the fingers and description of two new instruments. Surg Gynecol Obstet. 1918;126:103–10.
7. Kleinert HE, Kutz JE, Ashbell T, Martinez E. Primary repair of lacerated flexor tendons in "No Man's Land". Proceedings, American Society for Surgery of the Hand. J Bone Joint Surg Am. 1967;49:577.
8. Young RES, Harmon JM. Repair of tendon injuries of the hand. Ann Surg. 1960;151:562–6.
9. Verdan C. Réparation primaire des fléchisseurs en dehors des coulisses osteo-fibreuses des doigts. In: Chirurgie Réparatrice et Fonctionnelle des Tendons de la Main. Paris: L'Expansion Scientifique Française; 1952. p. 174–6.

10. Verdan C. La réparation immédiate des tendons fléchisseurs dans le canal digital. Acta Orthop Belg. 1958;24(Supplement III):15–23.
11. Riccio M, Battiston B, Pajardi G, Corradi M, Passaretti U, Atzei A, Altissimi M, Vaienti L, Catalano F, Del Bene M, Fasolo P, Ceruso M, Luchetti R, Landi A, Study Group on Tendon Adhesion of Italian Society of Hand Surgery. Efficiency of Hyaloglide in the prevention of the recurrence of adhesions after tenolysis of flexor tendons in zone II: a randomized, controlled, multicentre clinical trial. J Hand Surg Eur. 2010;35:130–8.
12. Doyle JR, Blythe W. The finger flexor tendon sheath and pulleys: anatomy and reconstruction. In: Hunter JM, Schneider LH, editors. American Academy of Orthopaedic Surgeons symposium on tendon surgery in the hand. St. Louis: Mosby; 1975. p. 81–7.
13. Idler RS. Anatomy and biomechanics of the digital flexor tendons. Hand Clin. 1985;1:3–11.
14. Verdan CE, Michon J. Le traitement des plaies des tendons flechisseurs des doigts. Rev Chir Orthop. 1961;47:290–6 and 386.
15. Kleinert HE, Weiland AJ. Primary repair of flexor tendon lacerations in zone II. In: Verdan C, editor. Tendon surgery of the hand. Edinburgh: Churchill Livingstone; 1979. p. 71–5.
16. Tang JB. Flexor tendon repair in zone 2C. J Hand Surg Br. 1994;1994(19):72–5.
17. Moiemen NS, Elliot D. Early active mobilization of primary flexor tendon repairs in zone 1. J Hand Surg Br. 2000;25:78–84.
18. Barton NJ. Experimental study of optimal location of flexor tendon pulleys. Plast Reconstr Surg. 1969;43:125–9.
19. Urbaniak JD, Cahill JD, Mortenson RA. Tendon suturing methods: analysis of tensile strengths. In: Hunter JM, Schneider LH, editors. Symposium on tendon surgery. St Louis: Mosby; 1975. p. 70–80.
20. Mason ML, Allen HS. The rate of healing of tendons. An experimental study of tensile strength. Ann Surg. 1941;113:424–59.
21. Strickland JW. Flexor tendon surgery. Part 1: primary flexor tendon repair. J Hand Surg Br. 1989;14:261–72.
22. Savage R. In vitro studies of a new method of flexor tendon repair. J Hand Surg Br. 1985;10:135–41.
23. Savage R, Risitano G. Flexor tendon repair using a "six strand" method of repair and early active mobilisation. J Hand Surg Br. 1989;14:396–9.
24. Wade PJF, Wetherell RG, Amis AA. Flexor tendon repair: significant gain in strength from the Halsted peripheral suture technique. J Hand Surg Br. 1989;14:232–5.
25. Lin GT, An KN, Amadio PC, Cooney WP. Biomechanical studies of running suture for flexor tendon repair in dogs. J Hand Surg Am. 1988;13:553–8.
26. Kubota H, Aoki M, Pruitt DL, Manske PR. Mechanical properties of various circumferential tendon suture techniques. J Hand Surg Br. 1996;21:474–80.
27. Elliot D, Moiemen NS, Flemming AFS, Harris SB, Foster AJ. The rupture rate of acute flexor tendon repairs mobilized by the controlled active motion regimen. J Hand Surg Br. 1994;19:607–12.
28. Harris SB, Harris D, Foster AJ, Elliot D. The aetiology of acute rupture of flexor tendon repairs in zones 1 and 2 of the fingers during early mobilization. J Hand Surg Br. 1999;24:275–80.
29. Wong JK, Alyouha S, Kadler KE, Ferguson MW, McGrouther DA. The cell biology of suturing tendons. Matrix Biol. 2010;29:525–36.
30. Murphy FG. Repair of laceration of flexor pollicis longus tendon. J Bone Joint Surg Am. 1937;19:1121–3.
31. Silfverskiöld KL, Andersson CH. Two new methods of tendon repair: an in vitro evaluation of tensile strength and gap formation. J Hand Surg Am. 1993;18:58–65.
32. Sirotakova M, Elliot D. Early active mobilization of primary repairs of the flexor pollicis longus tendon. J Hand Surg Br. 1999;24:647–53.
33. Sirotakova M, Elliot D. Early active mobilization of primary repairs of the flexor pollicis longus tendon with two Kessler two strand core sutures and a strengthened circumferential suture. J Hand Surg Br. 2004;29:531–5.
34. Tsuge K, Ikuta Y, Matsuishi Y. Intra-tendinous tendon suture in the hand. Hand. 1975;7:250–5.
35. Tsuge K, Ikuta Y, Matsuishi Y. Repair of flexor tendons by intratendinous suture. J Hand Surg. 1977;2:436–40.
36. Tang JB, Shi D, Gu YQ, Chen JC, Zhou B. Double and multiple looped suture tendon repair. J Hand Surg Br. 1994;17:699–703.
37. Tang JB, Gu YT, Rice K, Chen F, Pan CZ. Evaluation of four methods of flexor tendon repair for postoperative active mobilisation. Plast Reconstr Surg. 2001;107:742–9.
38. Giesen T, Sirotakova M, Copsey AJ, Elliot D. Flexor pollicis longus primary repair: further experience with the tang technique and controlled active mobilization. J Hand Surg Eur. 2009;34:758–61.
39. Savage R. The mechanical effect of partial resection of the digital fibrous flexor sheath. J Hand Surg Br. 1990;15:435–42.
40. Tomaino M, Mitsionis G, Basitidas J, Grewal R, Pfaeffle J. The effect of partial excision of the A2 and A4 pulleys on the biomechanics of finger flexion. J Hand Surg Br. 1998;23:50–2.
41. Mitsionis G, Bastidas JA, Grewal R, Pfaeffle HJ, Fischer KJ, Tomaino MM. Feasibility of partial A2 and A4 pulley excision: effect on finger flexor tendon biomechanics. J Hand Surg Am. 1999;24:310–4.
42. Franko OI, Lee NM, Finneran JJ, Shillito MC, Meunier MJ, Abrams RA, Lieber RL. Quantification of partial or complete A4 pulley release with FDP repair in cadaveric tendons. J Hand Surg Am. 2011;36:439–45.
43. Kwai Ben I, Elliot D. "Venting" or partial lateral release of the A2 and A4 pulleys after repair of zone 2 flexor tendon injuries. J Hand Surg Br. 1998;23:649–54.

44. Mason ML, Shearon CG. The process of tendon repair. Arch Surg. 1932;25:613–92.
45. Mason ML. Primary and secondary tendon suture. A discussion of the significance of technique in tendon surgery. Surg Gynecol Obstet. 1940;70:392–402.
46. Mason ML, Allen HS. The rate of tendon healing. Ann Surg. 1941;112:424–59.
47. Gelberman RH, Amilf D, Gonsalves M, Woo S, Akeson WH. The influence of protected passive mobilisation on the healing of flexor tendons: a biochemical and microangiographic study. Hand. 1981;13:120–8.
48. McGrouther DA, Ahmed MR. Flexor tendon excursions in 'No-Man's Land'. Hand. 1981;13:129–41.
49. Slattery PG, McGrouther DA. A modified Kleinert controlled mobilisation splint following flexor tendon repair. J Hand Surg Br. 1984;9:217–8.
50. Silfverskiöld KL, May EJ. Flexor tendon repair in zone II with a new suture technique and an early mobilization program combining passive and active flexion. J Hand Surg Am. 1994;19:53–60.
51. Chow JA, Thomes LJ, Dovelle S, Milnor WH, Seyfer AE, Smith AC. A combined regimen of controlled motion following flexor tendon repair in "no man's land". Plast Reconstr Surg. 1987;79:447–53.
52. Duran RH, Houser RG. Controlled passive motion following flexor tendon repairs in zones II and III. In: Hunter JM, Schneider LH, editors. American Academy of Orthopaedic Surgeons symposium on flexor tendon surgery in the hand. St. Louis: Mosby; 1975. p. 105–14.
53. Strickland JW, Glogovac SV. Digital function following flexor tendon repair in Zone II: a comparison of immobilisation and controlled passive motion techniques. J Hand Surg. 1980;5:537–43.
54. Small JO, Brennen MD, Colville J. Early active mobilisation following flexor tendon repair in zone 2. J Hand Surg Br. 1989;14:383–91.
55. Cullen KW, Tolhurst P, Lang D, Page RE. Flexor tendon repair in zone 2 followed by controlled active mobilisation. J Hand Surg Br. 1989;14:392–5.
56. Bainbridge LC, Robertson C, Gillies D, Elliot D. A comparison of post-operative mobilization of flexor tendon repairs with "passive flexion - active extension" and "controlled active motion" techniques. J Hand Surg Br. 1994;19:517–21.
57. Baktir A, Türk CY, Kabak S, Sahin V, Kardas Y. Flexor tendon repair in zone 2 followed by early active mobilization. J Hand Surg Br. 1996;1996(21):624–8.
58. Savage R. The influence of wrist position on the minimum force required for active movement of the interphalangeal joints. J Hand Surg Br. 1988;3:262–8.
59. Dowd MB, Figus A, Harris SB, Southgate CM, Foster AJ, Elliot D. The results of immediate re-repair of zone 1 and 2 primary flexor tendon repairs which rupture. J Hand Surg Br. 2006;31:507–13.
60. Kulkarni M, Harris SB, Elliot D. The significance of extensor tendon tethering and dorsal joint capsule tightening after injury to the hand. J Hand Surg Br. 2006;31:52–60.
61. Elliot D, Harris SB. The assessment of flexor tendon function after primary tendon repair. Review. Hand Clin. 2003;19:495–503.
62. Stefanich RJ, Putnam MD, Peimer CA, Sherwin FS. Flexor tendon lacerations in zone V. J Hand Surg Am. 1992;17:284–91.
63. Yii NW, Urban M, Elliot D. A prospective study of flexor tendon repair in zone 5. J Hand Surg Br. 1988;23:642–8.
64. Gerbino PG, Saldana MJ, Westerbeck P, Schacherer TG. Complications experienced in the rehabilitation of zone 1 flexor tendon injuries with dynamic splinting. J Hand Surg Am. 1991;16:680–6.
65. Evans RB. A study of the zone 1 flexor tendon injury and implications for treatment. J Hand Ther. 1990;3:133–48.
66. Boyes JH, Stark HH. Flexor tendon grafts in the fingers and thumb. A study of factors influencing results in 1000 cases. J Bone Joint Surg Am. 1971;53:1332–42.
67. Smith AM, Evans DM. Biomechanical assessment of a new type of flexor tendon repair. J Hand Surg Br. 2001;26(3):217–9.

Injuries: Tendon – Extensor

4

Simon L. Knight

Keywords
Extensor tendon • Extensor tendon repair • Boutonniere • Carducci's test • Mallet finger • Elson's test • Boye's test • Sagittal band

Introduction

Extensor tendon injuries are common. They are diverse in nature. Their complexity is often underestimated and it is difficult to get consistently good results after their repair.

In this chapter I will discuss the surgical anatomy of extensor tendons and the clinical diagnosis of tendon injury and pitfalls of diagnosis. Closed injures to extensors include: mallet finger, Boutonnière injury and Sagittal band rupture. Open extensor tendon injuries are classified according to the level of injury. The principles of surgical treatment of open extensor tendon injuries at different sites will be detailed and the rehabilitation and results of these repaired extensor tendons will be discussed.

S.L. Knight, FRCS
Department of Plastic Surgery,
Leeds General infirmary, Leeds, West Yorkshire, UK
e-mail: simon.l.knight@gmail.com

Anatomy

The general organisation of the extensors is into two groups. A superficial group, which takes origin from the common extensor origin of the lateral epicondyle and a deep group, which originates from the bones of the forearm and the interrosseus membrane. In the distal forearm, three of the deep group of muscles emerge on their way to the thumb (Abductor Pollicis Longus, Extensor Pollicis Brevis, Extensor Pollicis Longus) separating the superficial group into two groups of three muscles: the radial group (Brachioradialis, Extensor Carpi Radialis Longus, Extensor Carpi Radialis Brevis) and the posterior group (Extensor Digitorum Communis, Extensor Digiti Minimi, Extensor Carpi Ulnaris). The only remaining muscle, Extensor Indicis, stays deep all the way to its insertion into the index finger.

As the extensor tendons cross the wrist, they pass under the extensor retinaculum. This is a strap-like ligament, which takes origin from the anterolateral border of the radius just lateral to Pronator Quadratus and winds obliquely over the dorsum of the wrist to insert into the Triquetrum

I.A. Trail, A.N.M. Fleming (eds.), *Disorders of the Hand: Volume 1: Hand Injuries*,
DOI 10.1007/978-1-4471-6554-5_4, © Springer-Verlag London 2015

Table 4.1 Extensor retinaculum compartments

1	Abductor Pollicis Longus
	Extensor Pollicis Brevis
2	Extensor Carpi Radialis Longus
	Extensor Carpi Radialis Brevis
3	Extensor Pollicis Longus
4	Extensor Digitorum Communis
	Extensor Indicis
	Posterior Interosseus Nerve
5	Extensor Digiti Minimi
6	Extensor Carpi Ulnaris

and Pisiform. It is important to note that there is no direct attachment of this ligament to the Ulna, allowing the tension in the ligament to remain constant throughout supination and pronation. The ligament prevents bowstringing of the extensor tendons when the wrist is extended.

From the deep surface of the extensor retinaculum a series of fibrous septa pass deeply, dividing the system into six compartments (Table 4.1), which prevent lateral subluxation of the extensor tendons over the convex surface of the wrist. The compartments are numbered one to six from the radial to the ulnar side. The first compartment contains the Abductor Pollicis Longus and the Extensor Pollicis Brevis. The second compartment contains the Extensor Carpi Radialis Longus and Extensor Carpi Radialis Brevis tendons. The third compartment contains the Extensor Pollicis Longus tendon as it winds around Lister's tubercle. The fourth compartment is the largest compartment; it sits over the distal radius and contains the Extensor Digitorum Communis tendons, the Extensor Indicis tendon and the terminal branch of the Posterior Interrosseus nerve. The fifth compartment lies over the distal radio ulnar joint and contains the Extensor Digiti Minimi tendon. The sixth compartment contains the Extensor Carpi Ulnaris tendon. Synovial sheaths surround the tendons of each compartment.

There is a variable arrangement of the extensors on the dorsum of the hand [1]. The most common arrangement is a single Extensor Digitorum Communis tendon to each of the fingers. The index and little fingers have an additional tendon, the Extensor Indicis and the Extensor Digiti Minimi, allowing the border digits a greater degree of independent control than the middle and ring fingers. The Extensor Digiti Minimi usually consists of two parallel tendons; it is the predominant extensor to the little finger. In some cases the Extensor Digitorum Communis tendon to the little finger is absent and replaced by a junctura from the ring finger.

Proximal to the Metacarpophalangeal joints Juncturae Tendinum provide inter connections between the Extensor Digitorum Communis tendons [2]. These are variable. The most common arrangement is a strong junctura between the little and ring finger tendons, often replacing the tendon to the little finger, as explained above. There is usually a junctura from the ring to the middle finger tendon, and finally there is often a flimsy transverse junctura between the Extensor Digitorum Communis tendons of the middle and index fingers, which passes superficial to the Extensor Indicis tendon.

Over the proximal phalanx the extensor tendon trifurcates into a central slip, that inserts into the base of the middle phalanx, and two lateral bands that pass along either side of the middle phalanx. The lateral bands coalesce over the distal interphalangeal joint and insert into the distal phalanx as a single tendon. The lateral band and the central slip each receive contributions from the interroseus and lumbrical muscles to complete the extensor expansion.

In contrast to the flexor tendon system, which is safely cocooned within the concavity of the transverse and longitudinal arches of the hand, the extensor apparatus is precariously balanced on the convex surface of these arches, and the working position of the tendons is maintained by a sophisticated series of ligaments. At the wrist, the compartments of the extensor retinaculum prevent lateral subluxation. The extensors to the fingers are stabilised over the dorsum of the hand by the Juncturae Tendinum. Over the metacarpal head the extensor is maintained in a central position by the sagittal bands. Across the back of the middle phalanx the Triangular Ligament extends between the two converging lateral bands and prevents their volar subluxation. The Transverse Retinacular Ligament extends from the side of

the proximal interphalangeal joint to the lateral bands and prevents their dorsal subluxation. The Oblique Retinacular Ligament arises from the flexor sheath and the proximal phalanx and passes obliquely dorsally and distally, blending with the lateral bands just before their insertion. It becomes tight as the proximal interphalangeal joint extends, and stabilises the final insertion of the extensor tendon [3].

The extensor tendon in the finger is a complicated and incompletely understood structure. It is beyond the scope of this chapter to dwell on the intricacies of this extraordinary mechanism. However, the principle of isometry [4] must be introduced. The relative lengths of the lateral bands and the central slip and their position relative to each other throughout their excursion are crucial to the coordinated function of the interphalangeal joints. An injury to the central slip will affect the function of the lateral bands because their exact relationship to each other is disturbed and vice versa. The disruption of isometry is responsible for the swan neck deformity following a mallet injury and the boutonnière deformity. The loss of isometry is responsible for the early physical signs following injuries to this region and the restoration of isometry between the central slip and the lateral band is the goal of surgical repair of these tendons and their rehabilitation.

Brachioradialis and Extensor Carpi Radialis Longus are supplied by the Radial nerve; Extensor Carpi Radialis Brevis is supplied by the Posterior Interrosseus nerve before the nerve pierces the Supinator muscle; the remaining muscles are supplied by the Posterior Interosseous nerve after it emerges from the Supinator muscle.

The blood supply of the proximal tendons is via the adjacent muscle. Within the synovial reflections, under the extensor retinaculum, there is a blood supply passing through the synovium in a form of a mesentery. Over the dorsal aspect of the hand and fingers, the tendons are richly supplied by blood vessels from the adjacent tissues extending into the paratenon. The Extensor Pollicis Longus tendon has a poor blood supply around the pulley, which is formed by the combination of the Extensor Retinaculum and Lister's tubercle, and is particularly vulnerable to attrition ruptures at this point. The insertion of the extensors into the distal phalanx has a particularly poor blood supply [5], accounting for its vulnerability to injury and the prolonged healing time of the tendon at this site.

Table 4.2 Extensor tendon excursions (mm) per 10° of joint motion

Joint	Thumb	Index	Middle	Ring	Little
Wrist	1.5	2.0	2.0	2.0	1.4
MCP	1.2	1.5	1.5	1.5	1.0
PIP	0.9	0.8	0.8	0.8	0.6
DIP	–	0.6	0.8	0.6	0.6

After Elliot and McGrouther [36]

The gliding surfaces of the extensor tendons are maintained at the wrist by the synovial reflections which separate the tendon from the unyielding tunnels of the surrounding extensor retinaculum. Over the dorsum of the hand and the fingers, there is a combination of paratenon and fat separating the tendons from the underlying phalanges and metacarpals, and the overlying skin. The relatively small excursion [6] of the distal tendons (Table 4.2) and their proximity to the underlying phalanges commonly causes tendon repairs within the fingers to get stuck.

Diagnosis

The diagnosis of tendon injury is usually straightforward. Acute division of a tendon results in lack of function, which is usually obvious to both the patient and to the examiner. With practise, the diagnosis can usually be made from the history, the site of the wound, and the posture of the hand. The relaxed hand assumes a characteristic position, which is governed by the balancing tonic forces of the flexors and the extensors. With the palm uppermost, the wrist usually lies in a position of 30° of dorsiflexion, and the thumb and the fingers are partially flexed. The fingertips lie on the same curve as they gently cascade into increasing flexion from the index to the little finger. Interruption of this cascade signifies a tendon injury.

These general observations should be followed by specific examination of the tendons to avoid common pitfalls in diagnosis (Table 4.3).

The Abductor Pollicis Longus and the Extensor Pollicis Brevis can be palpated on the radial side of the anatomical snuffbox when the thumb is held in extension. Specific distinction between injuries to these two tendons is usually academic, but is possible: Abductor Pollicis Longus is tested by abducting the first metacarpal against resistance (Fig. 4.1); Extensor Pollicis Brevis is tested by trying to extend the metacarpophalangeal joint of the adducted thumb against resistance (Fig. 4.2).

Table 4.3 Pitfalls of diagnosis

Injury	Misdiagnosis	Diagnosis
Rupture of EPL	Mallet thumb	Absent EPL at wrist
Division of EDC on dorsum of hand	Missed diagnosis. Intact Junctura allow continuing extension	Examine EDC to each finger separately to detect weakness
Central Slip Rupture	Volar Plate Injury	Hyperextension of DIP joint. Positive Carducci, Elson and Boyes tests
Division of ECRL and ECRB	Missed diagnosis. (EDC is weak wrist extensor)	Test wrist extension with fist clenched (Removes EDC action)
Subluxation of Extensor at MCP joint	EDC tendon rupture	Restoration of active extension when MCP joint passively extended.
Posterior Interrosseus Nerve Injury	Divided Extensor tendons in forearm	Pattern of involvement with maintenance of Radial wrist extension

On the ulnar side of the snuffbox, the Extensor Pollicis Longus can be palpated. This tendon may be tested by actively extending the interphalangeal joint of the thumb against resistance. Injuries proximal to the metacarpophalangeal joint of the thumb may give rise to diagnostic confusion, as it may still be possible to extend the interphalangeal joint weakly, using the intrinsic muscles. An excellent test for determining the integrity of the Extensor Pollicis Longus in this situation is to attempt to raise the thumb in a dorsal direction (Fig. 4.3). This is the only tendon that is able to pull the thumb dorsally out of the plane of the hand.

Extending each of the fingers against resistance tests the Extensor Digitorum Communis tendons (Fig. 4.4). Injury of one of the extensor tendons immediately proximal to a Junctura Tendinum may result in only a few degrees loss

Fig. 4.1 Abductor Pollicis Longus is tested by abducting the 1st metacarpal against resistance and palpating the intact tendon in the anatomical snuffbox

Fig. 4.2 Extensor Pollicis Brevis is tested by trying to extend the MCP joint of the adducted thumb against resistance

Fig. 4.3 Extensor Pollicis Longus is tested by trying to raise the thumb dorsally

Fig. 4.4 Extensor Digitorum Communis Tendons are tested by extending each of the fingers against resistance

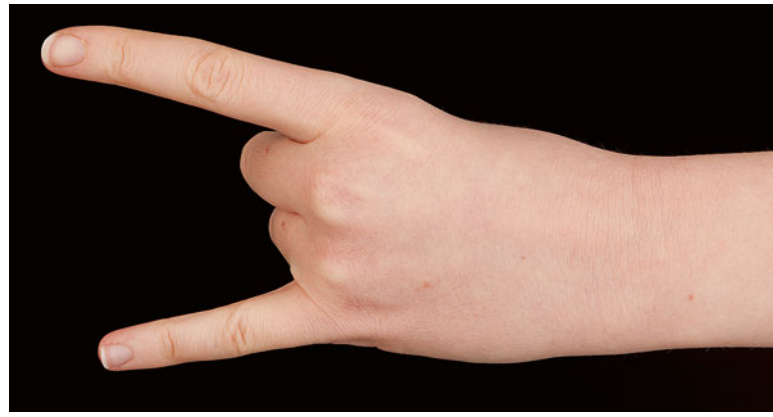


Fig. 4.5 Extensor Indicis and Extensor Digiti Minimi allow independent extension of the border digits

Fig. 4.6 Extensor Carpi Radialis Longus and Brevis can be palpated on the radial side of the wrist with the fingers tightly clenched

of active extension, and the diagnosis may be overlooked unless each finger is examined separately.

The little and the index fingers each have an extra extensor (Extensor Indicis and Extensor Digiti Minimi) which allows them to extend independently (Fig. 4.5).

The Extensor Carpi Radialis Longus and Extensor Carpi Radialis Brevis, may be palpated on the radial side of the wrist, whilst dorsiflexing against resistance. It is important to perform this test with the fingers tightly flexed (Fig. 4.6), in order to remove the secondary effect on wrist extension by the Extensor Digitorum Communis, which may mask an injury to the wrist extensors if the test is performed with the fingers extended (Fig. 4.7).

Extensor Carpi Ulnaris can be palpated on the dorsum of the wrist immediately distal to the head of the ulna. The tendon sits in a groove on the Ulna head and its position relative to the hand changes as the carpus rotates around the Ulna on supination and pronation. In full supination the tendon sits on the posterior surface of the wrist and its prime function is that of dorsiflexion. In full pronation the tendon sits on the ulnar side of the wrist and acts as an ulnar deviator. Examination is usually performed with the hand in a pronated position. The integrity of the tendon can be confirmed by palpating the tendon whilst deviating the wrist in an ulnar direction against resistance (Fig. 4.8).

One of the most difficult diagnoses to establish is division of the central slip of the extensor

Fig. 4.7 A common pitfall. Wrist extension examined with the fingers extended. Extensor Digitorum Communis acts as a wrist extensor and camouflages dysfunction of the primary wrist extensors

Fig. 4.8 Extensor Carpi Ulnaris tested by ulnar deviation of the wrist against resistance

tendon. The classical Boutonniere shape of the finger does not develop immediately. Before this happens, the signs are subtle. There are, however, three useful clinical tests.

Carducci's test [7] is a test whereby the extensor tendon is tightened by simultaneously flexing the wrist and the metacarpophalangeal joint. When the central slip is intact, the proximal interphalangeal joint automatically extends (Fig. 4.9). Failure to do so suggests that the extensor mechanism has been divided. This is a useful test, and can be performed in anaesthetised, inebriated or uncooperative patients.

Elson's test [8], by contrast, does require the co-operation of the patient. It is a sophisticated test and has two components. The first component of the test looks at the effect of the division of the central slip on extension of the proximal interphalangeal joint. The second component of the test looks at the secondary effects of the injury on the distal interphalangeal joint. To perform the test, the proximal interphalangeal joint is bent to a right angle. It is possible to do this in mid-air or, alternatively, the end of a table can be used. An attempt is then made to extend the proximal interphalangeal joint against the resistance of the examiner's finger (Fig. 4.10). Failure to extend the joint confirms that the central slip is probably divided. When a patient with an intact central slip is examined in this way the distal

Fig. 4.9 Carducci's Test of central slip integrity. Simultaneous flexion of the wrist and MCP joint causes the PIP joint to extend. A lag of 15° or more indicates central slip disruption

interphalangeal joint is flail and it is not possible to extend it. By contrast, when the central slip has been divided, proximal retraction of the unrestrained extensor tendon tightens the lateral bands, and extension at the distal interphalangeal joint occurs. Elson's test is positive when there is failure to actively extend the proximal interphalangeal joint, and simultaneous hyperextension at the distal interphalangeal joint.

Boyes' test [9] is a third test, which is sometimes used to help with this diagnosis. This test consists of holding the proximal interphalangeal joint in extension and asking the patient to flex the distal interphalangeal joint. It is normally possible to flex to about 60° (Fig. 4.11). In central slip disruption the divided extensor tendon retracts proximally, tightens the lateral bands and prevents flexion of the distal interphalangeal joint. Conventional teaching suggests that this test is unreliable immediately after the injury and only becomes positive a few days later when the extensor has retracted proximally. Complete division of the central slip will, however, produce an immediate change in isometry that is clinically detectable. The test is modified by passively flexing the distal interphalangeal joint (rather than actively flexing it) and comparing the increased force required to flex the distal interphalangeal joint of the injured finger compared with the relative ease of flexing the uninjured fingers. The signs are subtle but are reliably present immediately after injury.

Fig. 4.10 Elson's Test. The PIP joint is bent to a right angle and an attempt is made to extend against resistance. When the central slip is intact it is possible to extend the PIP joint and the DIP joint sits in a semi-flexed position as illustrated. When the central slip is disrupted the PIP joint cannot be extended and the DIP joint hyperextends

Fig. 4.11 Boyes' test. An attempt is made to flex the DIP joint with the PIP joint held in extension. It is normally possible to flex the DIP joint as shown. When there has been disruption of the central slip, tightening of the lateral bands prevents flexion at the DIP joint

Closed Mallet Finger

Boyes first coined the term mallet injury [10]. The derivation of the term is obscure. It is one of the most common tendon injuries in the hand and describes the drooping fingertip caused by rupture of the extensor tendon insertion (Fig. 4.12a). It is an avulsion injury and is caused by a sudden flexing force on the tip of the finger, whilst actively extending the interphalangeal joints. Occasionally, a portion of the distal phalanx may be avulsed with the insertion of the tendon (Fig. 4.12b), which is visible on X-ray. With this mechanism of injury the collateral ligaments of the distal interphalangeal joint remain attached to the remainder of the distal phalanx and the distal interphalangeal joint does not sublux.

The "Baseball finger" (which is often classified as a form of Mallet finger) has a completely different mechanism of injury. It occurs when a hard object, such as a cricket ball (or baseball), hits the end of an extended finger. The articular surface of the distal phalanx, rather than the extensor tendon insertion, bears the brunt of the injury and an oblique fracture through the base of the distal phalanx occurs, causing the distal phalanx to sublux in a volar direction (Fig. 4.12c). Because the distal fragment is dorsally angulated the fingertip fails to droop in the manner of the true mallet finger, giving a different clinical appearance.

The diagnosis of a mallet injury is usually obvious. Diagnostic confusion may occur following spontaneous rupture of Extensor Pollicis Longus, which also presents with a drooping thumb tip. In any case of a suspected Mallet thumb, the integrity of the proximal Extensor Pollicis Longus should be checked to avoid confusing the two diagnoses.

Doyle [11] has classified the mallet injury into four types (Table 4.4). Type I injuries are closed; type II injuries are open; type III injuries are associated with loss of skin and tendon substance; type IV injuries involve a large bony fragment.

The majority of injuries fall into the type I category, and are treated conservatively by splinting

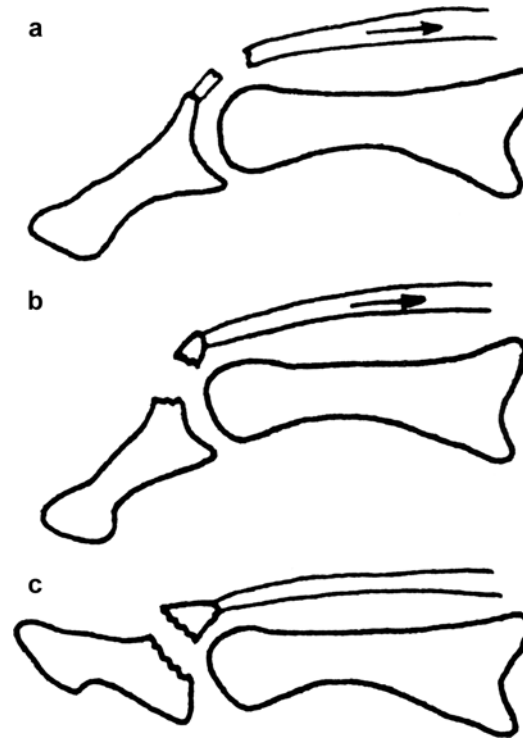


Fig. 4.12 (**a**) Mallet injury (**b**) Mallet fracture (**c**) Baseball finger

Table 4.4 Doyle [11] classification mallet finger

Type 1	Closed. Loss of tendon continuity +/− small avulsion fracture
Type 2	Laceration at or just proximal to DIPJ
Type 3	Deep abrasion with loss of skin, and tendon.
Type 4	Large mallet fractures: (a) Trans-epiphysial plate fracture (b) Hyperflexion injury with fracture articular surface of 20–50 % (c) Hyperextension injury with fracture of articular surface of more than 50 % with volar subluxation

the distal interphalangeal joint in extension. The splint may be custom-made or, alternatively, a polypropylene Stack splint [12] may be used. There is a temptation to slightly hyperextend the distal interphalangeal joint to allow closer apposition of the avulsed tendon insertion to the distal phalanx. This should be resisted. Hyperextension of the distal interphalangeal joint causes blanching of the skin over the back of the joint. Splintage in this position is associated with a high incidence

of dorsal ulceration of the skin [13]. The joint should be maintained in a comfortably extended (but not hyperextended) position. The splint needs to be worn for 6–8 weeks. It may be removed to wash the affected digit, but the distal interphalangeal joint should be supported at all times. Following the removal of the splint, many patients will develop an extensor lag of 10–15°. This is minimised by encouraging night splintage for a further 2 weeks. It has been observed that this extensor lag appears to improve over the next 6 months, as the scar tissue bridging the extensor defect contracts [14].

This is a common injury amongst sports players, who are always impatient to return to their activities quickly. The period of splintage may be shortened if the tension in the extensor insertion is reduced. Splinting the distal interphalangeal joint in extension and simultaneously splinting the proximal interphalangeal joint in flexion can achieve this. This uses the principle of isometry of the extensor mechanism to advantage (flexion of the proximal interphalangeal joint advances the central slip and slackens the lateral bands). Splintage of both joints is performed for 3 weeks, followed by 2 weeks' splintage of the distal interphalangeal joint alone [15]. This regimen requires close supervision to avoid a flexion contracture of the proximal interphalangeal joint.

When the mallet fracture is associated with subluxation of the distal phalanx, operative treatment is recommended. The subluxed distal phalanx is reduced and maintained in an extended position with a Kirschner wire. The Kirschner wire can be inserted longitudinally (which is technically easier) or an oblique Kirschner wire can be used. The potential advantage of the oblique Kirschner wire is that it is easier to remove should the wire fracture during the convalescence.

All other fractures, no matter how large the fragment, may be treated conservatively with a splint. Operative treatment has been recommended for the type IV mallet injuries, in which the avulsed fragment contains more than one third of the articular surface [16]. It is argued that the accurate reduction of the articular surface may prevent secondary arthritis. Surgical treatment of this condition is deceptively difficult. A number of techniques have been used, which include Kirschner wiring, stainless steel pull out sutures and screw fixation. Surgical outcomes in these cases are often poor [17] and there is no clear evidence that the risk of osteoarthritis is reduced [18]. In the author's practice, all mallet fractures, except those, which have subluxed, are treated conservatively with a splint no matter how big the fracture.

Late presentations of mallet fingers can also be treated conservatively with success [19]. Mallet injuries may present a month after the injury. It is still worth putting the patient through a regimen of splintage, and an improvement in the deformity can be anticipated.

In those cases that do not respond to treatment, the mallet deformity will persist, and, if the fingers are flexible, there is a tendency for the finger to adopt a swan neck deformity. Treatment of the chronic mallet deformity can be performed by either plicating [20] or grafting [21] the extensor tendon insertion. Those patients with PIP joint hyperextension can be treated with a central slip tenotomy [22]; it should be emphasised that the mallet deformity should have been present for at least 6 months, to ensure that the scar bridging the defect in the extensor insertion is mature, before performing this tenotomy.

Closed Rupture of the Central Slip (Boutonnière Injury)

The significance of this injury is that it is difficult to diagnose acutely and the consequences of the missed diagnosis are serious, with the development of the Boutonniere deformity. The term boutonniere is derived from the French "buttonhole" and aptly describes the process of the proximal interphalangeal joint (no longer constrained by the central slip of the extensor tendon) passing through the buttonhole [23] created by the two lateral bands (Fig. 4.13). The lateral bands then pass volar to the axis of the proximal interphalangeal joint and become flexors of this joint. The proximal migration of the unconstrained extensor

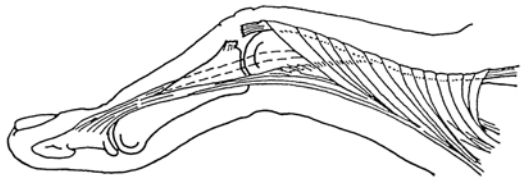


Fig. 4.13 Boutonniere injury. The central slip ruptures allowing the PIP joint to pass through the "button–hole" created by the intact lateral bands

apparatus tightens the lateral bands resulting in hyperextension of the distal interphalangeal joint and increasing flexion at the proximal interphalangeal joint [24]. The characteristic zig- zag deformity develops.

The history is usually one of stubbing the finger. The finger is swollen, and, at this stage, the classical Boutonniere shape may be absent. An X-ray may be normal, but occasionally demonstrates a fleck of avulsed insertion over the dorsal aspect of the proximal interphalangeal joint. The Boutonniere shape is often visible on the lateral X-ray, before it is clinically obvious in the swollen finger. These injuries should be treated with a high degree of suspicion. The diagnosis is confirmed by performing Carducci's, Elson's and Boyes' tests (see above). If there is any doubt about the diagnosis, the finger should be splinted with the proximal interphalangeal joint in extension, and the patient re-examined a week later, when the swelling has reduced.

The basis of treatment of the closed Boutonniere deformity is to keep the proximal interphalangeal joint extended for 6 weeks, leaving the distal interphalangeal joint free to flex. A number of methods of splintage are available. A thermoplastic or aluminium splint can be used or a Kirschner wire can be placed across the proximal interphalangeal joint. In the author's practice, the splint of choice is a small cylindrical plaster cast. In the early stages, the finger is swollen, and the cast will have to be exchanged every few days, until the swelling has subsided. The cast should not impede movement at the distal interphalangeal joint, which should be actively and passively mobilised throughout the period of splintage.

If the patient presents late, when the deformity has already become established, it is necessary to perform serial casting of the proximal interphalangeal joint, in order to correct the flexion deformity, before a definitive cast is applied for a period of 6 weeks, with continuing flexion exercises to the distal interphalangeal joint.

On removal of the splint, the proximal interphalangeal joint is splinted at night for a further 2 weeks.

Sagittal Band Rupture

The extensor tendon is maintained in its position on the apogee of the metacarpal head by the sagittal band aponeurosis, which is attached to either side of the tendon. The band passes around the metacarpophalangeal joint to insert into the volar plate and into the deep transverse metacarpal ligament. Sagittal band rupture almost always occurs in the middle finger [25]. It is caused by sudden flexion and ulnar deviation of the finger, which results in a tear in the radial sagittal band. This results in subluxation of the extensor tendon into the valley between the metacarpal heads.

The diagnosis is usually straightforward, and the tendon can be seen to sublux to the ulnar side of the metacarpal head on flexion of the metacarpophalangeal joint. In the subluxed position, it may not be possible to actively extend the metacarpophalangeal joint and a misdiagnosis of a ruptured extensor tendon may be made. However, when the metacarpophalangeal joint is passively placed into an extended position, the subluxed tendon resumes its original position, and active extension of the joint can be maintained. Visible subluxation when the patient flexes again, confirms the diagnosis.

It is the author's usual practice to repair acute ruptures and to splint the metacarpophalangeal joint in extension for 4 weeks.

However, if the condition is recognised immediately, it is possible to treat it conservatively [26] by splinting the metacarpophalangeal joints in extension for 3 weeks. The splint is then removed and the hand re-examined. If the extensor tendon

remains appropriately located conservative treatment is continued and protected for another 3 weeks with neighbour strapping, to prevent ulnar deviation of the finger.

Operative intervention is indicated with late presentation of the subluxation, or if there is a failure of conservative treatment. It is usually possible to define the tear and to repair it directly. There are a number of procedures described, using flaps of Juncturae Tendinum [27] or flaps of the extensor tendon [28], to augment the repair when the remaining sagittal fibres are deficient. In the author's experience, these are almost never required. Even at late exploration, there are almost always residual sagittal fibres to allow a direct repair to be achieved. If there is any tightness on the ulnar side, this is treated identically to the subluxation in rheumatoid disease, by division of the ulnar sagittal fibres, to allow a tension-free centralisation of the extensor.

Classification of Extensor Tendon Injuries

Verdan and Kleinert [29] classified extensor tendon injuries as occurring at eight levels. To this original classification, an additional proximal level in the forearm has been added, to give nine levels (Table 4.5). Zone 1 occurs over the distal interphalangeal joint; Zone 2 over the middle phalanx; Zone 3 over the proximal interphalangeal joint; Zone 4 over the proximal phalanx; Zone 5 over the metacarpophalangeal joint; Zone 6 over the metacarpal; Zone 7 over the wrist and under the extensor retinaculum; Zone 8 over the distal forearm; Zone 9 through the muscles of the proximal forearm.

Because of its particular anatomy, the thumb is labelled differently. Zone T1 occurs over the interphalangeal joint; zone T2 over the proximal phalanx; zone T3 over the metacarpophalangeal joint; T4 over the metacarpal; zone T5 over the radial side of the wrist

As a simple mnemonic, it is relatively easy to remember the various levels; the odd numbers occur over joints, and the even numbers (except zone 9) are over bones.

Table 4.5 Classification of level of Extensor tendon injuries

Zone	Finger	Thumb (T)
1	DIP joint	IP joint
2	Middle Phalanx	Proximal Phalanx
3	PIP joint	MCP joint
4	Proximal Phalanx	Metacarpal
5	MCP joint	CMC joint
6	Metacarpal	
7	Wrist retinaculum	
8	Distal forearm	
9	Mid and proximal forearm	

Principles of Surgical Treatment for Open Injuries

Most open tendon injuries will require referral to a specialist. There is now little place for the inexperienced surgeon to attempt even simple repairs. Where specialist advice is not immediately available, the injury should be fully documented, and the wound cleaned, closed and dressed under local anaesthetic. The injured part should be splinted, and arrangements made for specialist treatment as soon as possible. Ideally, this delay should not exceed 24 h. Surgical treatment for most tendon injuries of the upper limb may be performed using a local or regional nerve block, although occasionally general anaesthesia may be preferred. The use of a tourniquet, loupe magnification and prophylactic antibiotics are recommended.

The surgical technique is important. Every effort should be made to ensure that the blood supply to the tendon ends is not damaged and care should be taken not to strip the paratenon from the surface of the tendon. Before placing any sutures in the tendon, it is essential to relax the tendon in question by extending the adjacent joints. Ideally, the severed tendon ends should gently abut each other prior to the suture being placed. No attempt should be made to haul the ends of the tendon together; the sutures will cut out, cause the tendon to fray, and make the repair progressively more difficult.

In contrast to flexor tendons, which remain cylindrical throughout most of their length, the

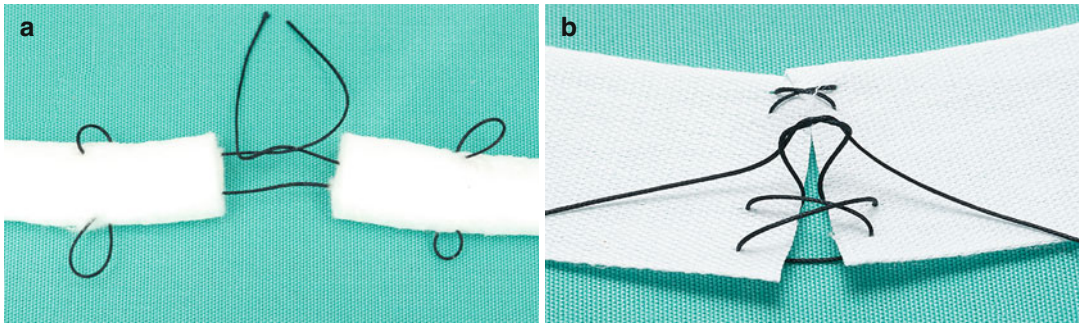


Fig. 4.14 (**a**) Modified Kessler core suture. (**b**) Figure of eight suture; knot is tied on the deep surface

cross-sectional geometry of the extensor tendon changes as it progresses distally. At the proximal end, the tendon has a cylindrical shape, which lends itself to repair using a core suture such as a modified Kessler suture or a Bunnell suture. Over the finger, however, the tendon flattens and a mattress or figure of eight sutures have traditionally been used in this position.

With the advent of active mobilisation techniques following extensor tendon repair, there has been considerable recent interest in the strength of the various suture techniques. Of the four traditional techniques, the Kessler suture and the Bunnell suture have been shown to be stronger than the mattress or figure of eight sutures [30]. More recently, there has been interest in the use of epitendinous sutures [31], such as the Silfverskiold suture or a running interlocking horizontal mattress suture [32], both of which have been shown to be stronger than the core suture techniques.

There is no one correct way of suturing an extensor tendon. The technique should allow apposition of the tendon ends, without causing a shortening of the tendon, and should have enough strength to prevent a gap developing if active rehabilitation is used. If possible, the knots should be placed deeply, and their bulk should be kept to a minimum, in order to prevent painful and palpable lumps on the dorsum of the hand.

Non-absorbable, mono-filament sutures such as prolene are recommended, and should be inserted using a round bodied needle, to prevent fraying of the tendon ends, and to avoid dividing previous passes of the suture, both of which may occur if a cutting needle is used. Braided, non-absorbable sutures, such as Ethibond, may also be used, but are more difficult to handle. Because of the occasional problems associated with the knots, and the bulk of the suture on the dorsum of the hand, some surgeons prefer long-acting, absorbable sutures, such as PDS.

The author's preference is to use a modified Kessler core suture (Fig. 4.14a) to repair the proximal extensor tendons (zones 5–8) and interrupted figure of eight sutures to repair the distal tendon injuries (zones 1–4). The figure of eight sutures are inserted in an inverted fashion so that the knot always sits on the deep surface of the tendon (Fig. 4.14b); this gives a flat repair with little bunching of the tendon and the sutures are impalpable. It has been shown that in experimental conditions the single figure of eight suture was not as strong as a single Kessler suture [33]. However, this does not mirror surgical practice, as multiple figure of eight sutures are usually required to complete the repair of these flat tendons, giving a strength that will be comparable or exceed the strength of a single Kessler suture.

A summary of the repair techniques by zone is suggested (Table 4.6). A more comprehensive summary of surgical repair in different zones is described below.

Table 4.6 Suggested scheme for repair of open tendon injuries

Zone	Repair	Rehabilitation
1	5.0 prolene figure of eight sutures	K wire DIPJ 4 weeks. 2 weeks external splint
2	Complete division: 5.0 prolene fig 8 suture	K wire DIPJ 4 weeks. 2 weeks external splint
	Single lateral band: leave unrepaired	Protected movement in neighbour strapping 4 weeks
3	Isolated Central slip division: 4.0 prolene fig 8 sutures	K wire PIP joint in extension 4 weeks. 2 weeks external splint. Actively mobilize DIP joint throughout.
	Central slip and lateral bands divided: 4.0 prolene fig 4.8 sutures	Immobilize PIP and DIP joints in extension 4 weeks.
4	Complete division: 4.0 prolene core suture to tendon, 4.0 prolene fig 8 to expansion	External splint. Immobilize or controlled active extension 4 weeks
	Incomplete division	Protected active movement in splint 4 weeks
5 6 7	4.0 prolene core suture	External splint. Immobilize or controlled active extension. 4 weeks
8	Tendon: 3.0 prolene core suture	External splint. Immobilize or controlled active extension. 4 weeks
	Musculotendinous junction: 3.0 prolene multiple fig 8 sutures	Immobilize in splint 4 weeks
9	3.0 PDS to muscle belly	Immobilize in splint 3 weeks

Open Zone 1 Injuries (Distal Interphalangeal Joint)

Division of the extensor tendon results in a mallet deformity (Doyle type 2). The extensor tendon is thin and diaphanous and is difficult to suture. Securing the distal interphalangeal joint in extension with a Kirschner wire prior to suturing the tendon facilitates the repair. The tendon is sutured with fine figure of eight sutures knotted on the deep surface of the tendon. The distal interphalangeal joint is held in extension with the Kirschner wire for 4 weeks and a further 2 weeks of external splintage before actively mobilising the finger.

Fig. 4.15 Zone 1 injury with skin and tendon loss in infant

Skin and tendon loss may complicate injuries in this zone (Doyle type 3). Children with small defects (Fig. 4.15) get remarkably good results with simple conservative treatment, by splinting the tip of the finger in extension for 6–8 weeks whilst the wound heals. In adults the results are poorer and almost all patients develop stiffness in the distal interphalangeal joint. The treatment will depend on the depth and extent of the soft tissue loss. Where there are some remnants of the extensor tendon and paratenon, the distal interphalangeal joint is held in extension with a Kirschner wire and a skin graft is placed over the unsutured tendon. Where there is a greater amount of soft tissue loss, flap reconstruction and tendon grafting will be required. The author's usual choice of flap in this area is a cross finger fascial/fat flap covered with a skin graft.

Open Zone 2 Injuries (Middle Phalanx)

If both of the lateral bands are divided, these are each repaired, and the distal interphalangeal joint is immobilised in extension with a Kirschner wire, as with a zone one injury.

Because of the cylindrical nature of the finger, it is possible to get unilateral injuries to the lateral bands. Provided that one lateral band is intact, it is permissible to leave the other band unrepaired and to undergo a programme of protected mobilisation of the finger by strapping it to the neighbouring finger for 4 weeks.

Open Zone 3 Injuries (Proximal Interphalangeal Joint)

Injuries that are confined to the central slip of the extensor tendon will result in a Boutonniere deformity if left untreated. The overlying skin laceration is often deceptively small (Fig. 4.16) and any sharp laceration in this area should be treated with suspicion and explored with care. The central slip is a substantial structure and is usually easy to repair. The repair is protected by immobilising the proximal interphalangeal joint in extension with an oblique Kirschner wire, carefully inserted to avoid tethering the lateral bands. The patient is specifically instructed to actively and passively flex the distal interpahalangeal joint throughout the convalescence to maintain the continuing excursion of the lateral bands. The Kirschner wire is removed at a month and substituted with a short external splint for another 2 weeks.

If both the central slip and the lateral bands are divided, these should each be repaired. The postoperative regimen differs because the distal interphalangeal joint also has to be immobilised, in order to fully protect the repair. This is performed with an external splint. The splint is retained for 4 weeks.

Where there is skin and tendon loss over the proximal interphalangeal joint, the skin defect is repaired with a local flap and the central slip is reconstructed with a tendon graft and bone anchor. This can be a free tendon graft of Palmaris Longus or a strip of extensor retinaculum. An ingenious alternative to a free graft is to use a distally based slip of the Flexor Digitorum Superficialis passed through a hole in the middle phalanx to emerge on the dorsal surface of the middle phalanx at the point of insertion of the central slip [34]. Other techniques have been described using remnants of local tendon: a turn over flap of the extensor in zone 4 [35] and medial transposition of parts of the lateral bands [36]

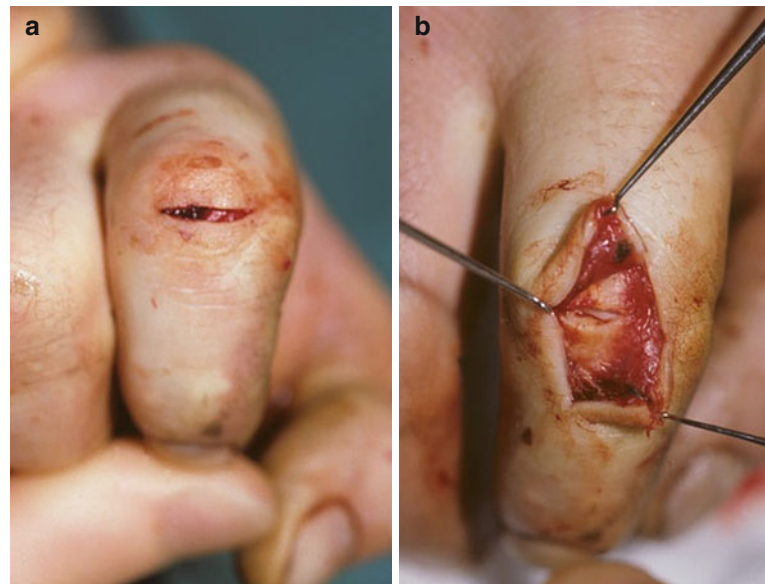


Fig. 4.16 Tiny cut over PIP joint (**a**) with complete division of central slip (**b**)

may each be used to reconstruct the central slip. The author's preference is to use a tendon graft.

Injuries in this zone are commonly complicated by postoperative stiffness in the proximal interphalangeal joint. This may be reduced by using an active postoperative rehabilitation regimen known as the short arc motion protocol [37]. This is a sophisticated and complicated regimen, whereby the patient is fitted with a static splint that holds the interphalangeal joints in extension and episodically exercises in 2 additional splints. One splint holds the proximal interphalangeal joint in extension while the distal interphalangeal joint is actively flexed and the other splint allows the proximal interphalangeal joint to actively flex to 30° and the distal interphalangeal joint to 25°. The patient actively extends the finger at each interphalangeal joint whilst using the exercise splints.

Open Zone 4 Injuries (Proximal Phalanx)

Complete injuries to the extensor tendon at this level are treated by direct repair of the extensor tendon. The thick central component of the extensor tendon will accommodate a core suture; elsewhere interrupted figure of eight sutures are used. The repair is protected with an external splint for 1 month.

Partial injuries are common, and the repair technique and rehabilitation are a matter of judgement. It is worthwhile flexing and extending all the joints in the finger, to see what happens to the partially divided tendon. If the ends remain in continuity throughout the excursion, it is possible to treat this conservatively by simply suturing the skin and moving the finger in a protected fashion. This avoids the period of stiffness that will accompany any formal repair and splintage.

Open Zone 5 Injuries (Metacarpophalangeal Joint)

At this level, the extensor tendon has a substantial central component, and complete division of this part of the tendon allows repair with a core suture. This however causes bunching of the tendon and the use of interrupted figure of eight sutures with buried knots may give a smoother and less prominent repair at this already prominent site. The repair is protected in a splint for a month.

A common injury at this level is the "fight bite", caused by punching an opponent in the mouth, and sustaining a partial injury to the extensor tendon on the teeth. These injuries should be treated with caution. The patients are rarely honest about the cause of the injury. The injury needs to be taken seriously because of the risk of infection of the metacarpo-phalangeal joint. Provided that the injury is treated within the first 24 h, before any signs of infection have developed, a copious lavage and antibiotic treatment will allow primary repair of the skin and tendon. If the treatment has been delayed, and cellulitis and infection are present, this should be treated with delayed primary repair, after an initial lavage, debridement and antibiotic treatment. These patients are always treated with strict immobilisation until the inflammation has settled. No attempt should be made to actively mobilise this group of patients post-operatively.

Open Zone 6 Injuries (Metacarpal)

The tendons in this zone are repaired with a central core suture, which may be augmented by a peripheral epitendinous suture for additional strength, if an active post-operative rehabilitation regimen is anticipated. The repair requires protection in a splint for a month. Injuries of less than 50 % of the breadth of the tendon should be ignored, and the patient immediately mobilised with protection.

Isolated injuries to the Extensor Indicis are rare. This diagnosis can usually be made preoperatively (Fig. 4.5), and the option of leaving the tendon unrepaired can be discussed with the patient.

The Extensor Digiti Minimi is usually the dominant extensor to the little finger, and should always be repaired if it has been completely divided.

Open Zone 7 Injuries (Wrist)

Injuries over the dorsal aspect of the wrist occur through the extensor retinaculum. The tendons often retract proximally, and may be more difficult to locate than injuries in other areas.

Injuries to the finger extensors should be repaired with a core suture. It may be necessary to excise part of the extensor retinaculum, to allow full excursion of the repaired tendons without snagging on the retinaculum. Complete division of the retinaculum should be avoided to prevent later bowstringing. The repairs are protected by splinting the wrist in 45° extension, the metacarpo-phalangeal joints in 45° of flexion and the interphalangeal joints extended.

Injuries to the wrist extensors should be repaired with core sutures, and the wrist splinted in 45° of extension, allowing the metacarpo-phalangeal joints a full range of movement.

Open Zone 8 (Distal Forearm)

This is actually two zones: a distal zone through the tendons and a more proximal zone through the musculotendinous junction. They require different treatment.

Injuries through the tendons are repaired with core sutures. The repairs are strong and all post-operative rehabilitation options are available.

By contrast, injuries occurring at the musculotendinous junction are sometimes difficult to repair. These are repaired by a series of figure of eight sutures through the tendon end and into the substance of the muscle, which usually contains fibrous septi. This is a relatively fragile repair, and most patients, after this type of repair, will require immobilisation, with the wrist and metacarpo-phalangeal joints extended, for approximately 1 month.

Open Zone 9 (Proximal Forearm)

Injuries in this zone occur through the extensor muscles. These will heal rapidly, and can be repaired with absorbable mattress or figure of eight sutures. These injuries need careful preoperative assessment and diligent exploration of the wound to exclude concomitant injury to the Posterior Interosseous nerve. Immobilising the wrist and hand for a month protects the repair.

Rehabilitation and Results

Traditionally, extensor tendons are repaired by approximating the ends of the tendon with a suture, and immobilising the hand in a manner that minimises the tension on the tendon repair. Whilst such immobilisation allows the ends of the tendon to remain in contact with each other throughout the healing phase, it also causes the tendon to remain in constant contact with the surrounding injured tissues. The end result is a healed tendon, which cannot glide; this is functionally useless. Intensive physiotherapy is then required to allow the tendon to regain its normal excursion.

The most comprehensive review of the results of extensor tendon repair was performed by Newport et al. [38] who analysed the results using Miller's rating [39] which was considered to be the most accurate and discriminating method of assessing outcome (Table 4.7). Most of the patients in this series were treated with immobilisation. The results were poor, with only half the patients achieving good or excellent results (Table 4.8). The results were poorer if there was an associated injury, such as a fracture, and the results of repair of the proximal tendons (zones 5–8) fared better than those of the

Table 4.7 Miller's rating of extensor tendon results

Grade	Range of movement
Excellent	Full flexion and extension
Good	<10° loss of extension <20° loss of flexion
Fair	11–45° loss of extension 21–45° loss of flexion
Poor	>45° loss of extension and flexion

Table 4.8 Long-term results of extensor tendon repair

Factor	% good/excellent results
Overall	52 %
Associated injury (e.g.fracture)	Without – 64 % With – 45 %
Zone of injury	Zones 1 and 2 – 38 % Zones 3 and 4 – 33 % Zone 5 – 85 % Zone 6 to 8 – 65 %

After Newport et al. [38]

distal tendons (zones 1–4). The average loss of flexion was greater than the average loss of extension.

In the wake of the success of the various active and passive regimens of rehabilitation in flexor tendon repair, similar methods have been popularised in extensor tendon repair to try to improve the results. Following their repair, extensor tendons can now be managed by immobilization, by dynamic splintage or by undergoing a programme of protected active mobilisation (Fig. 4.17).

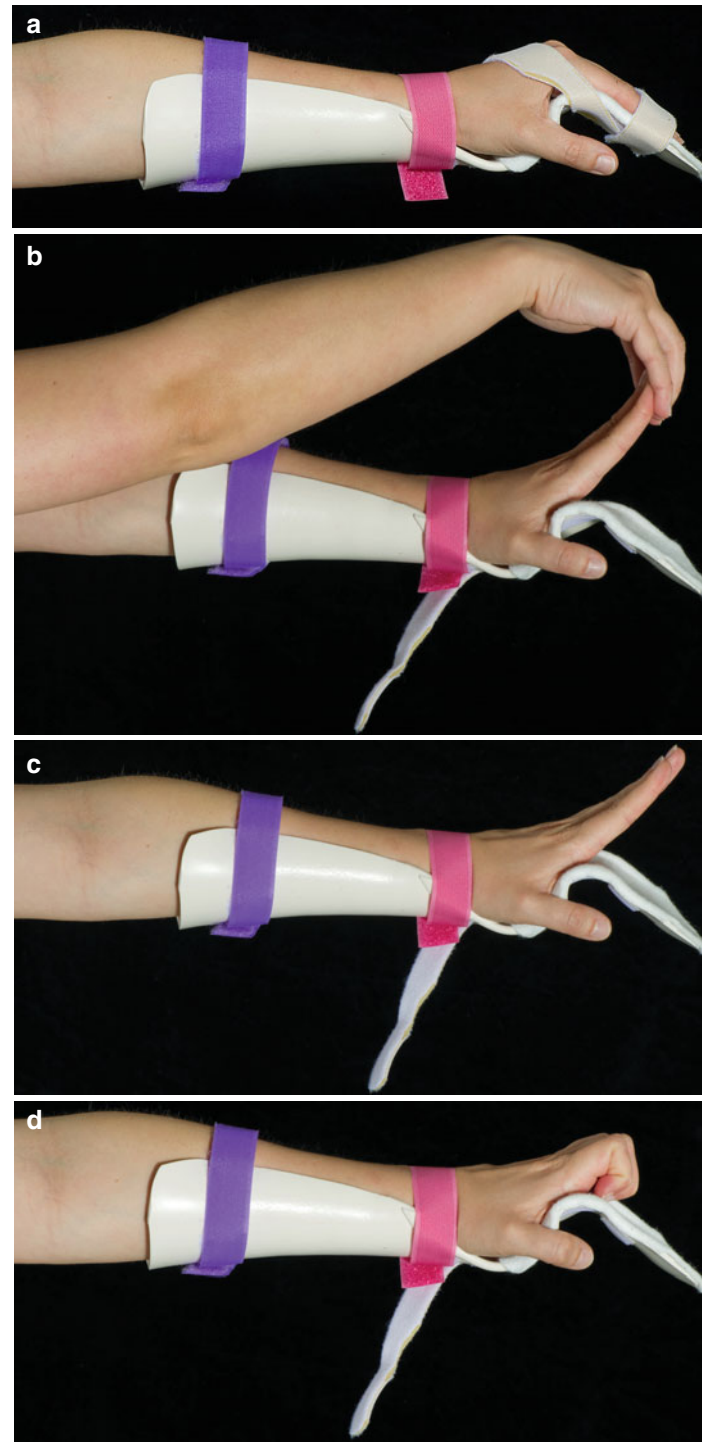


Fig. 4.17 Controlled active rehabilitation of extensors. (**a**) Hand splint (**b**) Passive extension (**c**) Active extension (**d**) Active flexion interphalangeal joints

Those patients treated with passive or active mobilisation programmes appear to rehabilitate more quickly than those who are immobilised in a static splint and obtain a better early range of movement. In most studies, however, there is a negligible difference between the various methods at 3 months [40–43].

In almost all these studies the mobilisation programmes have been performed on injuries sustained in zones 5 and 6 where the results for uncomplicated injuries tend to be good whatever method is used. The real challenge is to provide a good and predictable outcome for extensor injuries sustained within the finger itself (zones 1–4) and for those injuries complicated by associated fractures.

The choice of rehabilitation regimen will depend on the available physiotherapy, the cooperation of the patient, and the site and nature of the injury. In general, uncomplicated extensor tendon injuries in all zones can be safely treated by immobilisation. Active mobilisation programmes are usually reserved for injuries between zones four and eight and may shorten the convalescence. Whenever there is a complicating factor such as an associated fracture active mobilisation is recommended.

The future challenge is to develop robust extensor tendon repair techniques that allow immediate post operative mobilisation.

References

1. Godwin Y, Ellis H. Distribution of extensor tendons on the dorsum of the hand. Clin Anat. 1992;5:394–403.
2. Wehbe M. Junctura anatomy. J Hand Surg. 1992;17A:1124–9.
3. Ueba H, Moradi N, Erne HC, Gardner HR, Strauch RJ. An anatonic and biomechanical study of the oblique retinacular ligamant and its role in finger extension. J Hand Surg. 2011;36A:1959–64.
4. Zancolli E. The structural and dynamic bases of hand surgery. 2nd ed. J.B. Lippincott Company; Philadelphia and Toronto; 1979. p. 92–105.
5. Warren RA, Kay NRM, Norris SH. The microvascular anatomy of the distal digital extensor tendon. J Hand Surg. 1988;13B:161–3.
6. Elliot D, McGrouther DA. The excursions of the long extensor tendons of the hand. J Hand Surg. 1986;11B:77–80.
7. Carducci AT. Potential boutonniere deformity. Its recognition and treatment. Orthop Rev. 1981;10:121–3.
8. Elson RA. Rupture of the central slip of the extensor hood of the finger. A test for early diagnosis. J Bone Joint Surg. 1986;68B:229–31.
9. Boyes JH. Bunnell's surgery of the hand. 5th ed. Philadelphia: JP Lippincott; 1970. p. 440–1.
10. Boyes JH. Bunnell's surgery of the hand. 4th ed. Philadelphia: JP Lippincott; 1964.
11. Doyle JR. Extensor tendons- acute injuries. In: Green DP, editor. Operative hand surgery. 3rd ed. New York: Churchill Livingstone; 1993. p. 1925–54.
12. Crawford GP. The molded polythene splint for mallet finger deformities. J Hand Surg. 1984;9A:231–7.
13. Rayan RA, Mullins PT. Skin necrosis complicating mallet finger splinting and vascularity of the distal interphalangeal joint overlying skin. J Hand Surg. 1987;12A:548–52.
14. Burke F. Editorial. Mallet finger. J Hand Surg. 1988;13B:115–7.
15. Evans D, Weightman B. The pipflex splint for treatment of mallet finger. J Hand Surg. 1988;13B:156–8.
16. Neichajev IA. Conservative and operative treatment of mallet finger. Plast Reconstr Surg. 1985;76:580–5.
17. Stern PJ, Kastrup JJ. Complications and prognosis of treatment of mallet finger. J Hand Surg. 1988;13A:329–34.
18. Webhe MA, Schneider LH. Mallet fractures. J Bone Joint Surg. 1984;66A:658–69.
19. Patel MR, Desai SS, Bassini –Lipson L. Conservative management of chronic mallet finger. J Hand Surg. 1986;11A:570–3.
20. Stack HG. Mallet finger. Hand. 1969;1:83–9.
21. Kleinman WB, Peterson DP. Oblique retinacular ligament reconstruction for chronic mallet finger deformity. J Hand Surg. 1984;9A:339–404.
22. Lucas GL. Fowler central slip tenotomy for old mallet deformity. Plast Reconstr Surg. 1987;80:92–4.
23. Bingham DL, Jack EA. "Buttonholed" extensor expansion. Br Med J. 1937;2:701.
24. Zancolli E. The structural and dynamic bases of hand surgery. 2nd ed. J.B. Lippincott Company; Philadelphia and Toronto; 1979. p. 79–92.
25. Ishizuki M. Traumatic and spontaneous dislocation of extensor tendon of the long finger. J Hand Surg. 1990;15A:967–72.
26. Catalano LW, Gupta S, Ragland R, Glickel SZ, Johnson C, Barron OA. Closed treatment of non rheumatoid extensor dislocations at the metacarpophalangeal joint. J Hand Surg. 2006;31A:242–5.
27. Wheeldon FT. Recurrent dislocation of extensor tendons. J Bone Joint Surg. 1954;36B:612–7.
28. Carroll C, Moore JR, Weiland AJ. Posttraumatic ulnar subluxation of the extensor tendons: a reconstructive technique. J Hand Surg. 1987;12 A:227–31.
29. Kleinert HE, Verdan C. Report of the committee on tendon injuries. J Hand Surg. 1983;8A:794–8.
30. Newport ML, Pollack GR, Williams CD. Biomechanical characteristics of suture techniques in extensor Zone IV. J Hand Surg. 1995;20A:650–6.

31. Henderson J. Epitendinous suture techniques in extensor tendon repairs- an experimental evaluation. J Hand Surg. 2011;36A:1968–73.
32. Lee SK, Dubey A, Kim BH, Zingman A, Landa J, Paksima N. A biomechanical study of extensor tendon repair methods: introduction to the running-interlocking horizontal mattress extensor tendon repair technique. J Hand Surg. 2010;35A:19–23.
33. Newport ML, Williams CD. Biomechanical characteristics of extensor tendon suture techniques. J Hand Surg. 1992;17A:1117–23.
34. Ahmad F, Pickford M. Reconstruction of the extensor central slip using a distally based flexor digitorum superficialis slip. J Hand Surg. 2009;34A: 930–2.
35. Snow JW. Use of a retrograde tendon flap in repairing a severed tendon in the PIP joint area. Plast Reconstr Surg. 1973;51:555–8.
36. Aiche A, Barsky AJ, Weiner DL. Prevention of boutonniere deformity. Plast Reconstr Surg. 1979;46:164–7.
37. Evans RB. Early active short arc motion for repaired central slip. J Hand Surg. 1994;19A:991–7.
38. Newport ML, Blair WF, Steyers C. Long-term results of extensor tendon repair. J Hand Surg. 1990;15A:961–6.
39. Miller H. Repair of severed tendons of the hand and wrist. Surg Gynaecol Obstet. 1942;75:693–8.
40. Bulstrode NW, Burr N, Pratt AL, Grobbelaar AO. Extensor tendon rehabilitation. J Hand Surg. 2005;30B:175–9.
41. Chester DL, Beale S, Beveridge L, Nancarrow JD, Titley OG. A prospective, controlled, randomized trial comparing early active extension with passive extension using a dynamic splint in the rehabilitation of repaired extensor tendons. J Hand Surg. 2002;27B:283–8.
42. Khandwala AR, Webb J, Harris SB, Foster AJ, Elliot DA. A comparison of dynamic extension splinting and controlled active mobilization of complete divisions of extensor tendons in zones 5 and 6. J Hand Surg. 2000;25B:140–6.
43. Mowlavi A, Burns M, Brown RE. Dynamic versus static splinting of simple zone V and zone VI extensor tendon repairs: a prospective randomized, controlled study. Plast Reconstr Surg. 2005;1154:482–7.

General Principles of Skin Cover and Flaps to the Elbow, Forearm, Wrist and Hand

5

Stewart Watson

Keywords
Soft tissue reconstruction • Hand • General principles • Individualized • Wounds • Trauma • Compartment syndrome

Introduction

A surgeon must choose the reconstruction most appropriate to the patient in front of them, their life style and functional needs and also not forget the cosmetic consequences of their choice of reconstruction so important to many patients. Today there are a wide range of reconstructive options for most defects whether skin only or composite tissue defects.

This chapter will consider,

1. The general principles in the assessment and planning of trauma cases with skin defects.
2. The role of the anaesthetist in the initial management and subsequent reconstruction.
3. Specific assessments
   Vascularity
   Compartment compression and Escharotomy
   Wound contamination
   Debridement, judging tissue viability and degloving injuries
4. The planning of reconstruction in defects made in cancer treatment.
5. Skin cover, general principles and consideration of cosmesis
6. The steps of the reconstructive ladder.
   Primary and delayed primary wound closure.
   Secondary healing and the use of vacuum assisted dressings.
   Split skin grafting, the use of artificial dermis, full thickness grafts.
   Local flaps
   Distant pedicled flaps
   Free microvascular flaps.

The decision making progress is illustrated throughout this chapter by the clinical progress of a selection of patients whose clinical course is described as the chapter progresses.

The references chosen are by non means complete but will help the reader drill down on topics that they need to study in more detail. I apologies to authors whose work has not been referenced.

Trauma – General Principles

In the initial assessment of a wound of the upper limb it will become apparent whether the reconstruction requires skin cover only or is compound requiring orthopaedic, plastic and vascular

S. Watson, FRCS, MRCP
Plastic Surgery Unit, Wythenshaw Hospital Unit, Manchester M23 9LR, UK
e-mail: email@jswatson.co.uk

I.A. Trail, A.N.M. Fleming (eds.), *Disorders of the Hand: Volume 1: Hand Injuries*,
DOI 10.1007/978-1-4471-6554-5_5, © Springer-Verlag London 2015

surgical skills. These skills may be in the remit of one surgeon or require inter-disciplinary planning. The surgeons involved need all the equipment, operating time and post operative support provided by a well provisioned hand unit.

The limb injury may be part of a multiply injured patient in which case care of the upper limb has to be integrated with the total care of the patient. The patient may require Advanced Trauma Life Support (ATLS) assessment and resuscitation. All patients will require early anaesthetic assessment. The initial management of the patient and timing of treatment will depend on these factors. Treatment of the upper limb injury may not be life saving but the patients long term upper limb function will be vitally important to them and their family often dictating their quality of life and economic independence into the future.

The surgeon needs a full history of the mechanism of injury – for instance: high or low energy, crush or burn – as all details of the injury may be relevant to the management and are relevant to the prediction of the outcome. The upper limb injury with or without skin loss may in fact be a surgical emergency in its own right because of: an arterial injury and muscle anoxia; compartmental compression; wound contamination with organic material, grease or solvents; high pressure injection injuries or circumferential burns requiring escharotomies. The Surgical team needs to talk to the patient and relatives to get an understanding of the patient, the patient's life style, work and hobbies. The hand surgeon makes an initial assessment of the upper limb and available X-rays, arranges the relevant surgical specialties and reviews the special equipment needed with the theatre staff. Special investigations such as ultra sound, CT or MR scans may be required prior to surgery to further investigate the injury or to evaluate co-injuries or co-morbidities. Doppler studies of the arterial perforators will be required prior to flap surgery (see later).

Anaesthetic Management

At all stages of the management the surgical options have to be discussed with the anaesthetist. Modern anaesthetics allow us to successfully perform long and complex operations safely but the anaesthetist needs to be very much a part of the operation planning. Anaesthetist have different ways to maintain anaesthesia including volatile agents, regional blocks and continuous intra-venous anaesthesia, the choice is important for both the success of the operation and the post operative management. During the initial assessment and debridement it is essential to fully resuscitate the patient and optimize the patient's circulation. If the reconstruction progresses to flap surgery either at the first operation or at subsequent operations continuing anaesthetic planning is vital. Key measurements for successful flap surgery are the patient's core temperature and urine output. A core temperature below 37 °C with poor urine output is not favorable for successful flap surgery or for microsurgery. Peripheral and regional blocks can contribute to pain control and vasodilatation both intra and post operatively.

Trauma – Specific Considerations

Vascularity

The surgeon must realise that the presence of an arterial injury in the forearm, cubital fossa or more proximally in the arm is a surgical emergency. The circulation in the skin of the hand and the presence of a radial pulse does not reliably predict the circulation in the muscles of the forearm or hand. In the example (Fig. 5.1a) there is a deep cut into the cubital fossa dividing the brachial artery. The hand is pink and the radial artery pulse weak and the hand becomes warm with a stronger pulse after resuscitation. The patient was drunk and it would have been easy to say 'leave the case until tomorrow', but, at urgent exploration, poor perfusion of the forearm muscle Fig. 5.1b is apparent. After brachial artery reconstruction the muscle is again a healthy pink Fig. 5.1c. The arterial supply must be reconstructed within the anoxic period or a Volkmann's ischaemic contracture will develop.

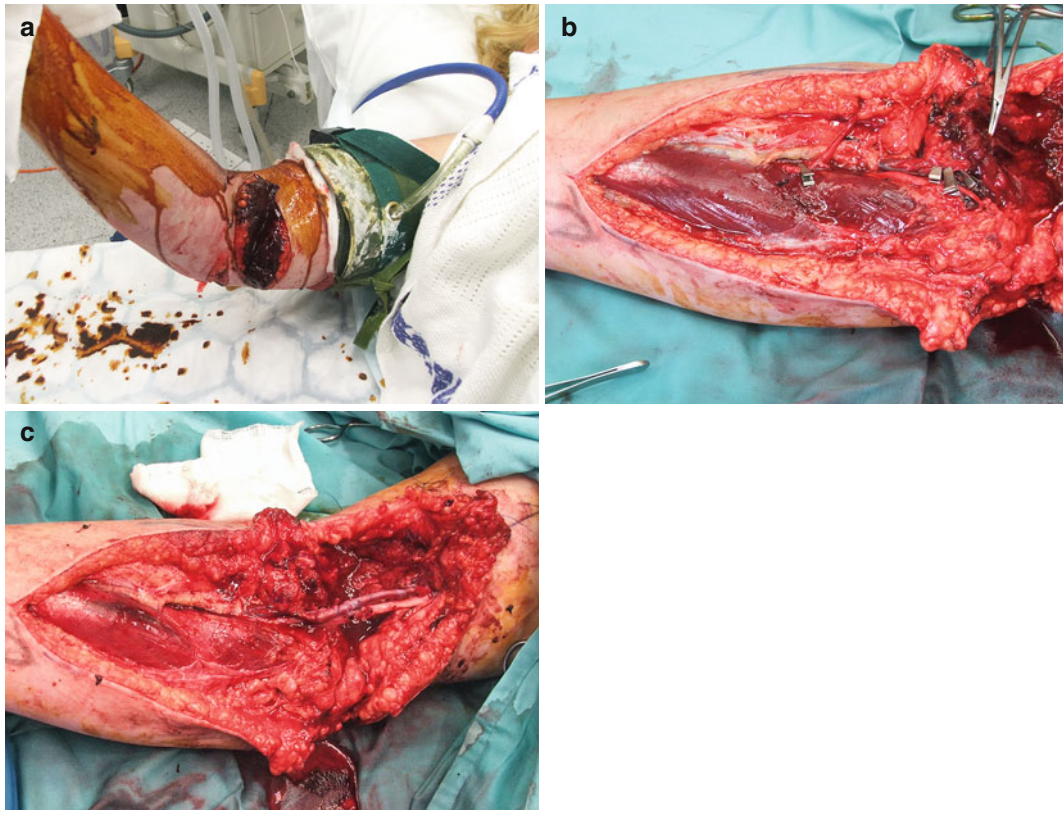


Fig. 5.1 (**a**) This patient who haemorrhaged severely after a glass laceration to the cubital fossa has a warm hand and a radial pulse after resuscitation. (**b**) During urgent exploration the forearm muscle is seen to be poorly perfused. (**c**) After reconstruction of the brachial artery to forearm, muscle is pink and well perfused.

Wound Contamination

Contamination with clothing, dirt, organic material, oils or paint is a surgical emergency. High pressure injection injuries of whatever injected agent, including air and water, is a surgical emergency. Urgent surgical exploration and debridement is essential.

Compartment Compression

Compartmental compression of the muscles of the forearm and hand is a surgical emergency. This may not be as common or as obvious as compartmental compression in the leg where pain is by far the earliest and best symptom upon which to make the diagnosis. The pain from compartmental compression is severe and out of proportion to what one would expect from the nature of the injury by itself. In the presence of a crush injury, or a period of muscle anoxia following arterial disruption, compartmental compression must be suspected and one must act to resolve the situation – it is not a diagnosis for a wait and watch approach. Intra-compartmental pressures must be measured with a suitable manometry device or the compartments explored and decompressed.

In the forearm a single long incision allows inspection and decompression of the flexors, incorporating a z plasty if it is to continue across the wrist to the carpel tunnel. The need to decompress the extensors would be very rare but must be considered if there is suspicion of raised pressures in the extensors. Decompression of the

small muscles of the hand requires linear incisions over the thenar and hypothenar eminences, first dorsal interosseous muscle to include the adductor policis brevis, and each dorsal compartment via two incisions centred over the index-middle and ring/little inter-osseus compartments.

When decompressing the hand most surgeons would also decompress the carpal tunnel and possibly Guyons canal as well. In the post operative period after hand, wrist or forearm injuries it may be clinically impossible for some days to evaluate if there is median or ulnar nerve compression in the carpal tunnel or Guyon's canal especially with multi level injuries and in sedated patients. This being the case it is usually felt that decompression at the wrist obviates the risk of a secondary injury to these nerves.

A compartment must be completely released to be effective, if a short section of a compartment is not released the muscle under this short section will be left under pressure. Fasciotomies in the forearm and hand are best done before the tourniquet is let down. In a bloodless field it takes only minutes to release these compartments fully. Surgical release of muscle compartments during the hyperaemic period following release of the tourniquet is bloody and slow.

Escharotomy in the Burnt Hand and Upper Limb

When there is a circumferential full thickness or deep dermal burn that section of the limb cannot expand in response to normal inflammatory exudation into tissue planes. Pressure within the tissues then increases and produces venous obstruction, hypoxia, further exudation and ultimately arterial obstruction. Like compartmental compression this is a diagnosis to actively consider and steps taken to exclude or treat. Treatment consists of a full thickness skin release, only rarely release of the deep fascia, from normal proximal tissue to normal distal tissue, 'from where it hurts to where it hurts'. This needs to be done both on the radial and ulnar boarders of the limb. It can possibly be done without the need for any anaesthesia but be aware it is not a trivial surgical procedure and can produce considerable bleeding if superficial veins are cut in the process. Sufficient surgical facilities and equipment are required to do this safely.

Wound Debridment, Judging Tissue Viability and Assessing Tissue Degloving

At initial exploration and debridement all tissues are assessed individually when the patient is warm and well resuscitated. Debridement starts with saline irrigation and removal of gross contamination by wiping tissues or the use of a soft brush. When there is dirt or particulate matter ingrained in the tissue the choice is to meticulously pick it out using magnification or to excise contaminated tissue. This is slow and laborious work but time well spent. There may come a time when a judgement has to be made between leaving some degree of contamination in tissues such as nerve and muscle that cannot be easily reconstructed and more radical tissue resection and reconstruction. Figure 5.2a–e illustrates a crushed forearm before and after debridement, and after reconstruction. This shows the level of macroscopic debridement that is essential before reconstruction can take place. In addition deep tissue biopsies for bacteriological culture should be taken.

Sub-cutaneous Tissue

The sub-cutaneous fat is crucially important as it is probably the most susceptible tissue to damage by a crush or shear injury and its viability can be difficult to evaluate initially. Skin degloving occurs when sub-cutaneous fat is sheared off the deep fascia thus disrupting any arterial supply coming to the sub-cutaneous tissue and hence to the skin from that area of fascia (see below for a discussion on the blood supply to skin). The extent of degloving of the skin must be evaluated to determine whether it is localized or extensive degloving Fig. 5.3a, b. The sub-cutaneous tissue under areas of local degloving can be impossible to fully clean. If there is contamination beneath degloved skin a full debridement may require resection of this tissue including the overlying

Fig. 5.2 (**a**) Road crush defect of the forearm. (**b**) Forearm after surgical debridement. (**c**) Forearm after bone fixation. (**d**) XR appearance after 18 years. (**e**) Reconstruction after a free latissimus dorsi flap and pedicled groin flap

skin. This is a difficult decision to make as it will create a bigger defect but doing so will reduce the possibility of subsequent wound infection. At 36–48 h after injury damaged and poorly vascularised fat is more easily identified because of its grey color or fixed red staining. There may still be thrombosed vessels among areas of scattered arterial circulation.

Skin

The circulation in the dermis must be separately assessed. If the dermis bleeds even small amounts of red blood it is probably viable. If the cut dermal edge oozes blue blood it most likely will die. At a second look operation at 36 or 48 h it is easier to evaluate dermal viability.

If degloved skin and subcutaneous tissue clearly has no circulation it has to be resected. If it has a dubious circulation or there is significant dirt contamination of the fat beneath viable skin the decision to preserve the skin can be difficult.

Two factors should be considered:

Firstly, newly devascularised skin and even skin with some superficial damage can be harvested either as a split skin graft (SSG) or a full thickness skin graft to use on the limb. Figure 5.3c

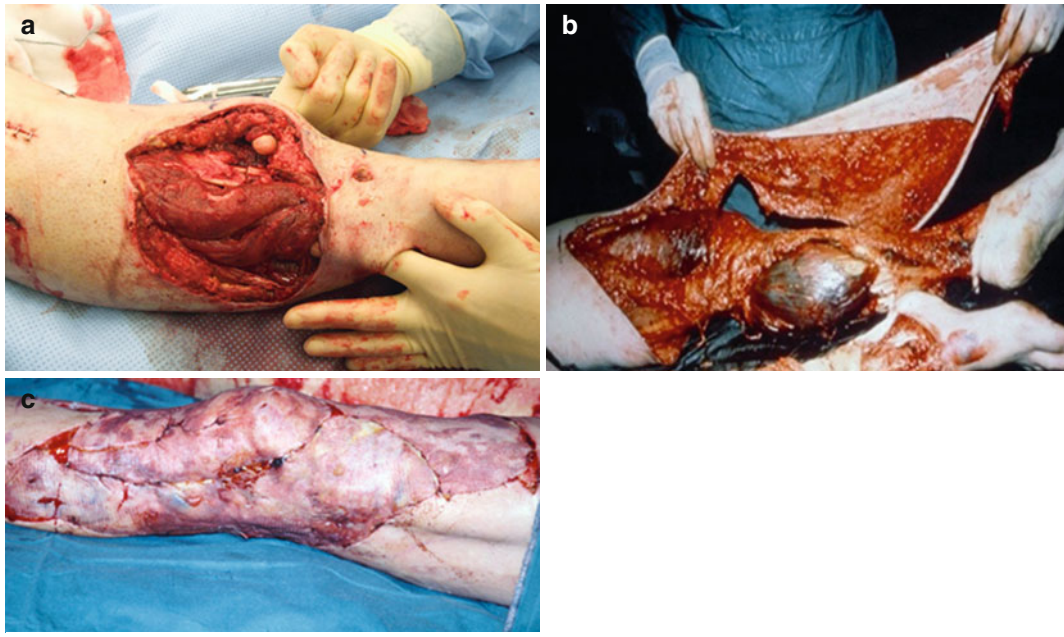


Fig. 5.3 (**a**) Localised degloving of the skin. (**b**) Extensively degloved skin. (**c**) The degloved skin has been defatted and re-applied either as full thickness graft over the patella or as split skin graft (SSG) on the night of the injury

shows extensive degloving of the legs. The skin is defatted for use as a full thickness graft over the patella and as a SSG on the rest of the leg. SSG can be meshed 2 to 1 or 4 to 1 and can be stored in the refrigerator for several days and used at a second look operation if this can be done within the legal requirements for the storage of human tissue. Full thickness skin graft (after removal of sub-cutaneous fat) has to be re-applied to the limb on the day of injury. Figure 5.4a, b shows a full thickness gloving injury to the palm and volar fingers. Figure 5.4c shows the palm skin replanted after defatting as a full thickness graft and its appearance at 3 weeks. Figure 5.4d shows the appearance at 6 months. Full thickness grafts are being inset into each finger in order to release a volar skin tightness but notice the general good quality of the replanted skin [1].

Secondly, the decision hinges very much on the consequences to the patient of leaving poorly vascularised or contaminated skin and subcutaneous tissue in-situ and thus risking slow healing and wound infection. If the limb injury is simple degloving with no injury below the deep fascia then slow healing with a risk of some infection and delayed healing may not be a serious problem. In such cases a conservative resection of skin and subcutaneous tissue leaving some poorly vascularised tissue which heals slowly may preserve dermis and reduce the need for additional skin grafts. If, however, there is damage beneath the deep fascia, particularly a fracture, then the risks of leaving damaged or contaminated sub-cutaneous tissue which might lead to infection and slow healing can have serious consequences on the bone healing. In this situation the safest strategy is to resect all the damaged or degloved sub-cutaneous tissue with the dermis above it. This will create a completely clean wound that can proceed to reconstruction with the expectation of good primary healing of all tissues. The advent of vacuum assisted dressings (VAC) (see below) has allowed delayed inspection and wound coverage to give us more time to decide on tissue viability and resection.

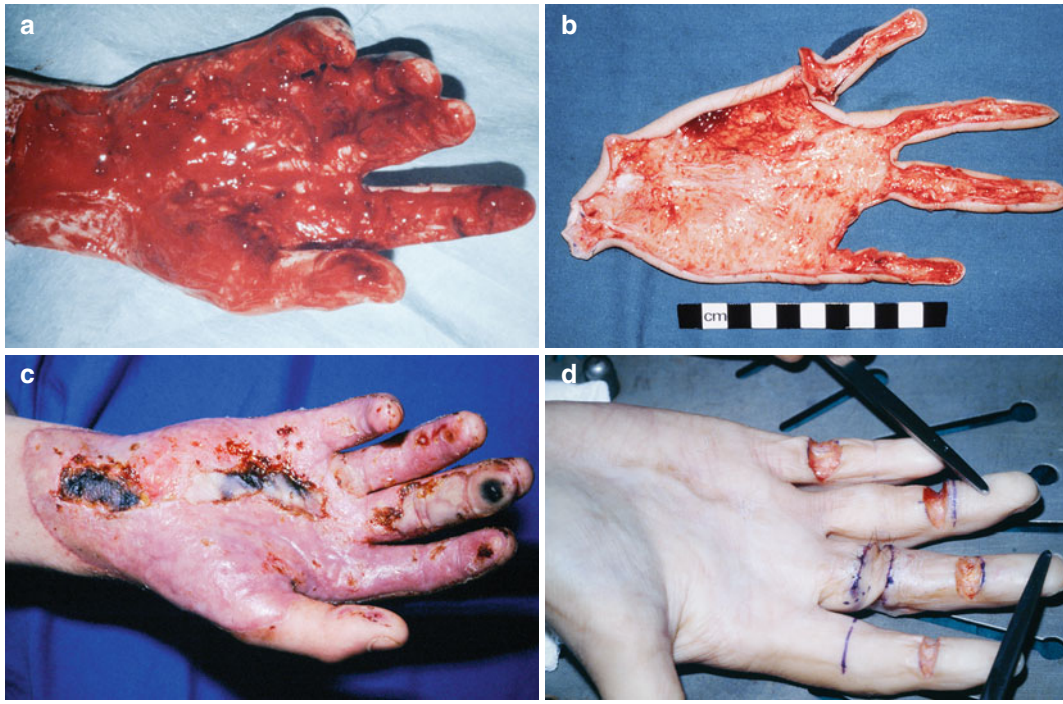


Fig. 5.4 (**a**, **b**) Complete full thickness skin degloving of the palm and volar fingers. (**c**) The appearance at 3 weeks after replantation. (**d**) At 6 months despite the general good appearance of the replanted skin there was a volar contracture which required release and full thickness skin grafts

Muscle

Muscle viability is also often difficult to completely evaluate at the first operation. Injured muscle may appear compromised but still have spots of arterial bleeding within its substance and still show contractility. Muscle degloved off its origin gives an indication of the severity of the injury. Obviously dead muscle must be resected but, if the patient's condition allows, muscle with an arterial input but of dubious viability may be left until a second look at 24–36 h.

Bone

Bone viability and contamination can be difficult to evaluate at the first wound assessment and this will obviously influence the method of bone stabilization. A second look may well be necessary and a revision of the original bone fixation may also be necessary, examples are given below.

Vessels

Vascular reconstructions must be performed and functioning before further reconstruction proceeds. Arterial and venous reconstructions must be flowing well, the vessels not kinked, redundant or constricted.

A third look operation can be planned if time and the patient's condition allows but again there will come a time when a judgement has to be made between further resection back to completely healthy tissue and the start of reconstruction.

Cancer Reconstruction

The patient undergoing surgery for upper limb cancer ablation should be discussed and planned at the appropriate Multi-Disciplinary Team (MDT) after staging to ensure optimal treatment. In most cases resection and reconstruction will be planned in one operation and there is time to plan details with the different surgical and anaesthetic specialties that will be involved. Radiotherapy may be planned before surgery in some sarcoma cases or radiotherapy and chemo therapy may be

planned after surgery in other cases. The need for adjuvant therapy will influence the choice of reconstruction required and flap reconstruction will almost invariably be needed rather than a SSG to ensure robust primary healing. The flaps most likely chosen will be single stage fasciocutaneous flaps (see below) or micro-vascular free flaps (see below) [2].

Skin Cover

General Principles

The concept of a surgical reconstruction ladder is traditionally presented in a supposed order of complexity of the reconstruction. Primary closure; delayed primary wound closure; secondary healing perhaps with the assistance of a vacuum assisted dressing device; split skin graft; artificial skin dermis and subsequent split skin graft; full thickness graft; local flap; distant pedicled flap; free microvascular flap. While this is a valuable concept it is however **best considered to be a guide or tool kit** to make a surgeon think of all the reconstructive options that may be available. The choice of reconstruction is made by considering the quality of reconstruction that the patient ideally requires, both functionally and cosmetically. Where composite tissue reconstruction is needed a decision has to be made between total primary reconstruction and staged reconstruction. The choice of reconstruction may be dictated by the patient's condition if they have multiple injuries or co-morbidities. Options have to be discussed with the patient. The surgeon's experience or a Unit's facilities should not be allowed to dictate the choice of reconstruction. The patient should be transferred to another unit if it is in their best interests. Careful planning and judgment is required.

With fractures or bone defects the aim is to get definitive bone fixation or reconstruction at the time of skin cover. Much has been learned from the management of compound tibia fractures and this should be extrapolated to upper limb compound injuries. Modern guidelines on the management of compound fractures emphasize the importance of the patient being moved to a unit with the necessary expertise for their combined management [3, 4]. Much depends on the local facilities but having a team with the necessary trauma experience is paramount. Some methods of bone fixation, particularly external fixators, can interfere with the raising and moving of a flap or make the revascularization of a free flap impossible dependent on the pin placements. If it is judged that the initial bone fixation is not optimal or the plastic surgeon cannot work around the fixation then the first few days is the time to change the fixation. The following two cases illustrate these principles of combined specialty working to achieve debridement and proper repair. It is necessary for the teams to persevere with these difficult cases despite the difficulties to achieve the best result possible reconstruction in the initial treatment period. This will always be preferable to late reconstruction.

This avulsion amputation of the hand Fig. 5.5a was successfully replanted. At 6 days says the hand is well perfused but the zone of injury looks very unfavorable Fig. 5.5b. If this replant was to heal it had to be resurfaced with a flap. It was decided to shorten the bones, add a plate to the radius and resurface the volar and radial surfaces with a pedicled groin flap Fig. 5.5c–f (see below). This was difficult surgery but not as difficult at it would have been 3 months later. Despite the unfavorable nature of the amputation the final function with bone union, sound skin healing, good ulnar nerve recovery and modest tendon function is very satisfactory to the patient.

This forearm revascularization Fig. 5.6a seen here at 2 weeks would not have progressed to healing. The volar surface was resurfaced with a free parascapular flap Fig. 5.6b, c (see below). The forearm soft tissues healed but there is a sinus and bony non-union Fig. 5.6d. The patient went on to a vascularised fibular transfer Fig. 5.6e–h. The final result was very satisfactory but this vascularised bone flap would most probably not have been necessary had there had been adequate bone debridement, shortening and fixation at the time of revascularisation.

In compound defects involving nerve and tendon defects whilst total primary reconstruction

incorporating a flap and nerve grafts or extensor tendon grafts is elegant and time-saving and is probably the gold standard, staged reconstructions may be expeditious for a particular patient in the long run. Examples would include those patients where co-morbidities precluded complex, lengthy

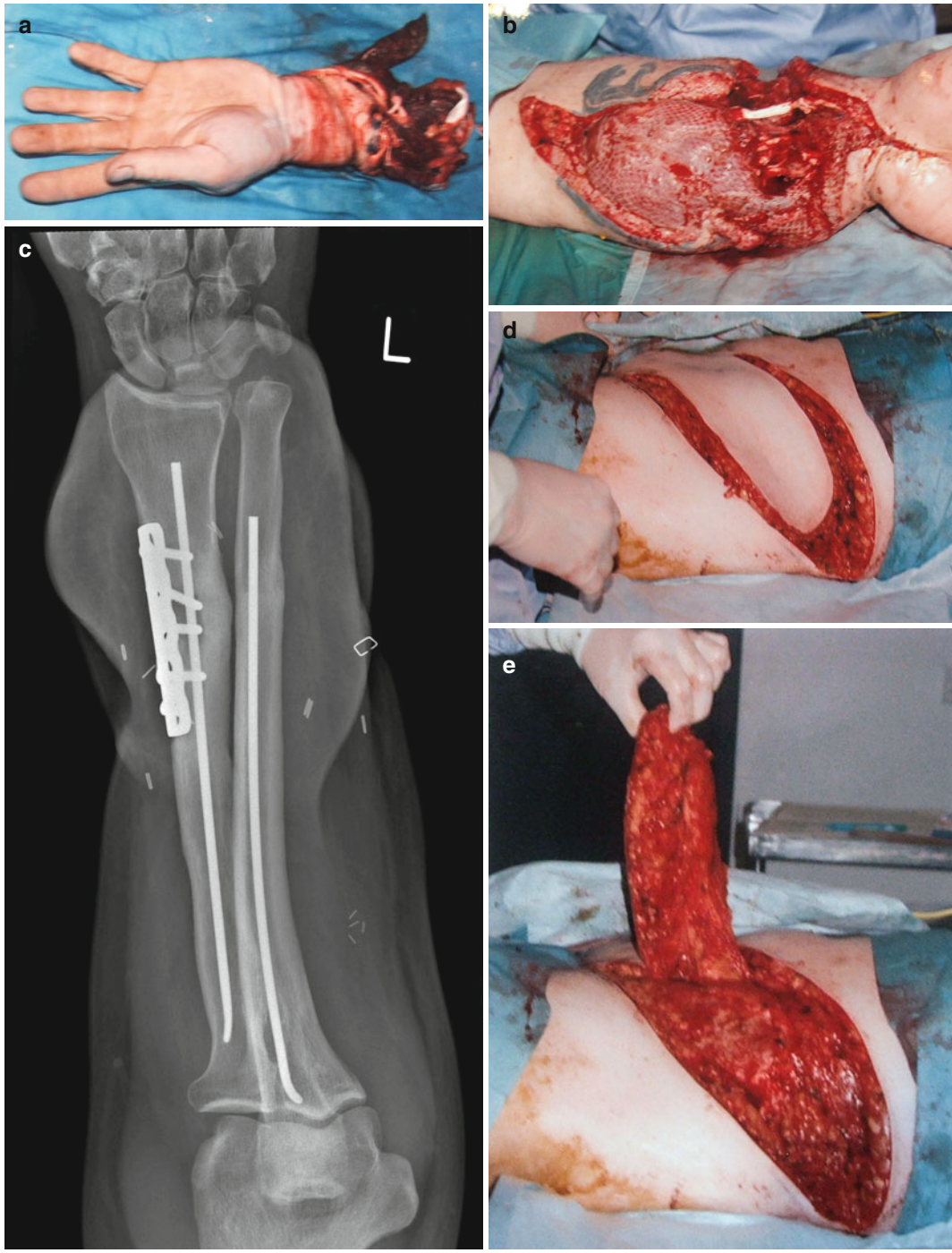


Fig. 5.5 (**a**) An avulsed hand amputation. (**b**) The appearance at 6 days. (**c–f**) At 6 days the bones were shortened and the fixation strengthened. The degloved areas were resurfaced with a pedicled groin flap

Fig. 5.5 (continued)

reconstruction. Figure 5.7a–e shows a compound defect of skin and ulnar nerve loss which, after debridement, is reconstructed with primary nerve grafts and a free lateral arm flap from the contralateral arm. In a case requiring skin cover and nerve grafts, if the length or degree of nerve injury is unclear, delaying nerve reconstruction 2–3 months for detailed assessment of the degree of nerve damage may be the best plan.

Similarly where there is skin loss and extensor tendon loss a primary reconstruction is the ideal. It can however, be reconstructed in stages and there are situations where this is the best plan. If the passive range of movement of joints is limited, or the state of the motor muscles is unclear, then a flap reconstruction with primary extensor tendon grafts will not produce as good a result as flap or SSG cover followed by hand therapy and then tendon grafts when the joints passive range of movement is maximal. One method to achieve staged reconstruction is illustrated in Fig. 5.8a–d. The wound on the dorsum of the hand was initially healed with a split skin graft (SSG). This is replaced by a pedicled forearm flap (see below for details) incorporating two tendons, the palmaris longus and a longitudinal split of the flexor carpi radialis. Tendon and nerve grafts are a valuable tissue resource of limited supply and must be used optimally.

Another factor to consider in staged reconstructions is the quality and durability of skin cover that is required should it need to be re-raised at a future operation for a tendon graft, bone surgery or nerve graft. If the skin cover will need to be re-raised then a flap with a skin paddle gives the best cover over the future reconstruction. A muscle flap with skin graft cover may be suitable for some defects but it will behave like a skin graft and be subject to contracture and reduced growth potential in children. It also can be difficult to re-raise and inset if further reconstruction is required, an example of this is illustrated later in the chapter.

Cosmesis of the Primary and Secondary Defects

Patients will accept scars at the primary site of injury or cancer ablation but scars at secondary surgical sites may cause considerable dissatisfaction, secondary functional problems and even successful medical legal action. **It is essential to discuss reconstructive options and secondary scars with a patient**. Secondary scars are shown and discussed in the examples used throughout this chapter but even a skin graft donor site on a thigh taken without discussion can become the focus of great dissatisfaction to a patient who likes to show their legs Fig. 5.9. The small SSG in this case could have been taken from the buttock.

Specific Wound Closure Techniques

1. **Primary and delayed primary wound closure** is used if there is a lot of tissue swelling initially and also used to close muscle

compartment release wounds. A delayed wound may still not close completely by direct suture and additional skin grafts may be necessary for a tension free closure.

Natural healing with dressings may be all that is possible in the elderly or seriously ill or where the medical condition and state of the tissues precludes reconstructive procedures. A very valuable dressing technique to consider in most wounds is negative pressure wound therapy (NPWT) originally described clinically by Argenta [5] and called sub atmospheric pressure dressing and now usually referred to as vacuum assisted therapy or VAC therapy. VAC therapy is an important development in wound management and used extensively, however, there is still little standardisation in its use and as yet few detailed clinical studies upon which to base best practice. This is highlighted by [6] **Glass and Nanchahal**. Most surgeons believe that VAC therapy can be effective at keeping wounds clean and 'buying time' before it is

Fig. 5.6 (**a**) A revascularised forearm with a wide zone of injury. (**b**, **c**) The defect is resurfaced with a free parascapular flap. (**d**) There is a persistant sinus and bony non-union. (**e**–**h**). The non union was healed with a free fibular flap with skin paddle

Fig. 5.6 (continued)

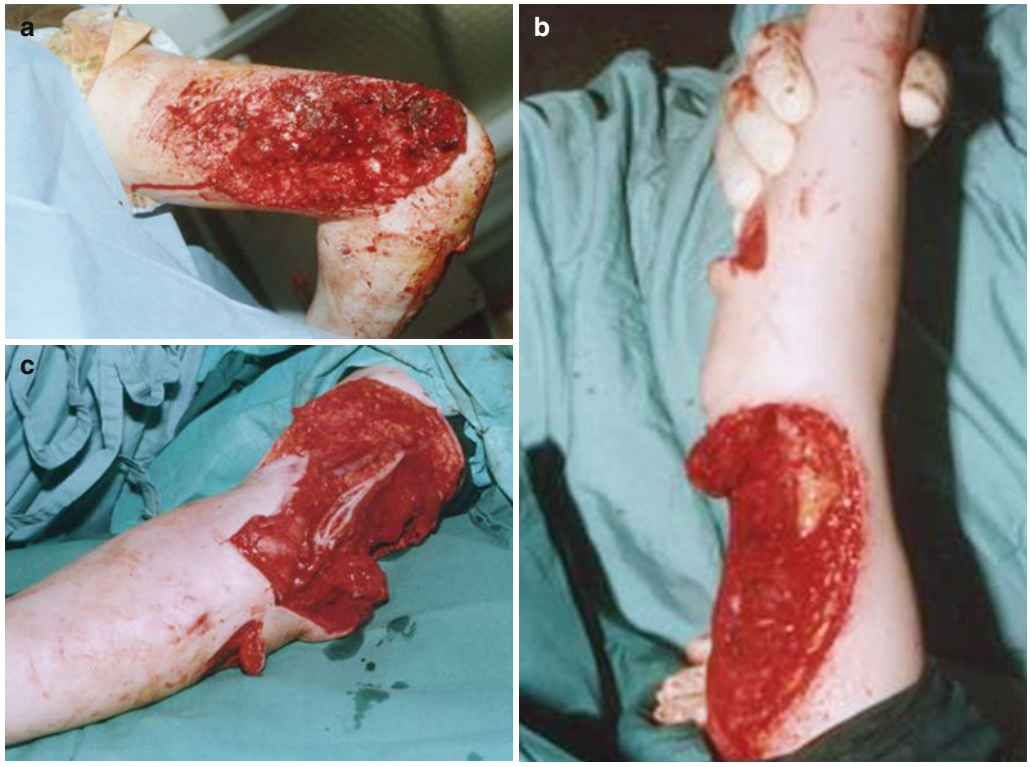


Fig. 5.7 (**a**) Wound above elbow before debridement. (**b**) Wound after debridement. (**c**) Cable grafts to the ulnar nerve in place.

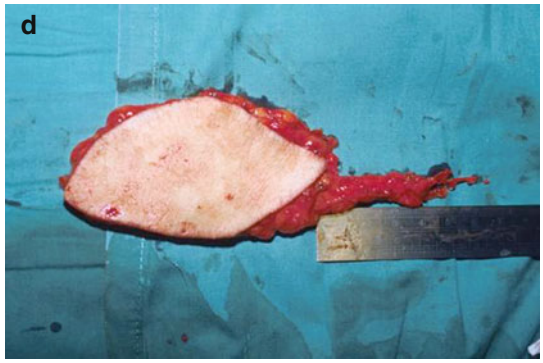


Fig. 5.7 (continued) (**d**) Free lateral arm flap raised. (**e**) Final result

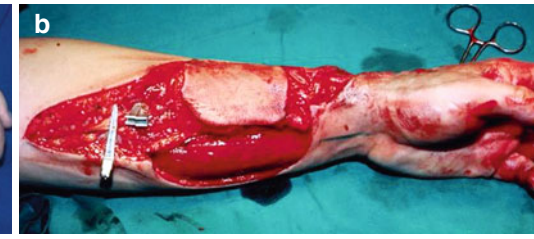


Fig. 5.8 (**a**–**d**) Loss of the extensor tendons to the index and long fingers which is reconstructed at a second stage by a pedicled forearm flap incorporating palmaris longus and a longitudinal half of flexor carpi radialis

Fig. 5.9 This split skin graft donor site on the front of the thigh has produced a hypertrophic scar and will leave a permanent mark. A more hidden donor site could have been chosen

possible to proceed to reconstruction. Compound wounds have progressed to healing under vacuum therapy [7]. Healing under vacuum therapy might not be the traditional first choice of reconstruction in a complex injury but it might be the most expeditious method in some patients. VAC therapy is an important part of the treatment options of the wounds of modern warfare [8]. Vacuum therapy can also be used over a meshed skin graft at the time of skin grafting if immobilization of the graft cannot be secured by other means in difficult anatomical areas.

The neonate has a great capacity to heal without surgical intervention as illustrated in Fig. 5.10a, b. This full thickness skin loss on the dorsum of the hand from a tissued drip progressed rapidly to healing in dressings.

2. **Split skin grafts (SSG) and meshed skin grafts** are the main stay of wound healing although the cosmetic appearance in term of color and surface texture can be poor. SSGs over the volar surface of joint have the potential to contract and in children will not grow with the child. Figure 5.11a, b shows a forearm wound satisfactorily covered by a meshed skin graft. In the example Fig. 5.12a–c however the wound was initially covered by a SSG which did not take over the median nerve possibly due to inadequate debridement. The full thickness defect is subsequently covered by a small microvascular free skin flap.
3. **Full thickness skin grafts** Full thickness grafts that take fully have important properties. They do not contract and in children they grow with the child. The scar around the full thickness graft, however, does not have the same growth potential so the edge of the graft needs to be carefully placed such that it falls along the mid axial line of a joint not over the volar surface. Full thickness grafts are used extensively on the volar surface of fingers in syndactyly release and in dermofasciectomies for Dupuytrens release. There is limited availability of these grafts so for bigger defects and for defects where the graft bed is not uniformly good a flap would be preferred to ensure good healing and not waste a valuable tissue.

Collagen Substitutes. The use of bovine collagen dermal substitutes is increasing and will become an important factor in wound healing in the future [9]. See the Chapter on Burns.

Flap Reconstruction

Flaps bring in their own blood supply and provide robust healing to an area. In the mid 1960s the blood supply to the skin was poorly understood and the only flaps available were so called random pattern flaps. In (Fig. 5.13a, b) this historic case illustrates a thin superiorly based random pattern abdominal flap comprising skin and

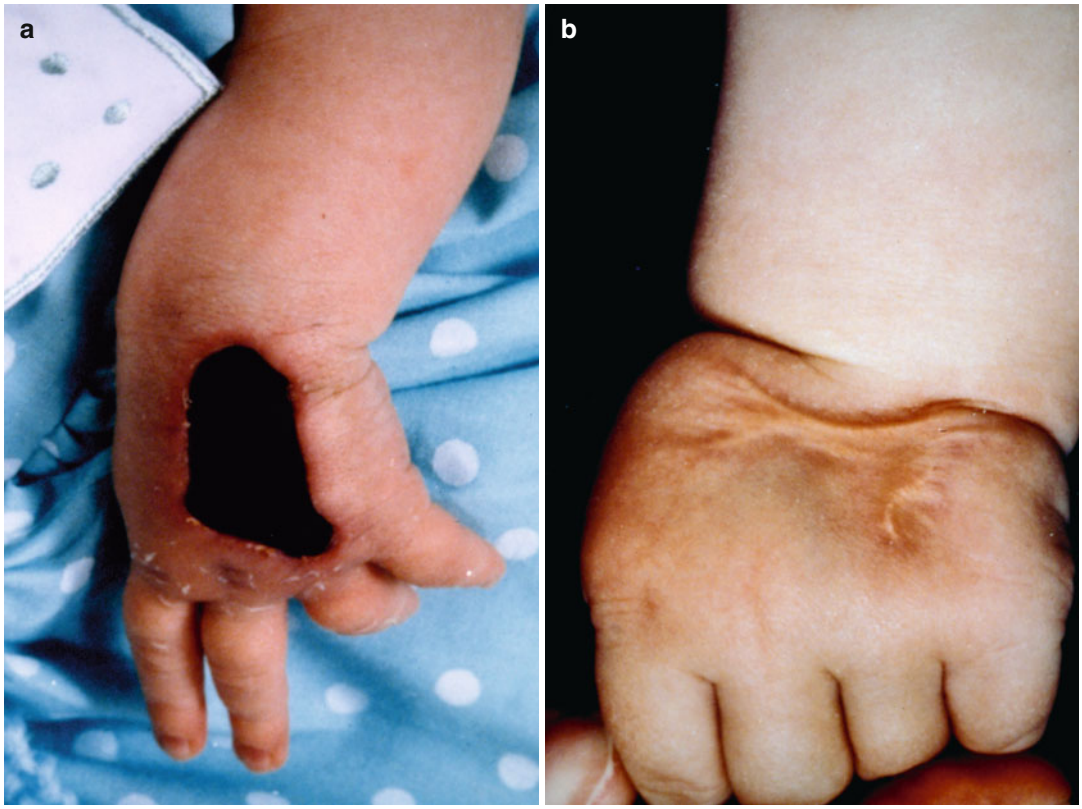


Fig. 5.10 (**a**, **b**) Shows necrotic skin on the dorsum of a neonates hand from a tissued drip and the natural capacity to heal at this age

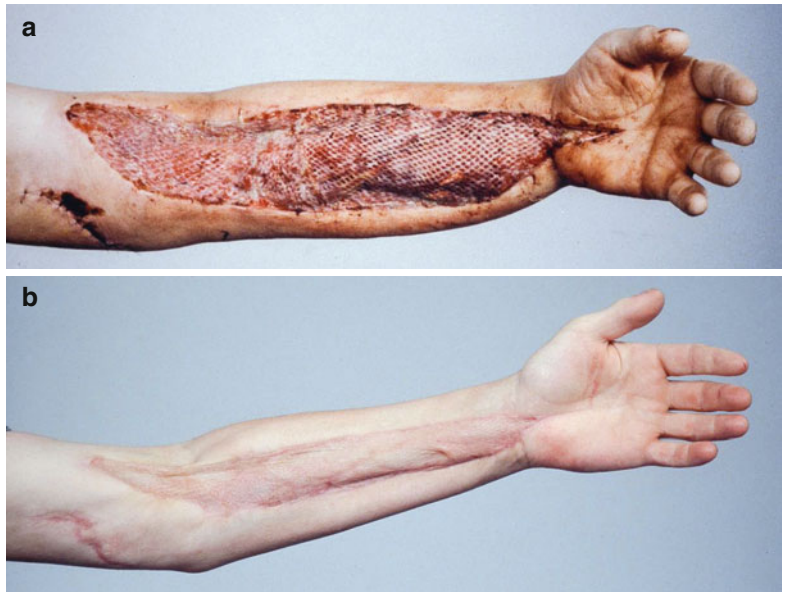


Fig. 5.11 (**a**, **b**) This forearm defect has been resurfaced by a meshed skin graft. There is a mild volar contracture of the forearm but it was not functionally significant in the age group of this patient

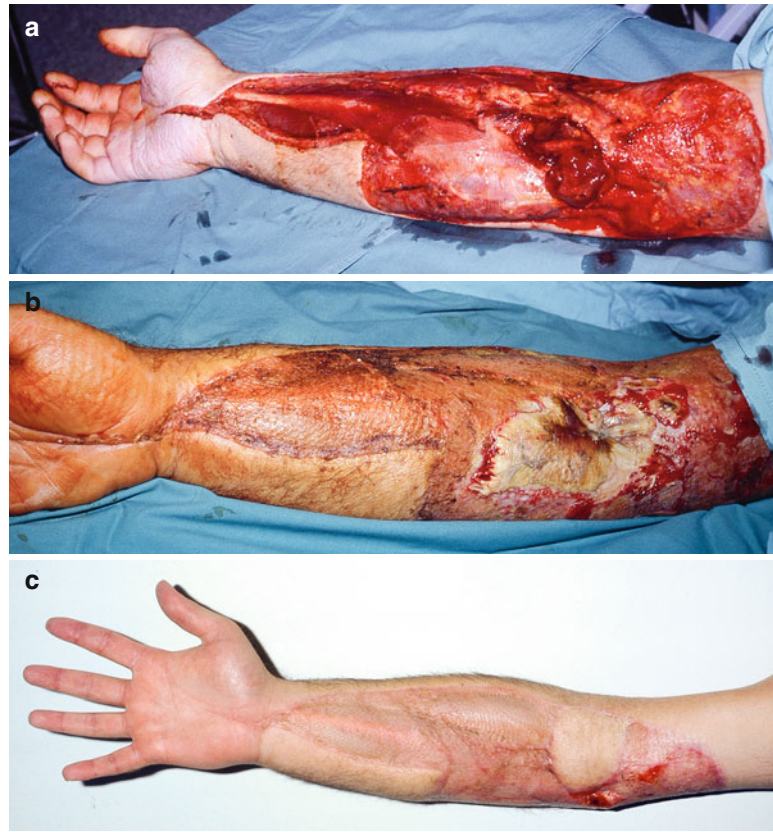


Fig. 5.12 (**a**–**c**) This forearm wound was initially covered by a SSG. This failed over the median nerve so to preserve the nerve it was covered by a small free microvascular flap

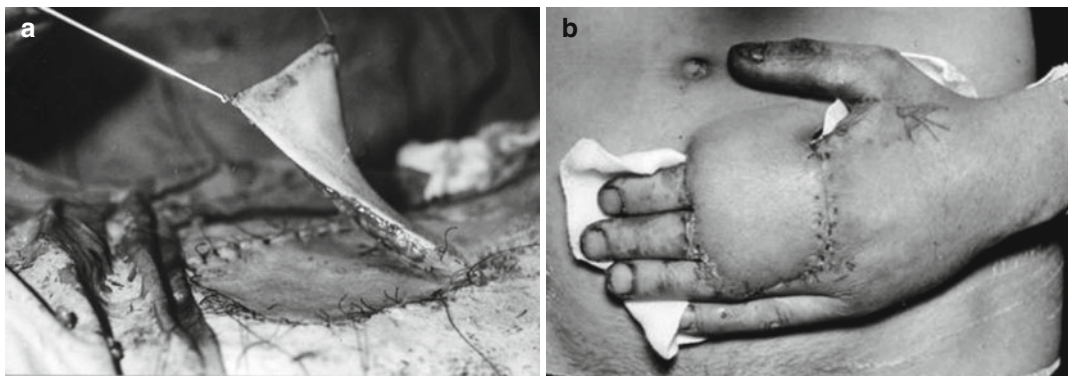


Fig. 5.13 (**a**, **b**) In this case from the early 1960s a defect on the dorsum of the hand is resurfaced by a superiorly based pedicled flap. It is called a random pattern flap because of the poor understanding at the time of the blood supply of skin

subcutaneous fat where the length of the flap was the same as its width. This flap requires division and subsequently the fingers require separating, perhaps involving four operations over 4 months but will provide an excellent result. Pedicled abdominal flaps continue to have a role in the reconstruction of upper limb defects (see below) although mostly are now designed with the base of the flap inferiorly to incorporate the blood supply from the superficial inferior epigastric and superficial circumflex iliac vessels.

We now have a good understanding of the anatomy of the blood supply to skin **Ref**. [10, 11, 12] and how to design flaps. One definition of flap designs is based on their tissue composition. (1) skin flaps (with subcutaneous tissue) supplied by vessels direct to the subcutaneous tissue and skin, the groin flap is the main flap in this group (2) fascio-cutaneous flaps with blood supply initially to the fascia and (3) myocutaneous flaps with the blood supply initially to the underlying muscle. To this can be added composite flaps containing in addition to skin possibly nerve, tendon or bone. The term axial pattern flap was introduced as a flap with a known artery running along it allowing a longer flap length to flap width ratio than the random pattern flap where there is no known artery running down the flap. There is further discussion on this later. Taylor's anatomical studies delineated 374 arteries of over 0.5 mm diameter reaching the skin in the human body and described as arterial perforators. These can be mapped with a hand held Doppler or, for more information on the size, depth and direction of the blood flow in these vessels, with a colour Doppler. Taylor introduced the term angiosome as the area of tissue supplied by a perforator. The branches of these perforator vessels connect to the next perforator vessels by a smaller number of vessels called choke vessels. The common factor in the clinical design of flaps today is to understand the arterial perforators and the vessels that they originate from and also the venous drainage [13]. Many flaps are still described along the anatomical lines above but increasingly new flaps are described on their perforator anatomy. This generation of perforator flaps have advantages in the upper limb. They have flexibility of design, they can be raised as thin flaps and can be designed as chimeric flaps carrying different tissue for example skin, fascia and bone. Flaps based on the dorsal scapular artery axis and the circumflex femoral artery axis are particularly relevant to upper limb reconstruction (see later for examples).

For an upper limb defect that requires a flap reconstruction it is usual now to have a choice of possible flaps that could be used. These have very different implications in terms of number and type of operation, time in hospital and scarring. The choice of flap in any particular patient is a matter of judgment and experience. Flaps are discussed below but the reader is referred to standard texts and to papers on individual flaps for full technical details before raising them [14, 15, 17].

Local Flaps

For very small defects up to 2 cm/3 cm diameter, often those made after skin cancer resection but not for trauma defects, small local flaps comprising skin and subcutaneous tissue only can be used. These local skin flaps can be raised along various geometrical patterns like V to Y advancement, rotation, transposition or keystone design. They are raised along the lines that tissue laxity dictates and with a length to width ratio of one. These flaps are often raised without perforator mapping first but it is logical at least to plastic surgeons to map the perforators which is simple to do and adds to the security of the design.

The predominant local flaps used in upper limb reconstruction now are fascio-cutaneous flaps. Their introduction along with a greater understanding of the blood supply of skin revolutionized flap design. Fascio-cutaneous flaps are used in the following four configurations:-

Proximally Based Fascio-Cutaneous Flaps

Proximally based fascio-cutaneous flaps are used extensively in the lower limb but only around the elbow in the upper limb. Figure 5.14a–c shows a lateral fasciocutaneous flap raised which can be transposed anteriorly to the cubital fossa or posteriorly for a defect over the olecranon.

Perforator Fascio-Cutaneous Flaps

Perforator fascio-cutaneous flaps are based on perforating arteries coming though the fascia which are mapped by Doppler. These flaps are used as local flaps or free flaps. The perforator artery arises from the parent artery beneath the deep fascia then passes through the deep fascia into the subcutaneous tissue where it supplies the subcutaneous tissue and skin. When the flap is

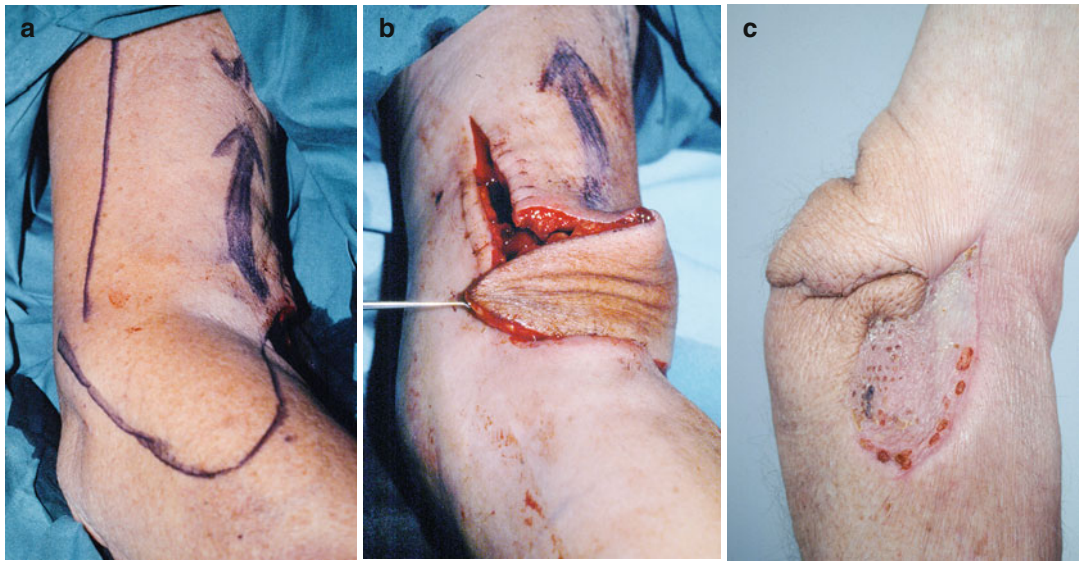


Fig. 5.14 (**a**–**c**) This lateral proximally based fascio-cutaneous flap can be transposed anteriorly to the cubital fossa or posteriorly to the olecranon

raised the fascia is left around the perforator to protect the perforator but as the flap paddle is raised away from the perforator the flap paddle can be raised with the fascia or if a thinner flap is required it is raised superficial to the fascia just below the Scarpa's fascia level [13]. Strictly speaking the flap can be described partly as a fasciocutaneous flap and partly as a skin flap. The new understanding of perforator flap anatomy makes it possible to raise much thinner flaps than before, particularly suitable in the upper limb. The surgeon must perform Doppler studies to fully map the arterial perforators in the area of operation before deciding on what options are available for the particular defect confronting the surgeon.

Figure 5.15a–c shows a small fascio-cutaneous flap completely islanded on an arterial perforator with its vena commitans. Once islanded completely then the flap will move sideways to cover the defect or alternatively could be rotated 180° providing the surgeon judges that this does not compromise the pedicle. The dorsal forearm defect in Fig. 5.16a, b although small has the extensor tendons and fracture exposed. The perforators have been marked and the dissection proceeds until the flap is fully mobilized on a single perforator. The perforator itself is dissected deeply to mobilise it and give the maximum movement to the flap. Then and only then does this flap have sufficient movement to cover the important part of the defect, the exposed tendons and fracture leaving the rest of the defect skin to be covered by a SSG.

Distally Based Fascio-Cutaneous Flaps

Distally based fascio-cutaneous flaps [16] have produced a major advance in flap reconstruction in both the upper and lower limb. With distally based flaps the arterial supply is retrograde but also the venous drainage flows distally out of the flap against the direction of the valves. A fully islanded fasciocutaneous flap relies on venous drainage through the vena commitans accompanying the arterial pedicle. A pedicled distally based flap might have the option of preserving a subcutaneous vein to supplement venous drainage. The radial forearm, Posterior inter-osseous and dorso-ulnar forearm flaps are the 3 classic reversed flow flaps used in the upper limb.

(a) **The radial forearm fascio-cutaneous** flap or Chinese flap [7], is raised with the radial artery and venae commitans. Figure 5.17a–c shows a typical skin marking with the artery

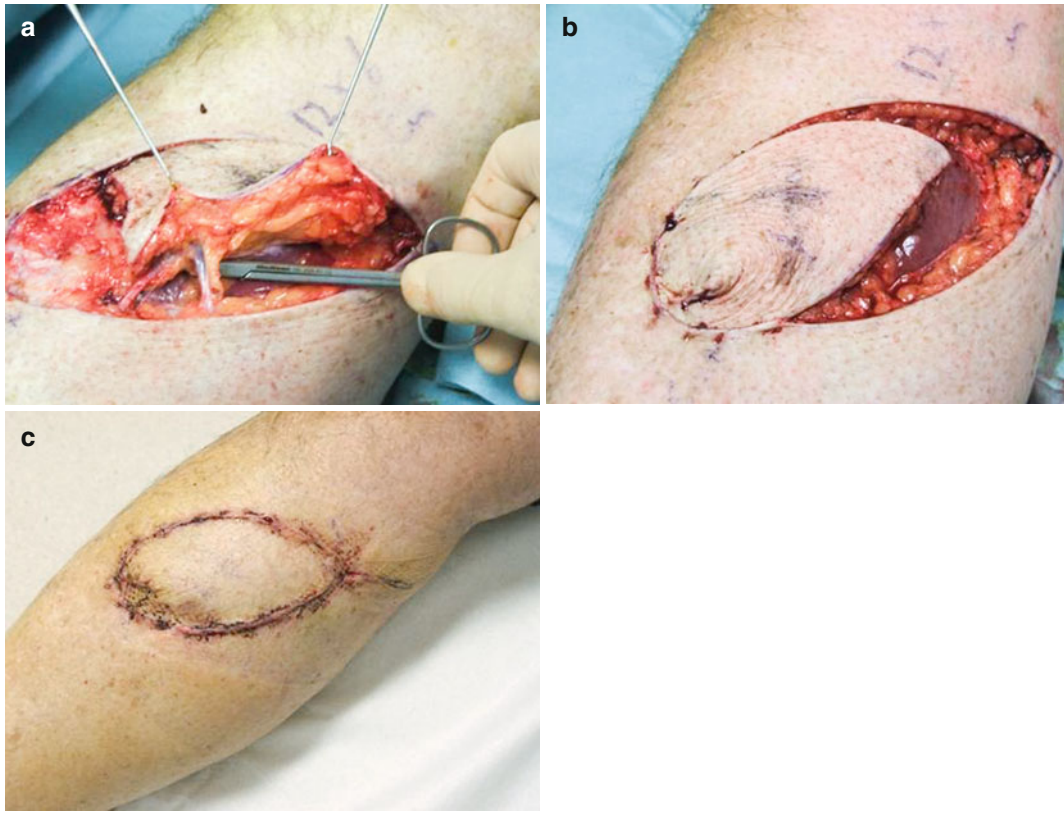


Fig. 5.15 (**a**–**c**) This is a small fascio-cutaneous island flap or it could be called a perforator flap which is mobilised fully on its perforator along with the vena commitans and advanced to fill this small defect

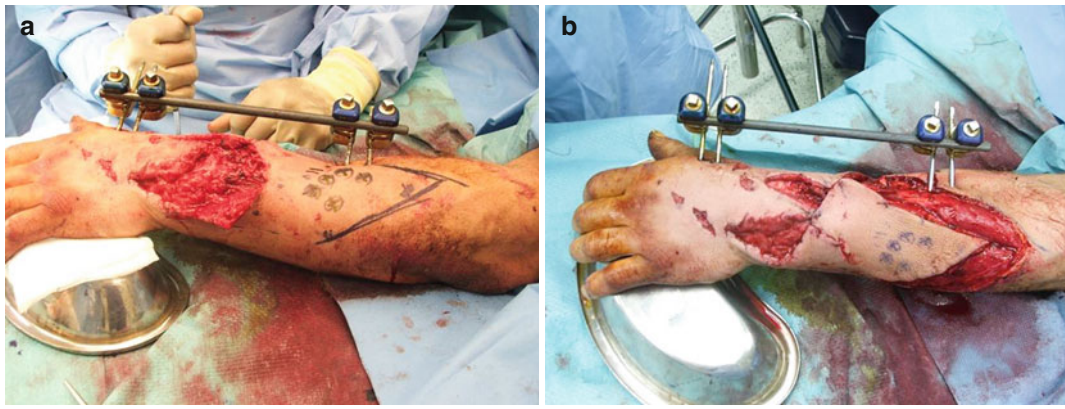


Fig. 5.16 (**a**) Defect on the dorsum of the arm with a small area of tendon and fracture exposed. (**b**) A perforator flap has been mobilised to cover the exposed tendon and bone, a split skin graft will be used to cover the rest of the wound

along the axis of the flap and the under surface of the flap. It can also be raised with vascularised bone from the radius. This fascio-cutaneous flap can be pedicled proximally or distally based or used as a free flap. Raising this flap sacrifices the radial artery input into the hand. This flap is extensively used as a donor free flap for intra oral reconstruction and has only very rarely been reported to cause a functional problem in the

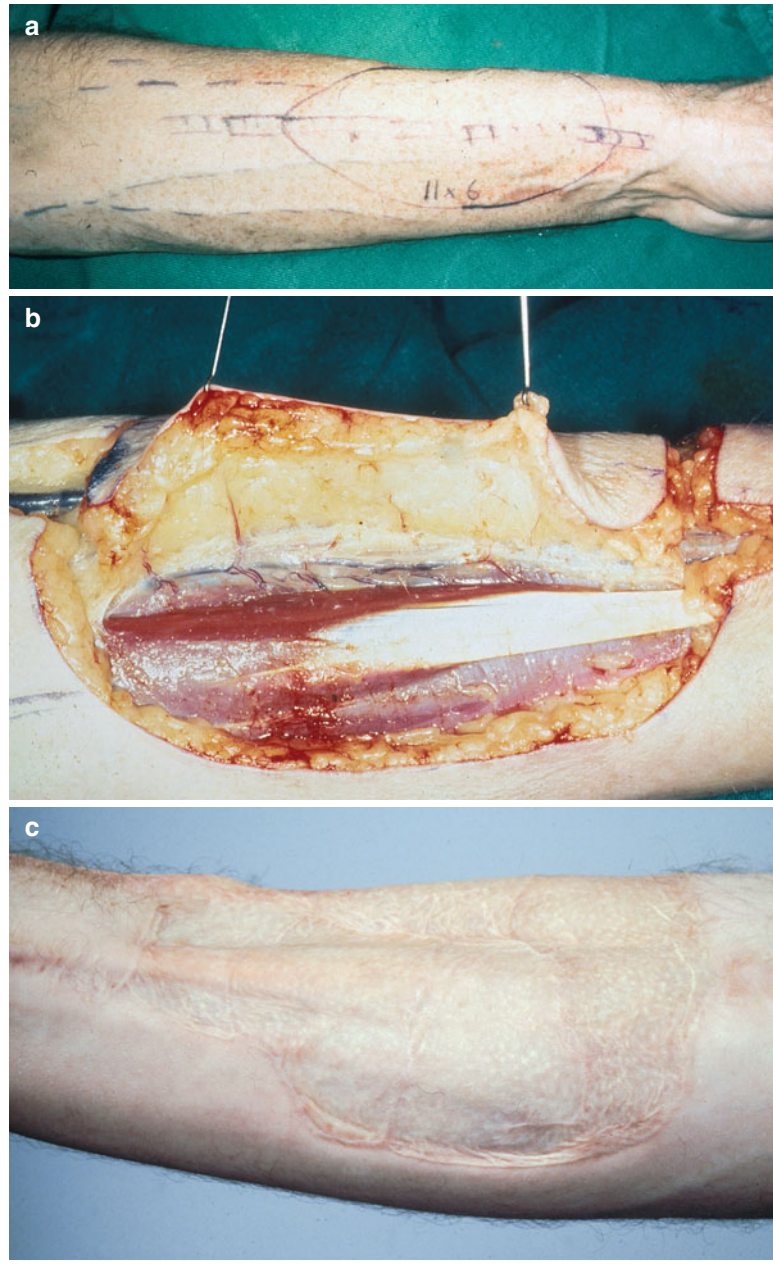


Fig. 5.17 (**a**–**c**) A demonstration of a radial forearm flap, its blood supply and then quality of the secondary defect after skin grafting. The cosmetic appearance of the secondary defect must be discussed with the patient pre operatively

hand. Before raising this flap a clinical Allen's test will confirm the dominance of the ulnar artery supply to the hand and obviate any concerns from dividing the radial artery. The flap secondary defect is skin grafted. Some patients quite reasonably object to the cosmetic appearance of this sunken skin graft on their forearm and this should be discussed with the patient before hand.

The flap is illustrated in Fig. 5.8 to include extensor tendon reconstruction using palmaris longus and longitudinal half of flexor carpae radialis. The following two examples demonstrate the potential of the radial forearm flap for reconstruction of the wrist, hand and fingers.

Figure 5.18a, b illustrates a long distally pedicled radial forearm flap used to resurface the Proximal Inter-phalangeal Joints (PIPJ's) of the left long and ring fingers after bilateral hand and other body burns. The pedicle is skin grafted and eventually the fingers separated. It is a large flap for this problem but without proper skin cover the extensor tendons will be lost and the PIPJ's will Boutonniere. In this patient with other burns flap options are limited. The final result is a full range of PIPJ movement Fig. 5.18c, d.

Figure 5.19a–e shows a distally based radial forearm flap to resurfaces the volar surface of the fingers and distal palm partially salvaging them. Without injuries to other parts of the body there would be alternative flaps that could be considered such as a pedicled abdominal flap or free tissue transfer but the final quality of the resurfacing is very satisfactory.

(b) **The Posterior interosseous island fasciocutaneous flap** (PIIF) [18] based on the posterior interosseous artery (PIA) and its perforators, is used as a distally based island flap or as a small free flap. The advantage of the flap is that raising it does not sacrifice a major artery into the hand. The potential disadvantage is its limited size and the scar it leaves on the dorsum of the forearm. It is a difficult

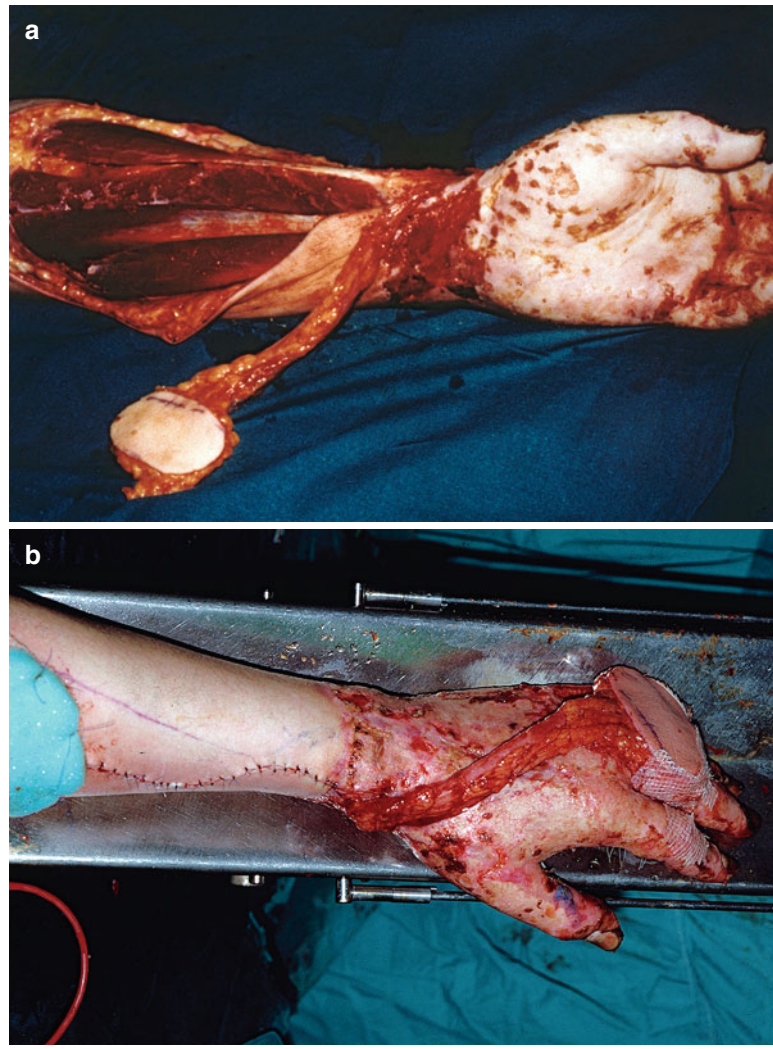
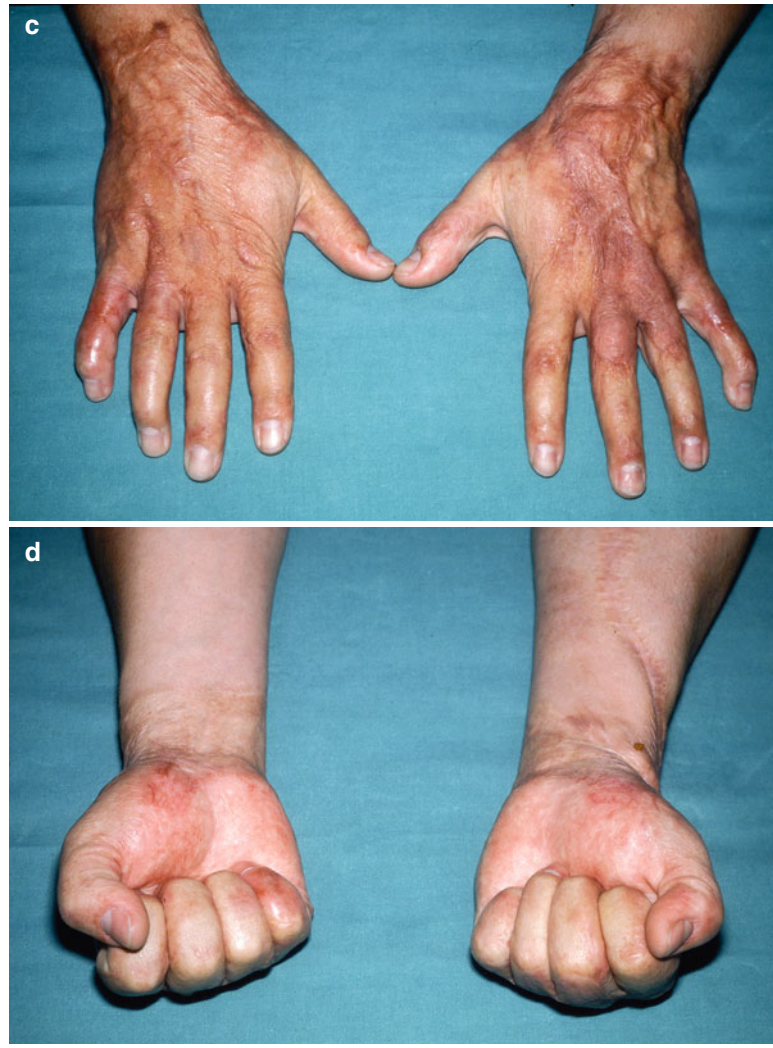


Fig. 5.18 (**a**, **b**) Shows a distally based pedicled radial forearm flap used to resurface the dorsum of the burnt Proximal Inter-phalangeal joints of the long and ring fingers. This flap requires multiple secondary operations to de-syndactylise the fingers and thin the flap. (**c**, **d**) Shows the final result and cosmetically and the preserved function of the Proximal Inter-phalangeal joints

Fig. 5.18 (continued)



dissection requiring ×3.5 loupe magnification. There are some patients where the PIA is anatomically defective but this will not be known until the artery is explored. Before dissecting this flap the arterial perforators on the dorsum of the forearm should be marked preoperatively so that if the PIA is defective an alternative flap can be planned. The key preoperative marking for the PIIF is to draw a line from lateral epicondyle to the ulnar head with the forearm pronated. The PIA lies in the septum between the finger extensors and the extensor carpi ulnaris. Caution is needed when there is thick subcutaneous tissue as it is easy to mark the skin paddle in the wrong place due to the mobility of the forearm skin and it is also easy to look for the PIA in the wrong muscle septum. As the dissection follows the PIA distally there is a low motor nerve branch to the extensor digitorum muscle to be preserved. The flap is transferred by rotating 180° on its long pedicle. The secondary defect and the inset over the pedicle may require covering with a SSG. Figure 5.20a, b shows a distally based PIIF on a long pedicle to reach the distal dorsum of the hand. There are cases where the skin on the dorsum of the wrist is slack and it is felt that the pedicle can be left in a tunnel but in most cases it is wiser to divide the dorsal wrist skin and inset the pedicle with thin SSG cover.

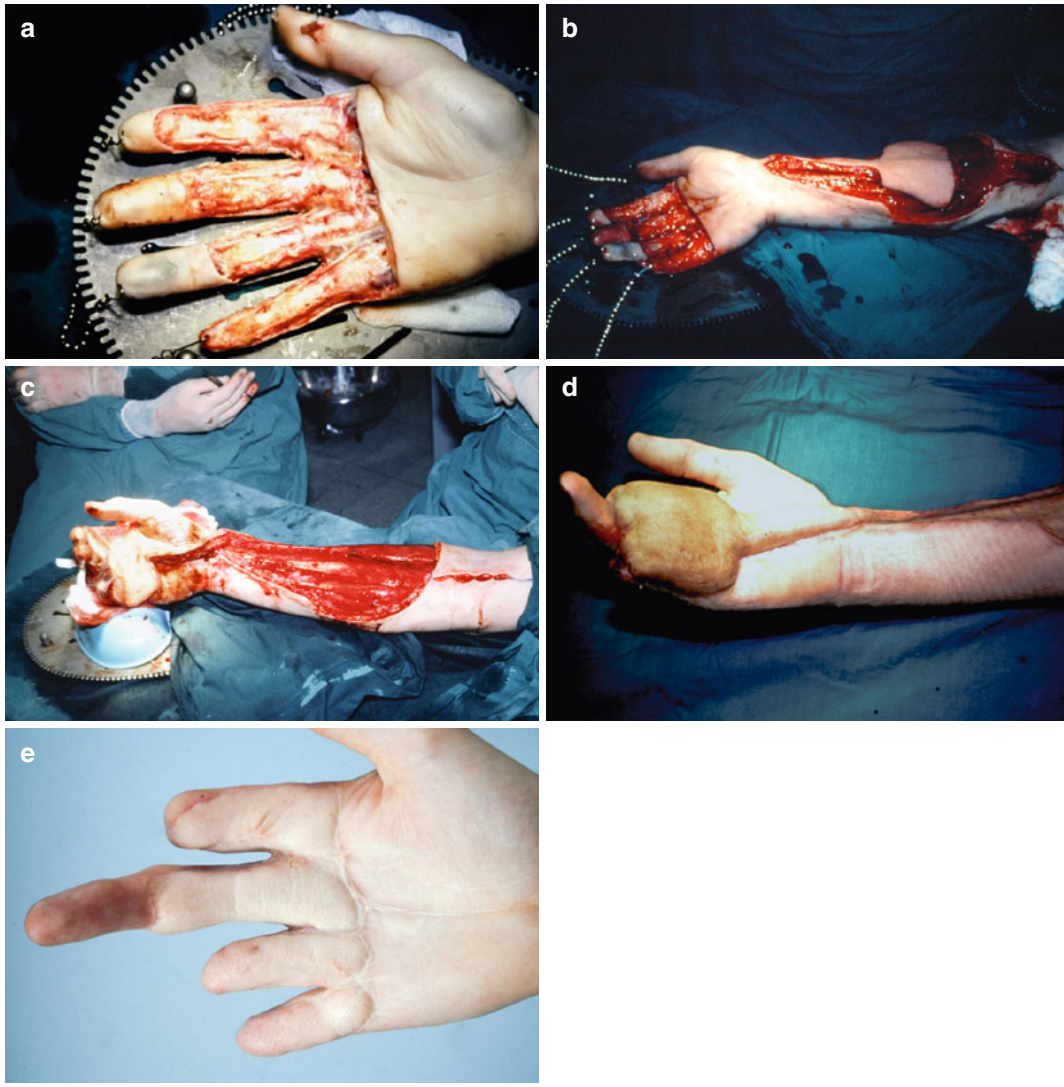


Fig. 5.19 (**a**) Shows a crush burn to the fingers and distal palm. (**b–d**) Illustrates the scope of the flap to reach distally on the fingers. (**e**) Shows the final result

The concept of rotating a flap 180°, so-called propeller flaps, is a neat cosmetic solution [19] but it requires judgement about the condition of the local tissues and the size of the perforator allows this rotation without compromising the blood flow in the pedicle. It also requires confidence in dissection.

(c) **The dorsal ulnar fascio-cutaneous flap** [20] is supplied by the recurrent dorsal branch of the ulnar artery and is a possible alternative to the posterior interosseous island flap for resurfacing the dorsum of the hand. The perforators must be mapped preoperatively. Figure 5.21a–e. shows the flap as a transposition flap with a broad base to cover exposed metacarpal fractures. This broad base produces a 'dog ear' much of which can be reduced when the flap in divided and inset giving an acceptable result. This flap could possibly be rotated 180° as a propeller on its vascular pedicle. This would be a neater inset but it is perfectly reasonable for a surgeon to adopt a cautious approach as illustrated above.

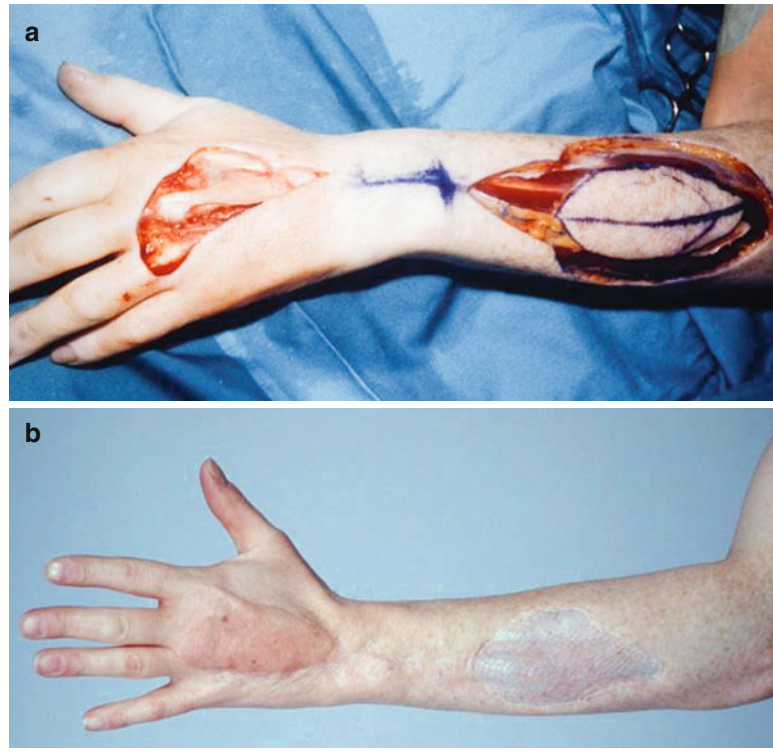


Fig. 5.20 (**a**, **b**) Shows a distally based pedicled posterior interosseous fascio-cutaneous flap to a small sized defect on the dorsum of the hand and the scarring it produces

Big defects around the elbow are suitable for a distally based flap based on recurrent arteries around the elbow these are well reviewed by [21, 22].

Adipo Fascial Turnover Flaps

Adipo fascial turnover flaps can be proximally or more usually distally based. They preserve the skin overlying the flap and raise the subcutaneous fat and the fascia only. The skin over the site of the adipo-fascial flap is raised with only a thin layer of fat and will subsequently be sutured back to the defect. The deeper fat and the fascia is then raised and transposed distally, 'like opening a book' and covered with a SSG. This is a neat solution for the dorsal thumb defect illustrated by Mark Pickford's case Fig. 5.22a–d. Careful to avoid dividing the radial sensory nerve.

Local **muscle transposition flaps** to fill small defects, surfaced by a SSG, are rarely used in the upper limb unlike in the lower limb where the gastrocnemius flap is extensively used to resurface the knee. Muscle function needs to be preserved in the upper limb and local fascio-cutaneous flaps or distant flaps are almost always a better option. Muscle flaps with skin graft cover are further discussed in the free flap section.

Distant Pedicled Flaps

Despite the evolution of fascio cutaneous flaps, perforator flaps and free flaps (below), distant pedicled flaps from the abdomen are still an important part of upper limb reconstruction and can be considered as a reconstructive option in many cases.

Inferiorly Based Abdominal Flaps

For anything other than a very small flap these are raised to include the superficial inferior epigastric artery (SIEA) making them axial pattern flaps and thus allowing a longer flap length to base width ratio of the flap. These flaps can be raised to transfer very large areas of skin both in width and length. Figure 5.23a shows a young man's hand with extensive skin loss and extensor tendon loss on the dorsum of the forearm and

Fig. 5.21 (**a**–**e**) Shows a broad distally based ulnar fasciocutaneous flap transposed 180° to resurface the dorsum of the hand with metacarpal fractures. Pre operative Doppler studies were performed to mark all the perforators on the dorsum. The intension was to raise a posterior interosseous island flap but the artery was deficient so this alternative flap was raised. The flap requires thinning and insetting but even so the cosmetic result is not good

hand. Figure 5.23b–d shows the feeding vessel of a wide flap trans-illuminated to show the vessels. Figure 5.23e, f shows the flap inset and being radically thinned by the technique of Raja Sabapathy and also show the final skin grafted secondary defect. His extensors were later reconstructed with two stage tendon grafts.

Intra-operative technique when raising a inferiorly based abdominal flap or a groin flap: having raised the flap the surgeon should first roughly inset the flap into the defect to check how the flap and the arm will lie after the final inset. The surgeon should then release these sutures and proceed to close the flap donor defect either

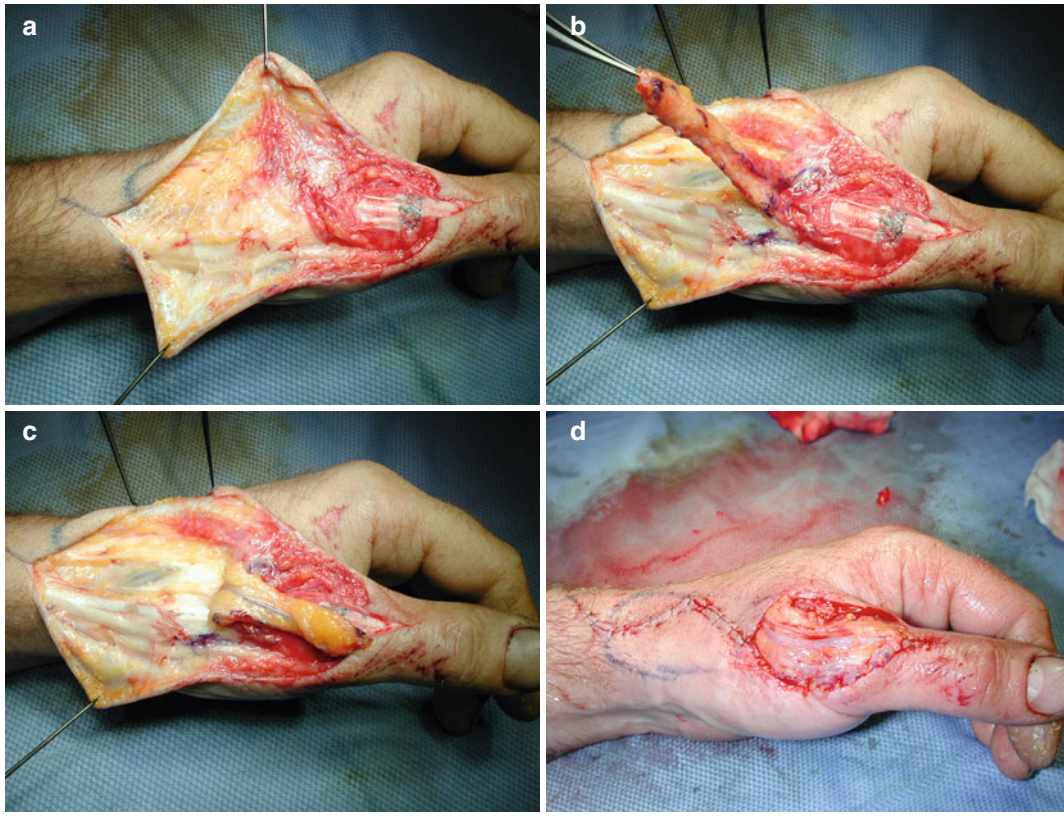


Fig. 5.22 (**a**–**d**) This distally based fascio-cutaneous turn over flap is a neat single stage solution to this dorsal thumb defect (Mark Pickford)

directly or by a SSG. If a SSG is used it can be placed over the base of the flap as well as the secondary defect to reduce the raw area. The flap is then re-inset concentrating on getting the maximum dermis to dermis contact of the flap to surrounding skin. A horizontal mattress suture is excellent for this. In fatter people the edges of the flap can be defatted to aid inset but the full thickness of the subcutaneous tissue has to be preserved around the central part of the flap and the SIEA.

Traditionally with good dermis to dermis inset these flaps are divided after 3 weeks. If doubt exists about flap "take" this time can be lengthened or the flap can be delayed. There is no exact science about this delay procedure and a cautious judgment has to be made depending on the dermis to dermis healing across the inset, the quality of the tissue healing in general and the extent of any extra tissue that is to be transferred (see extending the flap under pedicled groin flap below). If the flap is to be delayed the usual practice is to selectively ligate the feeding vessels to the flap and leaving the base of the flap intact. Seven to ten days later the whole flap is divided. This delay procedure induces relative hypoxia within the flap and will encourage neovascularisation from the native tissues into the flap. The author's practice is not to inset the flap at the time of flap division because of the risk of producing rim necrosis around the flap. A rim necrosis is less likely if the flap has had an arterial delay but the safest plan is to leave the flap inset open and close it a further week later or to allow natural healing in dressings.

Pedicled Groin Flap

The pedicled groin flap is an axial pattern skin flap based on the superficial circumflex iliac artery (SCIA) [23]. It remains a very important flap in upper limb reconstruction. The SCIA can

Fig. 5.23 (**a**) Large defect on the dorsum of the arm and hand with loss of extensor tendons. (**b**–**d**) A very large inferiorly based abdominal flap has been raised. You can trans illuminate it to see the feeding vessels. (**e**) The flap in inset and being radically thinned by the method of Raja Sabapathy (personal communications) where the flap is thinned from several incisions made around the perimeter. (**f**) Shows the abdominal scarring

be variable in its origin and it's course should be mapped with a Doppler if possible. The venous drainage is a superficial vein draining into the long saphenous vein and not a vena commitans of the SCIA. The standard surface marking for the artery is from a point arising two finger breadths below the inguinal ligament along the palpable line of the femoral artery medially to just below the anterior superior iliac spine (ASIS) laterally. Beyond this the flap becomes a random pattern flap and the skin should be designed on a 1:1 ratio width to length.

Some surgeons will perform a limited medial dissection of the flap first to establish the position

of the draining vein upon which to centre the flap but most surgeons will mark the standard position of the SCIA or the Doppler position of the SCIA and raise the flap initially from lateral to medial without a medial dissection first. Lateral to the ASIS in a thin patient the flap is raised at the junction of subcutaneous tissue and deep fascia. In a fatter patient the lateral part of the flap can be raised initially at the mid fat level shelving to full thickness as the ASIS is approached. If you are raising a very long groin flap it might be safer to raise it thicker rather than thinner. As you approach the ASIS beware not to damage the lateral cutaneous nerve of the thigh. Just medial to the ASIS the position of the SCIA should be seen or it can be trans-illuminated. If the vessel is not quite in the axis drawn the flap markings can be altered a little but once the position of the axial artery is determined the flap can be thinned at the edges to reduce flap bulk and help flap inset.

As a pedicled flap the groin flap does not usually have to be raised medial to the lateral border of the sartorious. As a free flap loupe dissection proceeds under the deep fascia, to protect the SCIA, up to its origin from the femoral or profunda femoris artery. The artery is usually less than 1 mm in diameter. The pedicled groin flap is a versatile flap which can be used for moderately large defects or small defects. It is generally regarded as a very cosmetically acceptable donor site.

Figure 5.24a–e shows a crush and full thickness burn of the skin, flexors and pulleys on the volar surface of four fingers: a difficult defect and reconstructive problem. The pedicled groin flap 'heals' the defect but required about 8 operations to separate the fingers and thin the flaps, some combined with two stage flexor tendon grafts and pulley reconstruction. This was a long series of operations for this young female patient but the final result is excellent and the donor scar confined to the groin. A free flap reconstruction might have been an alternative option.

Figure 5.25a, b is a neonate with an extensive defect on the forearm and hand from a tissued drip which clearly required a flap. A very long pedicled groin flap and SSG were used in this 2 month old child. Figure 5.25c–e shows an excellent long term result. See below for a discussion on the difficulties of pedicled flaps. The secondary defect must be closed with two layers of buried sutures to avoid suture marks.

Extending the Medial Length of a Pedicled Groin Flap or Abdominal Flap

Both the pedicled groin flap and the inferiorly based abdominal flap can be lengthened at their medial end if more skin is required to resurface a defect. This has to be done with careful use of the delay process. Figure 5.26a shows how this being done in an 8 year old child with a near circumferential congenital naevus on the thumb. Initially the ulnar and volar naevus was excised and attached to a pedicled groin flap Fig. 5.26b, c. The arterial supply to the flap was ligated at 3 weeks as a delay procedure as discussed above (not illustrated) then a week later the flap was divided with extra skin to provide a long tube Fig. 5.26d. A week later again the tube was opened and loosely inset Fig. 5.26e. A further week later the rest of the naevus on the radial and dorsal surface was excised Fig. 5.26f and fully inset Fig. 5.26g. It might be argued that this was an unnecessarily cautious approach and indeed it might have been but when a flap has had an axial arterial supply it is susceptible to a rim necrosis until it becomes randomly vascularised.

Cross-Arm Flaps

An unusual but useful use of the contralateral arm is as a de-epithelialised cross arm flap to resurface the dorsum of one or more fingers simultaneously. A case with a dorsal finger injury or several finger injuries over the distal phalanx exposing tendon and bone of the distal phalanx is a difficult reconstructive problem. A solution is to de-epithelialise an area of skin on the inner aspect of the opposite upper arm and to suture the exposed distal fingers to this de-epithelialised area. The arms are strapped together for 10–14 days then divided leaving the subcutaneous aspect of the 'flap' on the injured fingers. This needs to be subsequently thinned and covered by a SSG but therapy can be started on the dipj and pipj as soon as the 'flap' is divided. The donor arm is closed primarily.

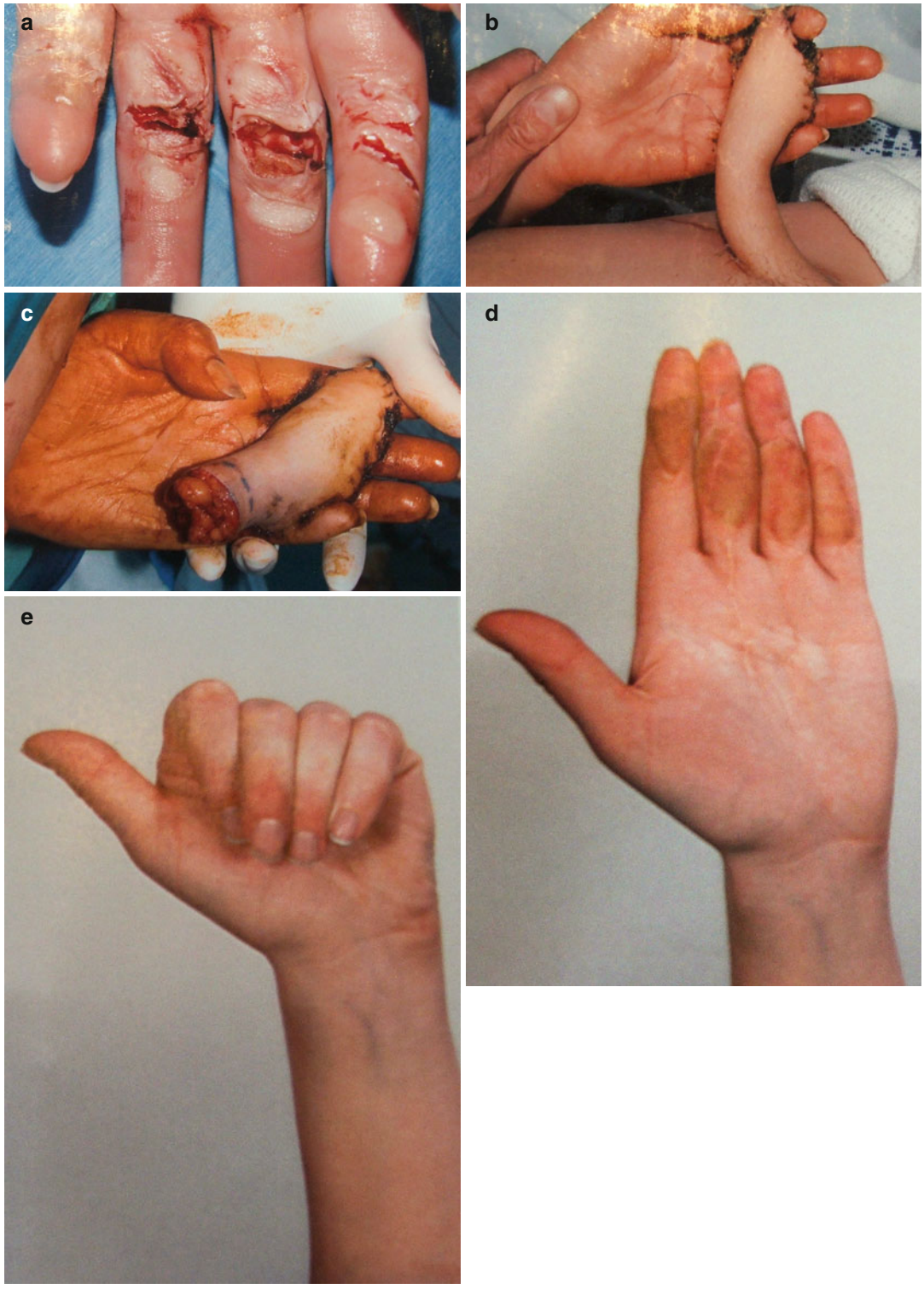


Fig. 5.24 (**a**) A four finger volar crush and full thickness burn of skin, flexor tendons and pullies. (**b**) The four fingers have been resurfaced by a pedicled groin flap. (**c**) The flap divided at 3 weeks. (**d**, **e**) The final result after many thinning operations and tendon grafts

Fig. 5.25 (**a**, **b**) Extensive near circumferential defect in a child in the first month of life from a tissued drip. (**c**) The defect was resurfaced with a pedicled groin flap. This is the appearance at 6 months post op. (**d**) Appearance at 6 years. (**e**) Appearance age 16 years

Micro Vascular Free Flap Transfers

Free microvascular tissue flaps transfer skin or composite tissue into an injured area in a single operation. These flaps require team work and microvascular expertise. The patient has to be medically fit for the microsurgery and understand the cosmetic implications. When all the flap options are considered if the micro vascular free tissue transfers are chosen then their versatility, easier post-operative wound care, ease of access for hand therapy care and splintage, shorter hospital stay and time to healing usually make them the solution of choice. The choice of flap requires careful consideration. It depends on the size and geometry of the defect, what type of skin and other tissue is required, the zone of injury and the position of the potential donor artery and vein. Free flaps offer the possibility of chimeric reconstruction where skin, fascial tissue and bone can be transferred together and independently inset into a compound defect.

Full descriptions of flap anatomy and flap elevation techniques may be sought in many published texts and papers. The aim here is not to repeat these excellent texts but to help the reader with practical thoughts on flap selection and technique.

Skin and Fasciocutaneous Free Flaps

1. The **free groin flap** was the original free flap [23]. In thin patients it is an excellent flap, in more obese patients harder to use. Marking and raising the flap has been discussed above. The flap artery is generally less than 0.9 mm which makes the medial dissection of the flap

Fig. 5.26 (**a**) Near circumferential congenital naevus on the thumb. (**b**) The ulnar and volar naevus excised. (**c**) A pedicled groin flap raised. (**d**) An arterial delay was performed then a week later the tubed groin flap was divided long but not inset at the time of division. (**e**, **f**) After another week the blood supply of the tissues seemed robust enough to allow the residual naevus on the dorsal radial surface of the thumb to be excised and tube to be opened and inset. (**g**) Final appearance

Fig. 5.26 (continued)

a difficult dissection in comparison to other flaps that are discussed below with larger vessels. The flap pedicle is short so the flap must be inset directly over the donor artery and vein to which it is to be anastomosed. One important practical point is to mark the flap artery and flap vein before they are divided with different distinctive 9/0 sutures in such a way that the two can be easily found and distinguished from each other when the flap is being inset. The artery is short and can retract into the subcutaneous tissue of the flap and be difficult to find. The vein can still be confused with the artery and should be marked distinctively when doing this and any free flap. The arterial anastomosis is usually end to side to the radial artery and the vein end to end with a superficial vein but the method of anastomosis has to be judged at the time. Figure 5.27a shows a free groin flap after 20 year. Figure 5.27b shows the donor site scar at the time of the original closure. The patient is slim but the flap has had one thinning procedure. The donor site shows how the scar can be kept short in free groin flaps.

2. The **radial forearm flap**. This flap discussed as a pedicled flap can be used as a free flap. There are cosmetic issues as discussed before but not functional issues. It provides a good skin paddle and a long pedicle. It is perhaps one of the quickest and securest flaps to raise. It provides a good skin paddle which is pliable. Because it uses a major artery it is very suitable to provide an arterial reconstruction as a 'run through' as well as providing skin cover. An alternative reconstruction would be a thinned perforator flap and arterial reconstruction based on the dorsal scapular axis.
3. The **anterior thigh flap** [28, 29]. The arterial supply is a perforator from the circumflex femoral artery. In the standard marking of the flap the positioning of the pedicle is towards the center

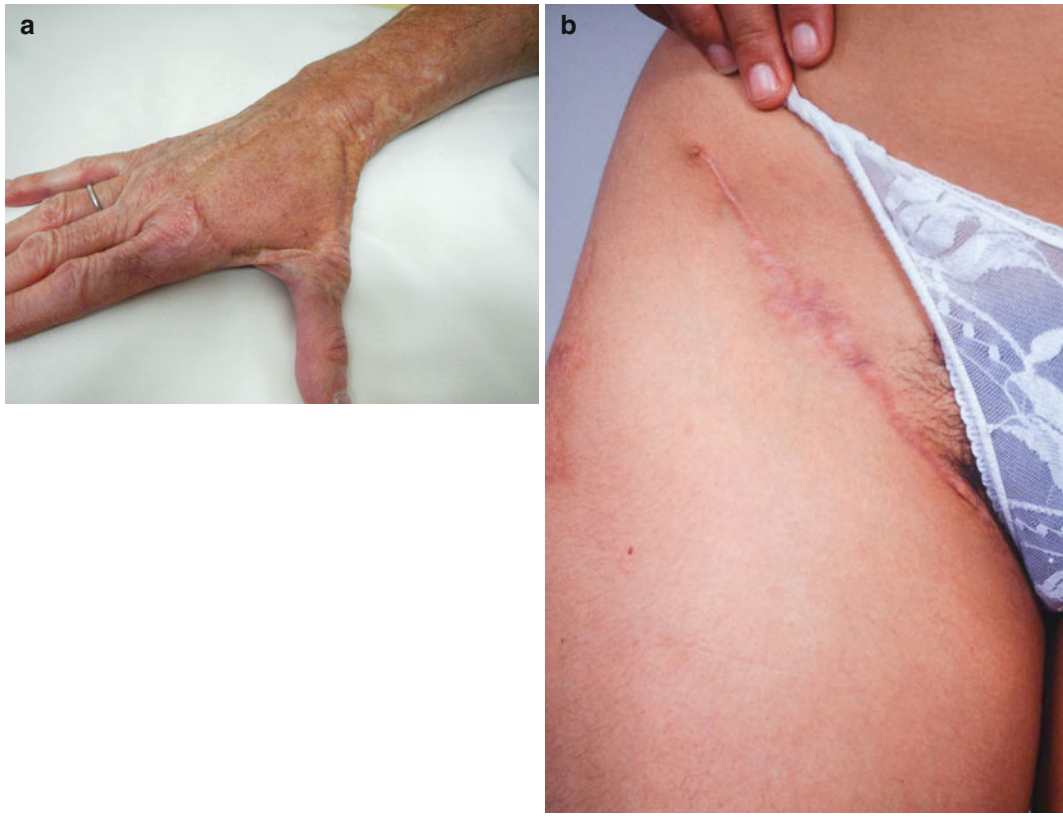


Fig. 5.27 (**a**) The appearance of a free groin flap 20 year after transfer. (**b**) The appearance of the free groin flap donor site after the original transfer. Note the short donor scar

of the flap. It has a variable pedicle length but can be up to 6 cm Fig. 5.28a–c. In many patients it is a naturally thin flap but this and other perforator flaps have been developed to be thinned as they are raised [24, 25]. Thinning perforator flaps is a valuable advance in flap technique but it does add another level of complexity to the dissection and the technique which has to be mastered. It makes them suitable in many patients for resurfacing the upper limb. Figure 5.28d–f shows an anterior thigh flap resurfacing the dorsum of the hand.

If a defect requires a longer flap and pedicle length to resurface a defect and reach the donor vessels more experience with the flap design is required than just the standard markings. In such a case it might be necessary to dissect the descending branch of the lateral circumferential artery intra muscularly to the next perforator. The surgeon needs to consider their experience of the flap against perhaps changing to another flap with better geometry to suit the defect in front of them. The donor scar of the anterior thigh flap is exposed on the front of the thigh and the donor scar quality depends on the size of the flap taken and whether a SSG is required. The patient has to be aware of the final level of scarring of the flap donor site.

4. **Lateral arm fascio-cutaneous flap** is a long narrow flap with a long arterial pedicle at its proximal end giving the flap a good reach. It may be raised from the ipsilateral arm to confine the scarring to one arm. If the flap is narrow the donor site can be closed primarily otherwise a SSG may be needed. Figure 5.7 shows the flap in use. For a patient who likes to show their arms it is not a suitable flap because the donor scar is both visible and sunken, Figure 5.29 shows the cosmetic

Fig. 5.28 (**a**) Standard markings of the anterior thigh flap. (**b**, **c**) shows the flap raised and its pedicle. (**d**–**f**) Shows the defect, flap in place and the thigh defect

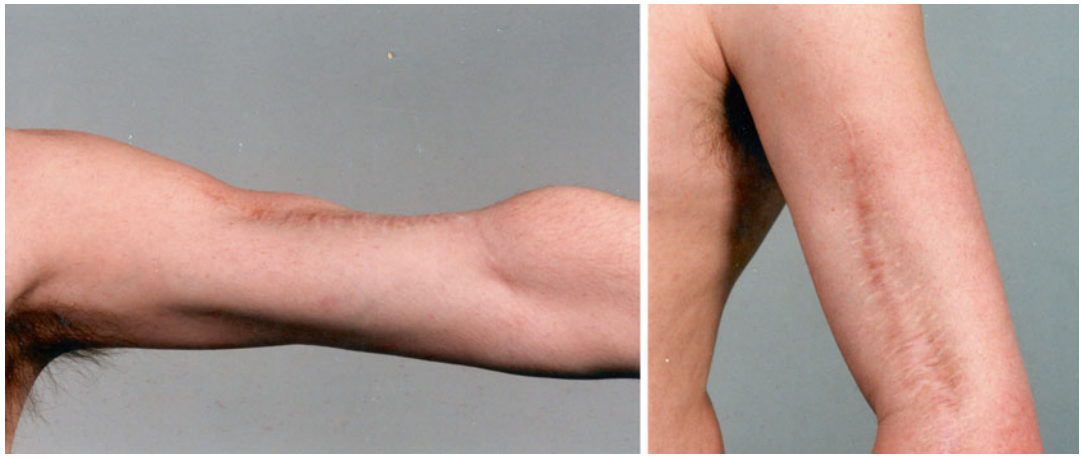


Fig. 5.29 Shows a typical appearance of a lateral arm flap donor site

appearance of the flap donor site. Disregarding the cosmetic side, if the geometry of the defect suits the flap it is a relatively easy flap to raise with good diameter vessels. One word of caution is to be aware of the radial nerve which may be damaged with careless proximal dissection as the vascular pedicle is followed into the humeral spiral grove to maximize the length of the pedicle. If a narrow tourniquet is to be used to aid the dissection it is important to minimise tourniquet time and pressure to prevent damage to the nerves.

5. **The dorsal circumflex scapular artery axis**. The original flaps on this axis were the **Scapular flap** and the **parascapular flap**. Figure 5.6c illustrates the use of the parascapular flap. They are long flaps and have a long reliable vascular pedicle at their end making them suitable when the geometry of the defect requires this length. In their original form they may be bulky and the skin itself thick and possible hirsute. The patient may need to be repositioned to raise the flap. This flap axis has been developed by [26] to raise the scapular and parascapular together as one continuous flap suitable for long defects with areas up to 257 cm^2. With this flap configuration the pedicle is towards the center of the flap. This axis has been further developed to include the **circumflex scapular artery perforator flap** [13, 26, 30], which is an important development of the concept of perforators flaps. Not only does it give the possibility of a skin paddle raised at the thickness of Scarpa's fascia and possibly further thinned primarily but it develops the concept of the chimeric construction of free flaps. The vascular axis has the potential to construct a flap with skin, fascial tissue and bone each on separate branches of the parent pedicle. The scar on the back will stretch in width if it is transverse but will be better quality where it is a vertical scar. Figure 5.30a–d shows a dorsal scapular axis flap with a double skin paddle and a vascularized bone graft to a three dimensional defect from an explosion, on the hand.
6. **Medial plantar flap** is an excellent example of a "like for like" flap tissue to resurface a defect in the palm. The secondary defect on the sole of the foot, however, is controversial and has to be considered very carefully. Potential complications include a hypertrophic scar on the inset of the foot, neuroma formation and cold intolerance.
7. **Tensor fascia lata flap i**s a large and long flap on a long robust pedicle, the transverse branch of the circumflex femoral artery. The flap is reliable and can be raised without turning the patient but it is a thick piece of tissue and flap donor is cosmetically noticeable. Now there is

Fig. 5.30 (**a**) Dorsal scapular axis flap. (**b**) Pedicle of the dorsal scapular axis flap and a vascularised bone graft on a separate pedicle of the vascular axis. (**c**) Volar flap in place and the vascularised bone graft at the base of the metacarpal fracture. (**d**) Dorsal aspect of the flap

greater choice of potential flaps this flap would seldom be required.

8. **Temporal fascia free flap**. This flap provides a small area of tissue but needs to be covered by a skin graft hence it will have the properties of a skin graft as discussed with muscle flaps. It can be a solution to a small defect over tendon, nerve, joint or in the palm which cannot be directly skin grafted. The donor involves a scar on the temple and the author has seen damage to the forehead branch of the VII nerve and follicle loss. Its choice should be weighed against alternatives.

Myo-Cutaneous Flaps

Latissimus dorsi flap. This flap has perhaps the longest and most reliable of flap pedicles, the thoraco-dorsal pedicle. The muscle can be raised to within 10 cm of the posterior iliac crest with or without a large skin paddle. A small flap can also be taken on this pedicle possibly preserving some latissimus dorsi function but this complicates the dissection and with the advent of perforator flaps small latissimus dorsi flaps would seldom be indicated. In a muscular patient the thickness of the muscle as well as the thickness of the back skin can produce an unsatisfactory appearance even after atrophy of the denervated muscle. It can be thinned at a later date but this will require radical thinning of the muscle and skin grafting. The young patient in example Fig. 5.31a–d has a neglected gunshot wound to the arm with a contracted elbow and a long zone of injury to the vessels. In a case like this a very secure reconstruction is needed and a long pedicle to get outside the zone of injury. A sufficient skin paddle has been taken to prevent a contraction across the cubital fossa and the rest of the muscle was grafted. Because she was not too muscular the final result is acceptable.

Fig. 5.31 (**a**) Contracted elbow from a neglected gunshot injury. (**b**) Defect after contracture release. (**c**) A latissimus dorsi flap raised and ready to transfer. (**d**) This flap has the size and pedicle length to securely resurface this wound but it will always be bulky

Non innervated muscle free flaps, using an expendable muscle like the gracilis with skin graft cover can be used for filling and resurfacing small defects. This method of reconstruction has two potential disadvantages. Firstly it will have the properties of a SSG and contract and in children the flap will not grow as a full thickness skin paddle would. Secondly if access is needed under the flap for secondary surgery it may not be so easy to re open and re close the wound as it would be with a full thickness skin paddle. The child in Fig. 5.32a, b suffered a supra condylar fracture and vascular damage resulting in loss of most forearm flexors and a volar skin defect. The initial wound healing was achieved by using a free rectus abdominus muscle flap covered by a skin graft. The choice of the rectus abdominus flap is controversial because of potential functional problems in the abdomen and the lack of a skin paddle. With the advent of perforator flaps this flap would probably not be chosen but it did achieve wound healing. The child developed a progressive volar wrist contracture with growth which was partly due to the internal scarring and partly due to the volar skin graft and underwent an inferiorly based abdominal flap to resurface the forearm.

Free Innervated Muscle Flaps

Latissimus dorsi flap. The pedicled latissimus flap is an ideal reconstruction for absent or denervated biceps. It can be islanded and its origin and insertion reattached. To judge the length and tension on the muscle at inset the muscle is marked with multiple sutures every 5 cm while the muscle is out to length before it is detached. A skin paddle will most likely be needed to get primary closure over the muscle bulk.

Gastrocnemius muscle. The child in Fig. 5.32d underwent a free gastrocnemius muscle flap with a skin paddle to motor the flexor tendons. With progressive growth there was a constant battle with volar wrist contracture Fig. 5.32e requiring a second abdominal flap and flexor tenolysis. Eventually, he had a proximal row carpectomy and wrist fusion to produce a

stable situation with a final range of movement as shown Fig. 5.32f, g. Normal sensation in the hand was preserved.

Gracilis muscle. The gracilis muscle flap is the work horse for biceps and forearm muscle reconstruction [31]. Figure 5.33 shows a child with upper limb arthrogryposis with a good passive range of movement of the elbow who underwent a right biceps reconstruction when 12 kg in body weight with a free innervated gracilis transfer. The muscle was innervated by an intercostal nerve. Good active range of movement of the elbow was achieved.

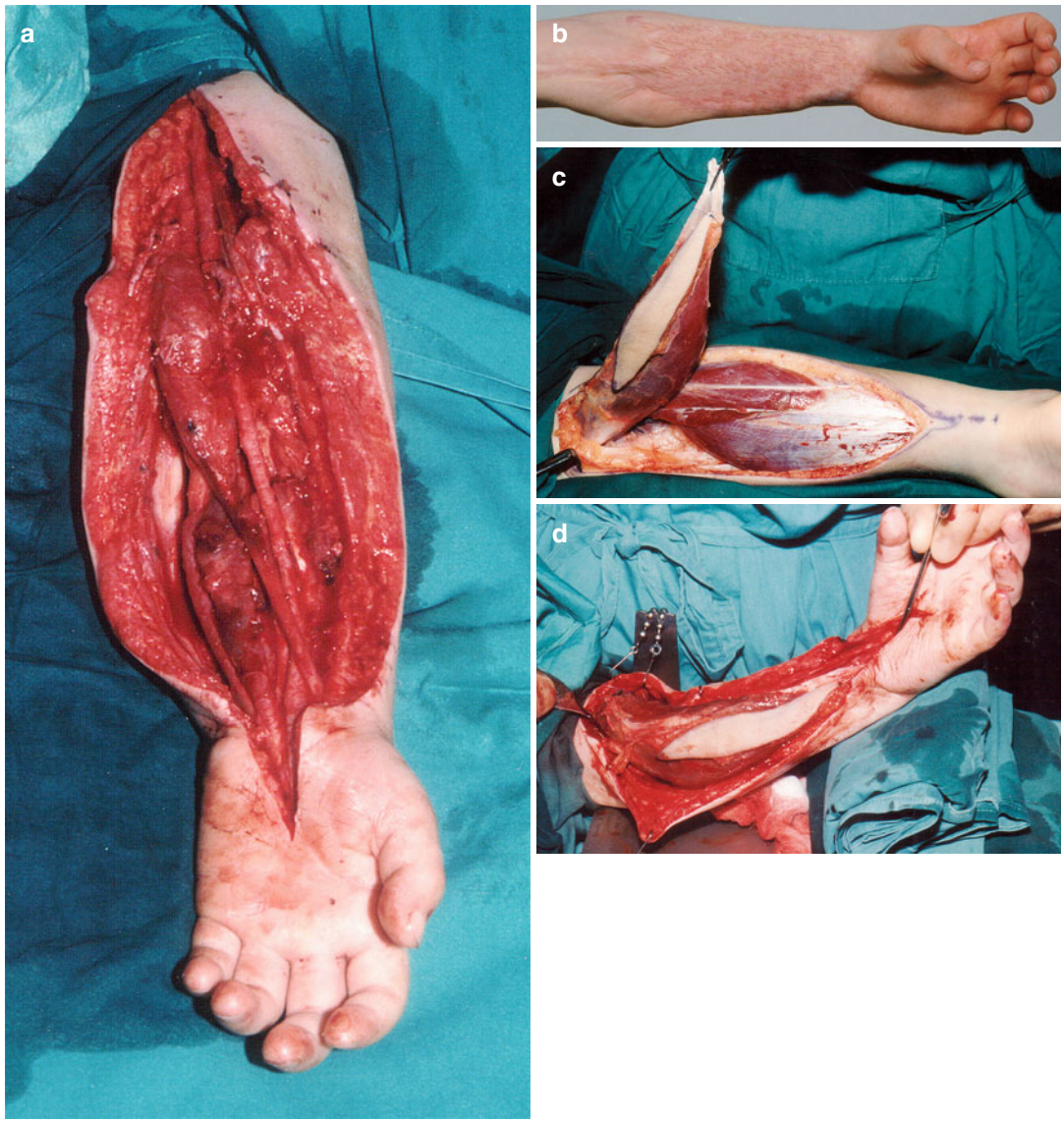


Fig. 5.32 (**a**, **b**) This child had a supra condylar fracture resulting in loss of the forearm flexor muscles and a skin defect which was reconstructed by a free rectus abdominus muscle flap and SSG. (**c**, **d**) After an abdominal skin flap he underwent a free innervated gracilis muscle flap to animate the forearm muscles. (**e**–**g**) The child battled with a forearm volar contracture for many years. The final solution was a proximal row carpectomy and wrist fusion after which he had the function as illustrated. Sensation remained normal thoughout

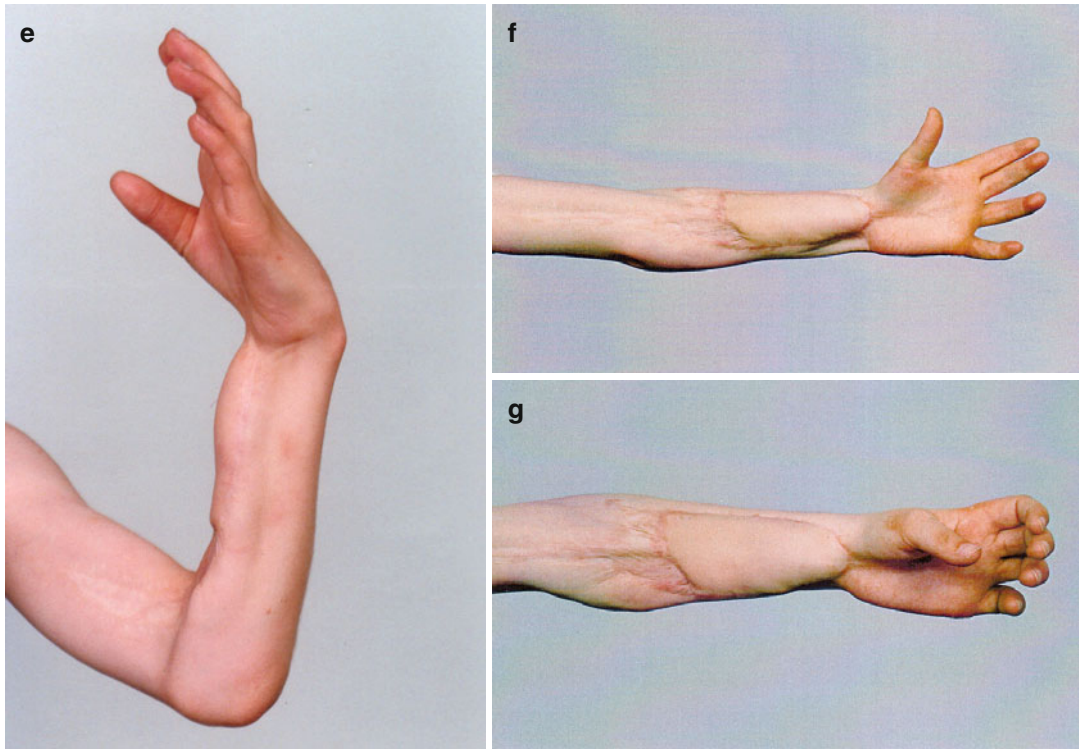


Fig. 5.32 (continued)

When Multiple Flaps Are Needed

There are times when multiple flaps may be required for upper limb reconstruction in the early reconstruction period. The sequencing and choice of flaps has to be carefully planned [8] has been referenced above in respect of the VAC care of wounds of modern warfare. In their paper they were faced with covering two upper limb wound and achieved this successfully with two simultaneous pedicled abdominal flaps.

The case Figure 5.2e shows a massive crushed and dirt ingrained forearm injury before and after debridement and bone replacement. A free latissimus dorsi flap was used but the distal end of the flap was not well perfused so rather than risk poor healing over the free bone graft a pedicled groin flap was used to cover the distal end of the wound at the same operation. The result was primary healing. Whilst this case, in common with the Tintle pedicled flap cases, has all the post-operative nursing and therapy difficulties of a pedicled flap the ultimate cover is good quality skin allowing for future surgery and for growth.

There may be a need for two flaps in some situations not only because of the size of the defect but because the need for a second flap may be become apparent after some aspect of the initial reconstruction fails. Figure 5.5 illustrates how the reconstruction plans have to evolve. Above all the message is, you have to constantly examine and analyses a case in the early stages and be prepared to change and add reconstructions until you have done the maximum reconstruction in this early period. The degree of persistence necessary to obtain the best possible result is seen in case Fig. 5.5. Figure 5.34a–d illustrates the planning and evolution of and a reconstruction in a severe hand injury. A free contralateral forearm flap was used to reconstruct the palm and dorsum of the hand at the time of attempted replantation of the thumb. The replant failed so

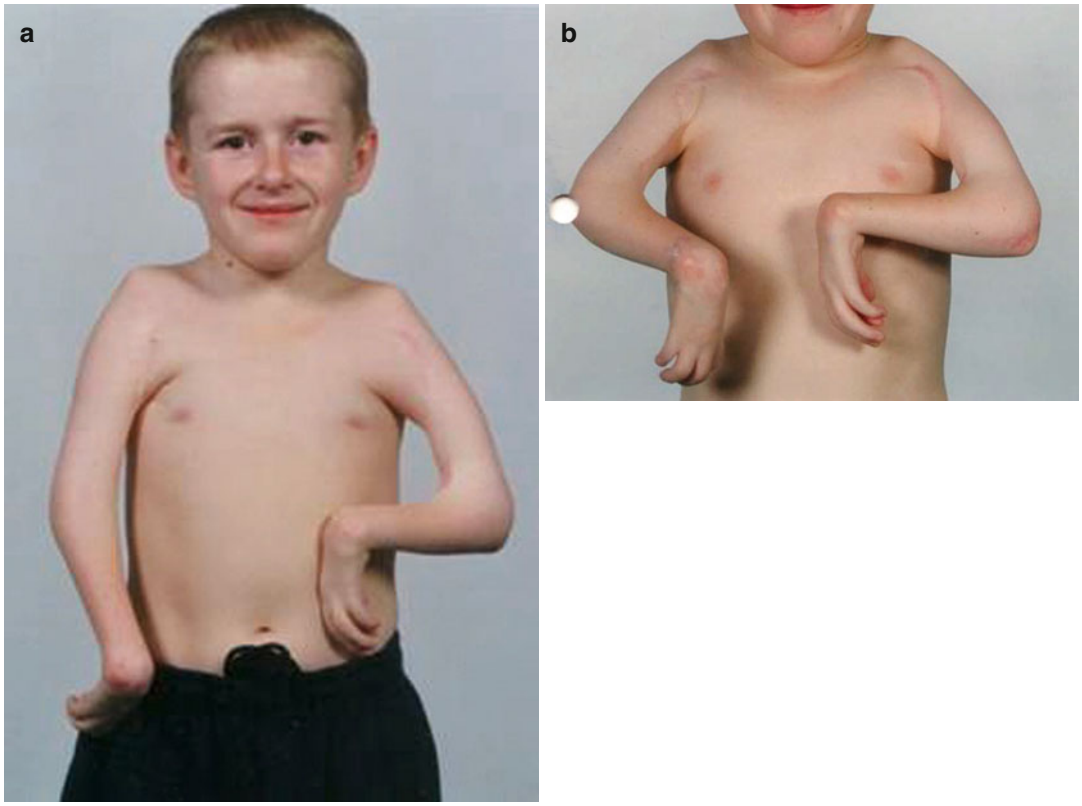


Fig. 5.33 (**a**) This boy suffers from *right* upper limb arthrogryposis and no biceps function. (**b**) The *right* elbow is powered by the free gracilis muscle

reconstructive options were discussed with the patient to maximize his hand function. The patient wished to proceed to a thumb reconstruction so a pedicled groin flap was used to resurface the radial side of the hand and prepare the ground for a toe to hand transfer. Figure 5.34e, f illustrates the final reconstruction with a hallux transfer which has kept him in a manual job for 23 years. The pedicled groin flap has the disadvantage of bulk but the combination of the two flaps, initially of course not intended, has worked very well.

How to Make the Decision on the Choice of Flap?

There are many considerations in any particular case and decisions must be patient centered. The choices offered to the patient are largely dependent on the geometry, size of the defect and type of tissue needed. If cosmesis is important to the patient this can be the biggest decision making factor in your case.

One of the difficulties with a pedicled flap is looking after the injured arm and hand during the 3 or 4 week attachment period especially in complex injuries like the replants illustrated in Figs. 5.5 and 5.34. The period of attachment is difficult for the patient and requires obsessive care by the medical staff. It is difficult to get adequate access for wound care of the injury site and donor site and it is difficult for the therapists to care for the injury site and also to maintain movement in the patients shoulder, elbow and wrist. The groin flap allows a little more movement of the arm than the broad based abdominal wall flap during the attachment period. But despite these difficulties they can be made to work well by a dedicated team of nursing, therapy and medical staff. In contrast a free tissue transfer requires technical expertise and

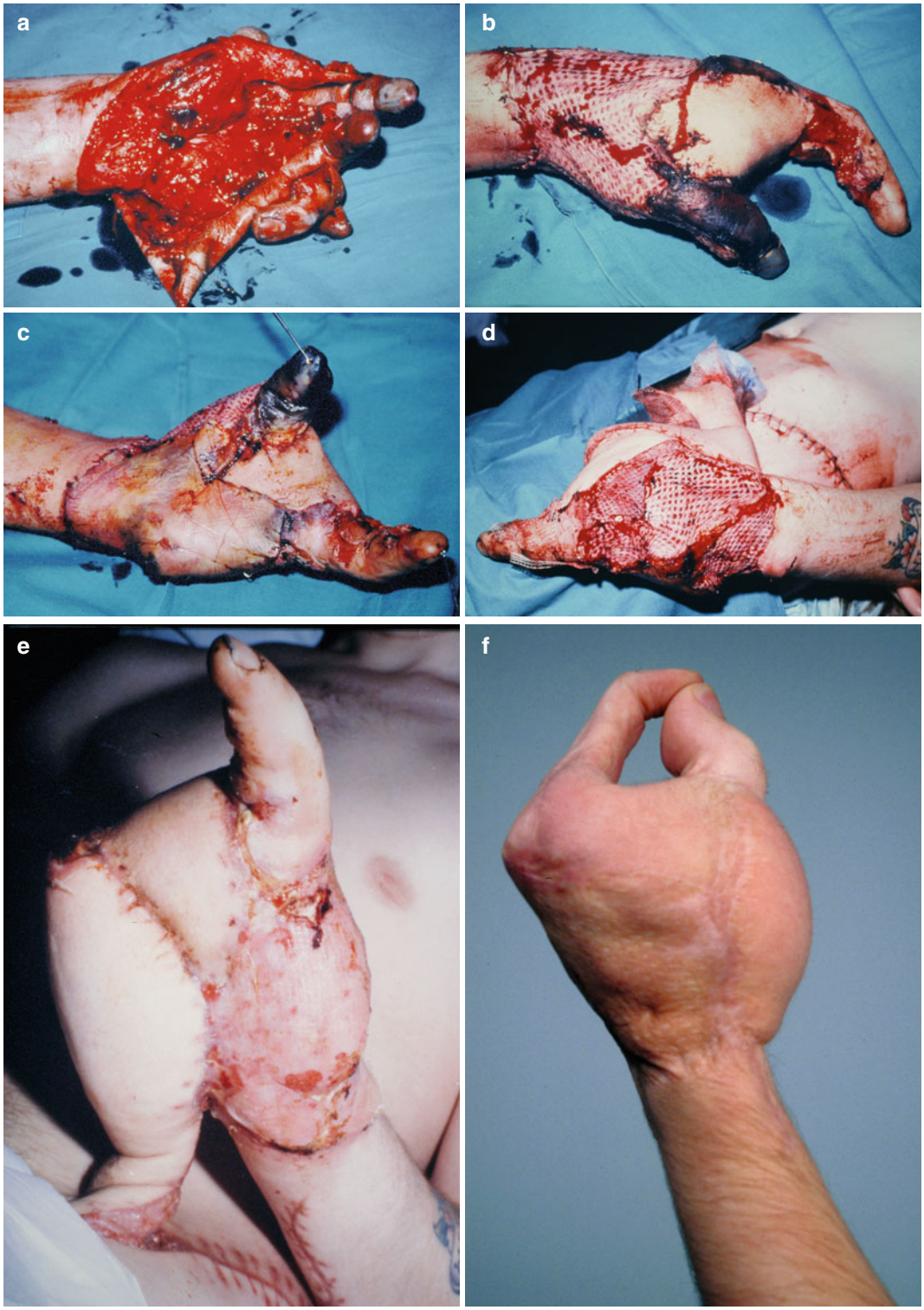


Fig. 5.34 (**a**) circumferential degloving of the hand. (**b**, **c**) Free contra-lateral forearm flap and failed thumb replant dorsal and volar views. (**d**) Pedicled groin flap to prepare for a toe to hand transfer. (**e**) Groin flap ready to divide. (**f**, **g**) Reconstruction after hallux to hand transfer

Fig. 5.34 (continued)

team work but postoperatively the arm can be elevated and it is much easier to attend to wound care and therapy care.

References

1. Mowatt DJ, Shah M, Watson JS. Palmar resurfacing techniques: an "ideal" opportunity and the importance of long-term follow-up. J Hand Surg Br Eur. 2002;27B(2):198–201.
2. Lohman RF, Nawabi AS, Reece GP, Pollock RE, Evans GR. Soft tissue sarcoma of the upper extremity: a five year experience at two institutions emphasizing the role of soft tissue flap reconstruction. Cancer. 2002;94:2256–64.
3. The management of open fractures. British orthopaedic Association and British Association of plastic surgeons. Published by the British orthopaedic Association; 1997.
4. Nanchalal J, Nayagam S, Khan U, Moran C, Barrett S, Sanderson F, Pallister I. Standards for the management of open fractures of the lower limb. London: The Royal Society of Medicine Press; 2009.
5. Argenta LC, Morykwas MJ. Vacuum-assisted closure: a new method for wound control and treatment: clinical experience. Ann Plast Surg. 1997;38:563–76; discussion 577.
6. Glass GE, Nanchalal J. The methodology of negative pressure wound therapy: separating fact from fiction. J Plast Reconstr Aesthet Surg. 2012;65:989–1001.
7. Greer SE, Longaker MT, Margiotta M, et al. The use of subatmospheric dressings for the coverage of radial forearm free flap donor-site exposed tendon complications. Ann Plast Surg. 1999;43:551554.
8. Tintle SM, Wilson K, Mckay PL, Andersen RC, Kumar AR. Simultaneous pedicled flaps for coverage of complex blast injuries to the forearm and hand (with supplemental external fixation to the iliac crest for immobilization). J Hand Surg Eur Vol. 2010;35E(1):9–15.
9. Weigert R, Choughri H, Casoli V. Management of severe hand wounds with integra dermal regeneration template. J Hand Surg Eur Vol. 2011;36E(3):185–93.
10. Taylor I. Chapter 15. In: Mathes plastic surgery, vol 1. 2nd ed. Saunders Elsevier; 2006.
11. Taylor GI, Palmer JH. The vascular territories (angiosomes) of the body: experimental study and clinical applications. Br J Plast Surg. 1987;40:113–41.
12. Cormack GC, Lamberty BG. A classification of fasciocutaneous flaps according to their patterns of vascularity. Br J Plast Surg. 1984;37:80.
13. Dabernig J, Sorensen K, Shaw-Dunn J, Hart AM. The Thin circumflex scapular artery perforator flap. J Plast Reconstr Aesthet Surg. 2007;60:1082–96.
14. Kim DY, Kim KS. Hand resurfacing with the superthin latissimus dorsi perforator based free flap. Plast Reconstr Surg. 2003;111:366–70.
15. Strauch B, Vasconez LO, Hall-Findlay EJ, Lee BT. Grabb's encyclopedia of flaps. 3rd ed. Philadelphia: Wolters Kluwer Lippincott Williams Wilkins; 2009.
16. Pauchot J, Chambert J, Remache D, Elkhyat A, Jacquet E. Geometrical analysis of the V-Y advancement flap applied to a keystone flap. J Plast Reconstr Aesthet Surg. 2012;65:1087–95.
17. Rayan GM, Chung KC. Flap reconstruction of the upper extremity. ASSH; 2009.
18. Ponten B. The fasciocutaneous flap: its use in soft tissue defects of the lower leg. Br J Plast Surg. 1981;34:215.
19. Amarante J, Costa H, Rees J, Soares R. A new distally based fasciocutaneous flap of the leg. Br J Plast Surg. 1986;39:338–40.
20. Yang GF, Chen PJ, Gao YY, Jiang SX, He SP. Forearm free skin flap transplantation. Clin Med J. 1981;61:139–41.
21. Angrigiani C, Grilli D, Dominikow D, Zancolli EA. Posterior interosseous reverse forearm flap: experience with 80 consecutive cases. Plast Reconstr Surg. 1993;92:265–93.
22. Brunelli F, Giele H, Perrotta R. Reverse Posterior Interosseous Flap based on an Exteriorized Pedicle to cover Digital Skin Defects. J Hand Surg [Br]. 2000;25(3):296–299.

23. Ono S, Sebastin SJ, Yazaki N, Hyakusoku H, Chung K. Clinical applications of perforator-based propeller flaps in upper limb soft tissue reconstruction. J Hand Surg. 2011;36A:853–63.
24. Becker C, Gilbert A. The distally bases ulnar island artery flap in hand reconstruction. Eur J Plast Surg. 1988;11:79–82.
25. Maruyama Y, Onishi MD, Iwahira Y. The ulnar recurrent fasciocutaneous island flap: reserve medial arm flap. Plast Reconstr Surg. 1987;79(3):381–8.
26. Davalbhakta AV, Niranjan NS. Fasciocutaneous flaps based on fascial feeding vessels for defects in the periolecranon area. Br J Plast Surg. 1999;52(1):60–3.
27. Mcgregor IA, Jackson IT. The groin flap. Br J Plast Surg. 1972;25(1):3–16.
28. Song YG, Chen GZ, Song YL. The free thigh flap: a new concept based on the septocutaneous artery. Br J Plast Surg. 1984;37:149–59.
29. Kimura N, Satoh K, Hasumi T, et al. Clinical applications of the free thin anteriolateral thigh flap in 31 consecutive patients. Plast Reconstr Surg. 2001;108: 1197–208.
30. Izadi D, Paget JTEH, Haj-Basheer M, Khan UM. Fasciocutaneous flaps of the subscapular artery axis to reconstruct large extremity defects. J Plast Reconstr Aesthet Surg. 2012;65:1357–62.
31. Kay S, Pinder R, Wiper J, Hart A. Microvascular free functioning gracilis transfer with nerve transfer to establish elbow flexion. J Plast Reconstr Aesthet Surg. 2010;63(7):1142–9.

Soft Tissue Cover of the Hand

6

Steven Lo and Mark Pickford

Keywords
Soft tissue cover hand • Current evidence • Comparative studies • Small free flaps • Flaps

Part 1. Non-flap Based Approaches to Soft Tissue Cover

Non-flap based approaches to fingertip reconstruction are often equal in many aspects to more complex flap reconstructions, and in some areas may in fact be superior.

Healing by Secondary Intention

A review of the use of semi-occlusive dressings found compete wound healing in all cases, with an average 2PD of 3.6 mm, sufficient for the recovery of tactile gnosis [1]. A study comparing a number of different methods of fingertip reconstruction found in favour of using dressings alone, particularly in terms of recovery of excellent 2PD of 3.8 mm and earlier return to work than other methods. Drawbacks of using dressings alone include the poor quality padding of the fingertip, as indicated by a relatively high scar sensitivity of 54 % [2].

Skin Grafts

Lister has recommended using FTSG on the functional side of the finger, to provide more robust cover and using SSG on the non-functional side to allow the wound to contract, thereby pulling in sensate, good quality skin from the surrounding tissue [3]. However, grafts should be used sparingly for fingertip reconstruction, as prospective reviews indicate that both split and full thickness skin grafts perform poorly in comparison to simple flaps, in terms of 2PD and scar sensitivity [2].

Composite Grafts

There are conflicting reports regarding the overall success rates and variables that affect success in composite grafts. One study has suggested that composite grafts are more likely to be successful if performed within 5 h. However, the outcomes

S. Lo, MA, MSc, FRCS Plast (✉)
Canniesburn Plastic Surgery Unit,
Glasgow Royal Infirmary, Glasgow, UK
e-mail: stevenjlo@gmail.com

M. Pickford, FRCS Plast
Department of Plastic Surgery,
Queen Victoria Hospital, East Grinstead, UK
e-mail: mark.pickford@qvh.nhs.uk

I.A. Trail, A.N.M. Fleming (eds.), *Disorders of the Hand: Volume 1: Hand Injuries*,
DOI 10.1007/978-1-4471-6554-5_6, © Springer-Verlag London 2015

in this study were based on parental questionnaire rather than clinical review, and the conclusions should therefore be interpreted with caution [4]. Other variables that have been correlated with composite graft failure include smoking and composite grafts proximal to the eponychial fold [5]. Given the conflicting evidence and lack of high-level evidence available, we consider composite grafts as non-urgent and indicated primarily in children. As simple flap reconstruction can provide near normal 2PD and rapid primary healing, one must carefully consider whether a composite graft is the most appropriate method in adults.

Part 2. Flap Cover of the Digits

Background

Functional Considerations

The fingertip is defined as the portion of finger beyond the insertion of the extensor and flexor tendons, but is often inaccurately described as the portion distal to the DIPJ. In terms of restoration of useful function to the hand, flap reconstruction is most relevant for amputations at the fingertip level. In amputations proximal to the fingertip and DIPJ crease, complex flap reconstruction does not result in significant functional gain. If one considers Swanson's classification of hand impairment, 50 % of the function of a digit is lost when the finger is amputated distal to the DIPJ [6]. It is therefore justifiable to preserve length in fingertip amputations, but once one has lost finger length proximal to the DIPJ, the benefit to preserving length is largely lost, and one would be better to consider revision amputation rather than flap reconstruction.

Sensory Considerations

Tactile gnosis, or the ability of the finger to "see", is one of the unique aspects of sensory restoration in fingertips, with Moberg showing that a 2PD less than 6 mm is required for normal tactile gnosis [7–9]. Although some have questioned the reliability of 2PD in isolation as a test for tactile gnosis, it remains one of the universal outcomes recorded in most studies [10]. That being said, tactile gnosis is more critical in the functional surfaces of a fingertip (ulnar thumb, radial index, radial middle and ulnar little in particular). In comparison, non-functional surfaces may be adequately resurfaced with methods that do not restore tactile gnosis (such as flaps with a 2PD >6 mm) without significant disability. Furthermore, less emphasis should be placed on 2PD with heterotopic flaps, due to problems with the dual location phenomenon. In such circumstances it is more important that attempts are made to circumvent the dual location phenomenon, rather than concentrate on restoration of 2PD. Additionally, 2PD cannot be considered in isolation without considering scar sensitivity. It is essential to avoid placing scars on the functional surfaces of the fingertips if possible, otherwise the reconstructed digit will be simply "bypassed".

Classifications

There are a number of classifications in use for fingertip injuries. Two variables are involved – firstly, length of amputation for which we use Ishikawa's classification (Fig. 6.1), and secondly, angle of amputation [11]. The angle of amputation may be referred to as volar oblique (or volar facing), dorsal oblique, transverse, radial and ulnar oblique.

Soft Tissue Cover of the Fingertips (Index to Little)

Surgical Techniques

For the purposes of this chapter, reconstructive options have been divided into those for the fingertip proper, dorsum of finger and volar surface of finger. The techniques are subsequently divided into homodigital, heterodigital, distant pedicled and free flaps. We cover each area in turn, but will address free flap reconstruction of the digits in a later section.

Homodigital Flaps

Options

I. VY Advancement
II. Hatchet Flap
III. Bilateral Lateral VY Flaps (Kutler/Segmuller flaps)

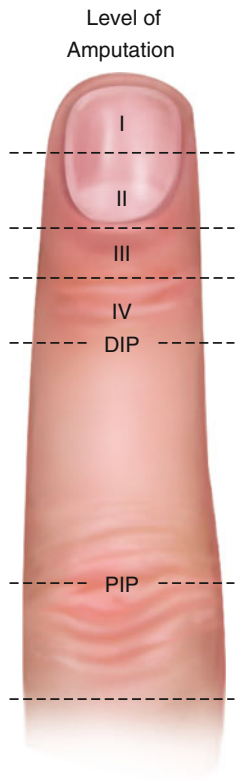


Fig. 6.1 Ishikawa levels of amputation

IV. Triangular Homodigital Advancement Flaps (Venkatswami flaps)
V. Reverse Homodigital Flaps

VY Advancement Flaps

Indications

Dorsal oblique and transverse amputations [3]. Amputation level up to midnail (Ishikawa I) for standard VY, or up to eponychial fold (Ishikawa II) for modified bipedicled VY

Technique and Refinements

Variations of VY advancement flaps have been described since 1935, but were popularized by Atasoy in 1970 [12, 13]. More recently, the VY flap has been modified as a neurovascular bipedicled flap, taken proximal to the DIPJ crease where the neurovascular bundles are more defined [14]. This allows advancement of up to 14 mm [15].

Limitations

Limited movement with standard VY

Advantages

Simple, good 2PD and aesthetics.

Outcomes

Lorea looked at 22 neurovascular VY advancement flaps, finding a static 2PD of 6 mm, 2 infections, 1 neuroma and 1 PIPJ flexion contracture [15]. Elliot reviewed 102 flaps, 46 original VY and 56 neurovascular VY. Cold intolerance was 13 % in both groups, and hypersensitivity noted in 14 % again in both groups. 2PD is not discussed in this paper [14]. In Ma et al. prospective comparative review of fingertip flaps, the VY flap fared well against other flaps in terms of scar sensitivity and 2PD of 4.3 mm [2].

Hatchet Flap

Indications

Ulnar or radial oblique, dorsal oblique, and transverse amputations. Particularly useful in resurfacing the functional surfaces of the index or little fingers.

Technique and Refinements

The hatchet flap is a rotation advancement flap initially described by Emmett for sites other than the fingertip, such as ischial and trochanteric pressure sores [16]. It has also been described for resurfacing small defects of the fingertip pulp [17]. In essence it is designed as a volar VY flap with three quarters of one side of the V left intact, on which the flap rotates and advances (Fig. 6.2). By placing the base of the flap on the functional surface of the finger, it obviates the problems of scar sensitivity. It is therefore an excellent option for preserving the functional borders of the index and little fingers.

Limitations

Limited amount of flap advancement

Advantages

Avoids placing scar on functional surface of digit

Fig. 6.2 Hatchet flap. (**a**) Transverse amputation middle finger. (**b**) Design of hatchet flap so that base is on the functional side of the middle finger (radial). (**c**) Flap rotates and advances. (**d**) Finger seen from the radial side – no scars are placed on this functional surface

Outcomes

Tuncali et al. described its use in 19 cases of fingertip injuries, with a 1 year follow up showing a 2PD of 6.3 mm, cold intolerance in 22 % and return to work in 5 weeks [17].

Bilateral Lateral VY Flaps (Kutler and Segmuller Flaps)

Indications

Subtotal finger pulp amputations

Technique and Refinements

The lateral VY flap was originally described by Geissendorfer in 1943, and later described in its bilateral form by Kutler [18, 19]. In its original form it was raised without isolating the neurovascular pedicle. This was subsequently modified by Segmuller and others by dissecting out the neurovascular pedicle, thus allowing greater flap advancement, and by raising it proximal to the distal phalanx [20, 21]. Some authors have extended as far as the proximal phalanx [22]. Although Kutler and Segmuller flaps are generally bilateral, it can also be used as a unilateral flap, more akin to a short Venkatswami flap, in order to avoid copious scarring of the volar finger. However, unlike the Venkatswami flap, the Segmuller flap does not cross the volar midline of the finger.

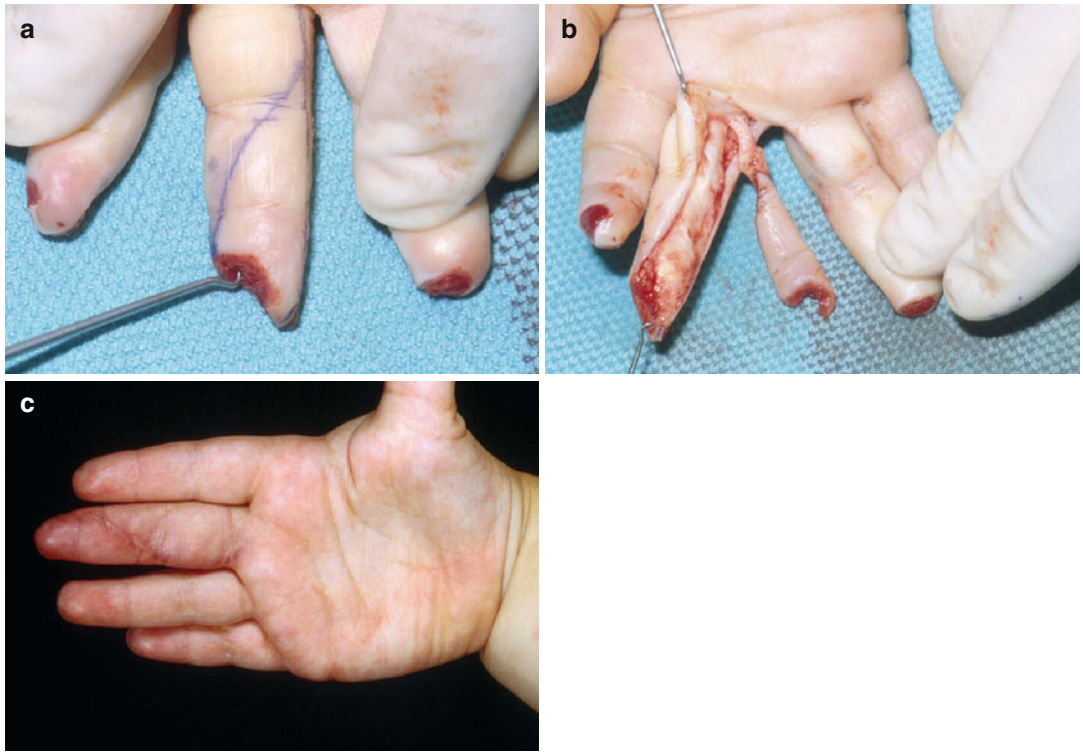


Fig. 6.3 Venkatswami flap. (**a**) Flap design, extended to base of finger. (**b**) Flap pedicle raised with adipose cuff. (**c**) Final result with no flexion contracture

Limitations

Kutler flap (unmodified) has relatively poor outcomes in comparison with other flaps. Places extensive scarring on the fingertip producing scar sensitivity and also DIPJ stiffness [2].

Advantages

Modified neurovascular pedicled flap is more versatile. Can raise one before assessing need for second flap. Both flaps retain innervation unlike the contralateral tip of the Venkatswami flap. Reliable.

Outcomes

Smith and Elliot reviewed 100 cases of the extended Segmuller flap, with 1 partial flap necrosis and 5 neuromas. Forty-five percent cases had normal static 2PD [22]

Homodigital Triangular Advancement Island Flaps (Venkatswami)

Indications

Volar or dorsal oblique laceration <2 cm.

Technique and Refinements

Described by Venkatswami in 1980, the homodigital triangular advancement flap has not found universal favour amongst hand surgeons due to perceived problems with flexion contractures (Fig. 6.3) [23]. The step-advancement modification by Evans and Martin in 1988 provides a logical solution, although formal outcomes have not been independently reported [24]. However, we have not found problems with flexion contracture when the flap is islanded completely and appropriate post-op therapy is instituted.

Neither have we found any great advantages to using the step-advancement flap, which additionally has no scope for maneuver once raised.

Limitations
Flexion contracture if night extension splint not used.

Advantages
Excellent 2 PD allows regain of tactile gnosis

Outcomes
Lanzetta reviewed 25 cases and found 1 case of necrosis, no neuroma, stable padding, hypersensitive scar in 12 %, cold intolerance in 80 % and extension lag in 7 cases (28 %) of 10–45° [21]. None of the patients with extension lag wore night extension splints as instructed, with no lag in patients who used the night splint. 2PD was 3–6 mm and in 92 % cases was equivalent to contralateral digit.

Reverse Homodigital Flap

Indication
Large volar oblique defects or total pulp loss.

Technique and Refinements
First described by a number of authors including Lai in 1989 [25]. This is a reverse flow flap raised at level of proximal phalanx, with or without the dorsal branch of the digital nerve, with a pivot point 5 mm proximal to the DIPJ where the check rein anastamosis enters the digital artery. There is conflicting evidence whether coaptation of the dorsal digital branch improves 2PD [26, 27]. However, digital nerve coaptation may have advantages in terms of cortical perception, as it may prevent the dual location phenomenon. Venous congestion can be a problem if the flap pedicle is skeletalised, but one can easily keep an adipose cuff, or alternatively preserve a volar vein with the flap.

Limitations
2PD insufficient for tactile gnosis. Tedious dissection

Advantages
Keeps donor site within injured finger

Outcomes
Yazar reviewd 64 cases and found a 2PD 5.7 mm (coaptation used in all cases), 1/64 partial flap necrosis, 3/64 flexion contracture and 2/64 neuromas [28] (Fig. 6.4).

Heterodigital Flaps

Options
I. Cross Finger Flap
II. Heterodigital Neurovascular island flaps (see later under Littler flap)

Cross Finger Flap

Indications
Subtotal pulp loss, dorsal finger defects (reverse cross finger)

Technique and Refinements
First described by Gurdin and Paganin 1950, the original description described both distally based and laterally based flaps [29]. It is more commonly performed as a laterally based fasciocutaneous flap, with care to preserve paratenon on the extensor tendon for grafting. The pedicle is traditionally divided at 14–21 days, although some authors advocate earlier division [30]. The "innervated cross finger flap" is a variation which additionally takes the dorsal branch of the digital nerve for co-aptation, with one study of 15 patients finding a static 2PD of 3.6 mm (compared with 6–8 mm for traditional cross finger) [31]. The "reverse" cross finger flap is essentially an adipofascial flap for dorsal rather than volar defects [32]. An extended reverse cross finger flap can be used for more extensive defects (Fig. 6.5).

Limitations
Donor finger morbidity, see outcomes section later.

Advantages
Can provide large size flap for subtotal pulp amputations.

Fig. 6.4 Reverse Homodigital. (**a**) Defect. (**b**) Flap designed on lateral aspect P1, dorsal branch digital nerve can be included. (**c**) Digital nerve left in-situ. (**d**) Flap pedicle raised with adipose cuff. (**e**) Flap in-situ

Outcomes

Nishikawa et al. looked at 15 patients – cold intolerance in 53 %, discomfort during manual work 50 %. 6/15 patients unable to use in precision tasks and tactile gnosis, all of these were index finger and were "bypassed" [33]. Paterson et al. examined outcomes of the donor finger (rather than the injured finger) in 17 cases, finding 8/17 stiffness, 10/12 cold intolerance, 8/17 altered pigmentation of graft. No statistical difference was found in stiffness between SSG or FTSG [34].

Fig. 6.5 Extended Reverse Cross finger flap: (**a**) Extensive dorsal defect. (**b**) Dermal flap raised. (**c**) Adipofascial flap raised in traditional manner. (**d**) Flap in-situ

Distant Flaps

In single digit reconstruction we opt for free flap transfer in preference to distant pedicled flaps. However, distant pedicled flaps remain our salvage option should a free flap fail, or in instances where patient comorbidity precludes free flap transfer. Options include the thenar flap, groin flap, cross arm and chest flaps.

Soft Tissue Cover of Defects Proximal to the Fingertip

Defects of the finger proximal to the fingertip do not require the specialized characteristics required for finger pulp reconstruction, such as tactile gnosis and cortical re-orientation. These defects are therefore more amenable to microvascular free flap transfer and heterotopic flaps, where restoration of 2PD is not as critical.

Dorsal Injuries

Nailbed Defects

Options

I. Turnover adipofascial
II. Free toenail
III. Reverse homodigital (see earlier)
IV. Reverse cross finger (see earlier)
V. Hatchet advancement flaps (see earlier)

Turnover Adipofascial Flap

Indications

Dorsal nail complex loss. Can be extended to include volar fingertip loss.

Technique and Refinements

The vascular supply comes from the distal dorsal arterial branches that originate just distal to the DIPJ, and that form a vascular network above the

extensor insertion. A base of least 5 mm is therefore preserved at the distal end of the flap, just proximal to the germinal matrix (Fig. 6.6). Some authors describe the use of this flap to resurface the distal fingertip pulp [35].

Limitations

We do not recommend this flap for finger pulp reconstruction as 2PD recovery is poor and there are better options available.

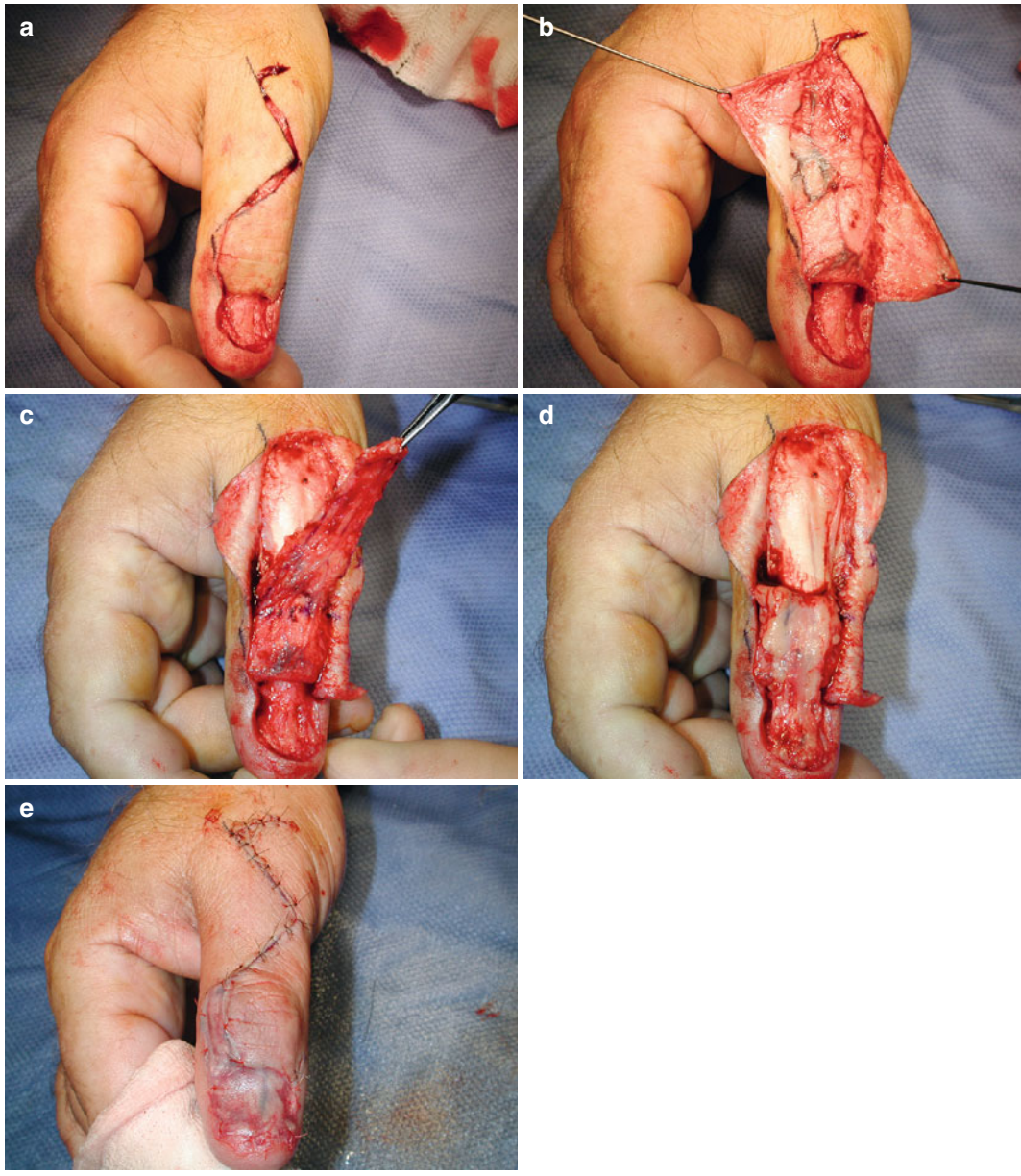


Fig. 6.6 Adipofascial turnover flap (**a**) Defect. (**b**) Flaps raised at dermis-adipose interface. (**c**) Adipofascial flap raised, preserving a base of at least 5 mm proximal to the germinal matrix. (**d**) Turned over. (**e**) Inset and SSG

Advantages
One of the most reliable flaps for nail complex cover

Outcomes
Series of 9 cases with 100 % survival. These were used to cover both the fingertip pulp as well as nail complex, resulting in a 2PD of 8 mm [35].

Microvascular Toenail Transfer

Indications
Nail complex loss for cosmesis or in particular occupations (such as string musicians). Particularly indicated for the thumb [36]

Technique and Refinements
Although toe transfers and variants thereof have existed for many decades, microvascular toenail transfers are comparatively recent [37]. The short pedicle transfer concept is emphasized by some authors, in which a 3 cm pedicle is taken and anastamosed to digital vessels, rather than to the vessels in the anatomical snuffbox [38]. As it is mainly cosmetic in nature, the short pedicle concept helps to minimize donor and recipient dissection and therefore limits cosmetic deformity. Artificial dermis can also minimize deformity of the donor site.

Limitations
Technically demanding, donor site morbidity

Advantages
Excellent cosmesis

Outcomes
Endo et al. described 19 cases of microvascular toenail transfer, with only 1 case of partial necrosis and all achieving normal nail growth. In this series average operating time was 3 h [38]. This compares favourably with non-vascularised toenail grafts, in which only 5 out of a series of 25 achieved acceptable cosmesis [39].

Defects of Dorsal Middle Phalanx

Options
I. Homodigital Adipofascial Turnover Flap (Merle flap)
II. Reverse cross finger (see previous)
III. Venous flow through flap (see later)

Homodigital Adipofascial Turnover Flap (Merle Flap)

Indications
Dorsal defects of the PIPJ and proximal three quarters of the middle phalanx. Extended modification can include up to DIPJ.

Technique and Refinements
Initially described by Voche and Merle in 1994, this flap is an adipofascial flap longitudinally based on one of the digital vessels [40]. There is no secondary defect but the flap itself requires a SSG. The original series described its use for PIPJ defects only, and for dorsal defects of up to two-thirds the width of the finger. More recently, it has been extended to allow flap reconstructions up to the DIPJ and full width of the finger (Fig. 6.7) [41].

Limitations
Flap requires skin grafting

Advantages
Preferred over the reverse cross finger flap as avoids prolonged immobilization and associated stiffness.

Outcomes
Published case series are relatively limited. In the extended Merle flap 3 cases were described with 100 % survival and 1 graft loss [41].

Defects of Dorsal Proximal Phalanx

Options
I. Dorsal Metacarpal Artery Perforator Flaps (Quaba Flap)
II. Venous flow through (see later)
III. Adipofascial turnover (see previous)

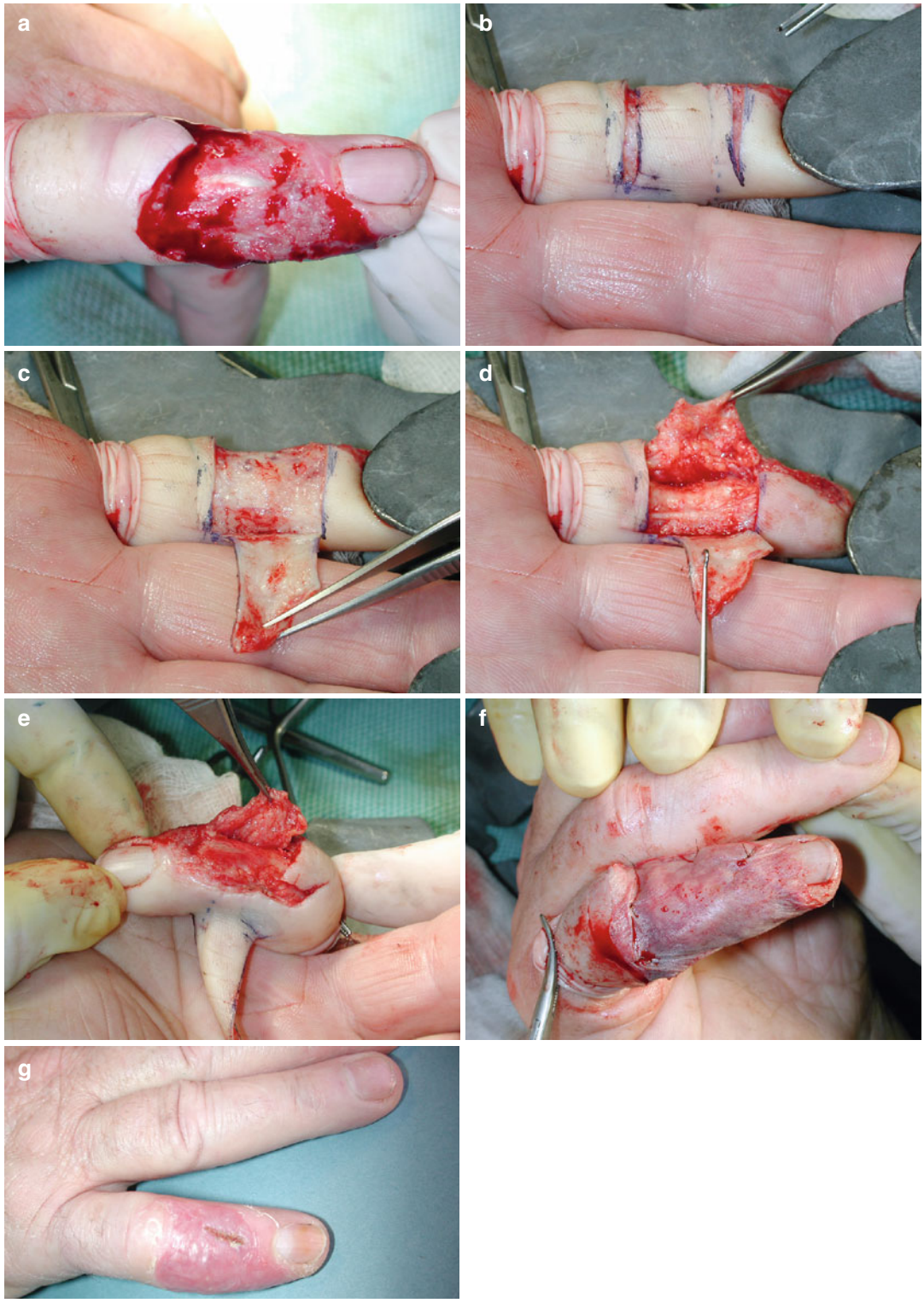


Fig. 6.7 Modified Merle flap. (**a**) Defect is dorso-ulnarly biased therefore the flap is raised on the closest (ulnar) neurovascular bundle. (**b**) Skin flap elevation. (**c**) Exposure of adipofascial plane. (**d**) Raising of adipofascial flap. (**e**) Inset. (**f**) Skin grafting of flap. (**g**) Final result

Dorsal Metacarpal Artery Perforator Flaps (Quaba Flap)

Indications

Dorsal proximal phalanx defects, webspace defects.

Technique and Refinements

First described as the "distally based hand flap" by Quaba in 1990, this perforator flap originates from the perforating branch going from the palmar to the dorsal metacarpal artery, approximately 0.5–1 cm proximal to MCPJ (Fig. 6.8) [42]. The original flap will reach up to the PIPJ. Murayama has described a similar flap, but incorporating the dorsal metacarpal artery proper into the flap [43]. There are no major advantages to this and it entails a more complex dissection. Extended variations of the dorsal metacarpal

Fig. 6.8 Quaba flap. (**a**, **b**) Defect exposing tendon of middle finger. (**c**) Doppler signal of perforating vessel. (**d**) Inset of flap and graft of secondary defect. It is normally possible to close the secondary defect primarily but in this case skin laxity was limited by previous amputations. (**e**) Example of Quaba flap with primary donor site closure

artery flap have been described which will reach up to the fingertip, but these result in an extensive scar not respectful of the dorsum of hand, and alternative options should be considered.

Limitations
Unreliable in hand infection

Advantages
Local source of well vascularised tissue, relatively straightforward

Outcomes
In a review of 69 cases, there were 7 partial losses and 3 total losses. All total losses occurred in hand infections, therefore it is not recommended in this scenario. No difference in outcomes between flaps raised on the radial or ulnar side of the hand, as long as the perforating vessel was present on Doppler [44].

Volar Finger Defects

Options
I. Cross finger (see earlier)
II. Free Venous flaps (see later)
III. Free PIA (see later)
IV. Free 1st webspace (see later)
V. Pedicled groin flap
VI. Reverse Radial Forearm

In volar finger injuries numerous flaps can be used. In small non-graftable defects a cross finger flap is a reliable option, but in larger defects we consider small free flaps to be an excellent method of resurfacing the finger, without damaging a normal donor finger. We describe the use of small free flaps later.

In single digit non-replantable degloving injuries, with the exception of the thumb, primary amputation should be strongly considered. Multiply injured fingers may require the use of a pedicle groin flap, reverse radial forearm or free flap. In volar injuries requiring revascularisation, a venous flow through flap can be used for both flap cover and arterial conduit. We have previously described the use of a syndactylised reverse radial forearm flap for multiple digit injury, using a caliber-matched perforator from the radial artery for revascularisation of a digit [45]. This technique allows simultaneous large surface area flap cover and revascularisation.

Outcomes of Fingertip Reconstruction

There is a paucity of high level evidence for outcomes in fingertip reconstruction, with the majority of methods supported by case series or expert opinion only. There are no level I or II studies on fingertip reconstruction.

Level III Evidence – Retrospective Comparative Reviews

There are a number of retrospective and prospective comparative reviews, but the majority of these are limited in their comparison of techniques.

Soderberg et al. looked at various methods of reconstruction (graft, primary closure and flaps) versus conservative management in fingertip amputations with bone exposure, in a retrospective comparative study [46]. These were divided into two groups, conservative versus surgical, with no sub-analysis of each type of closure method performed. There were 36 conservatively managed fingertips and 34 surgically treated, and follow up varied from 6 months to 4 years. The conservative treatment group fared better in terms of 2PD, pain and precision grasp. Number of lost working days was equivalent in both groups. This study suggests that conservative management of fingertip injuries results in better outcomes than surgical intervention. However, as there is no analysis of each individual surgical method, the poor results in the surgical group may have been biased by one particularly poor method, such as split skin grafting.

Ma et al. performed one of the largest prospective comparative studies, looking at 140 cases of fingertip injuries, with 7 different techniques [2]. These included SSG, FTSG, revision amputation, VY advancement, Kutler flaps, cross finger flaps and dressings alone. Final assessment was performed on all cases at 6 months and a standardized examination performed.

Outcomes were analyzed as follows (Table 6.1):

1. Healing problems – greatest in cross finger flaps with 27 % incidence of infection or graft loss, followed closely by Kutler flaps at 23 %. The other methods were comparable with healing problems in 11–17 %.
2. Cosmesis – scored from 1 (poor) to 4 (excellent) by both patient and the surgeon. In general the scores for all methods were similar, with the best scores for the VY plasty.
3. Scar tenderness. The scar sensitivity was worst for SSG (59 %) and dressings alone (54 %), correlating with the lack of thick padding over the distal phalanx.
4. Static 2 PD sufficient for tactile gnosis (<6 mm) was recovered with VY plasty, Kutler flaps, revision amputation and dressings. 2PD was worst in the skin grafted and cross finger flaps groups at 6.2–7.2 which would be insufficient for tactile gnosis.

Table 6.1 Level III evidence prospective comparative review (outcomes at 6 months) Ma et al. [2]

	Healing problems (graft loss, wound infection)/%	Appearance (1 poor – 4 excellent)	2PD/mm	Scar tenderness %	Stiffness (Loss of TAM in degrees)	Power grip/kg	Pinch grip/kg	Return to work/days
SSG	11	2.7	6.2	59	10	20.6	2.7	46
FTSG	14	2.6	6.8	26	14	23	3.2	51
VY Plasty	17	2.9	4.3	31	14	21.2	3	42
Kutler	23	2.3	3.9	31	18	22.2	3.5	52
Revision amputation	11	2.5	4.1	46	13	21.4	2.4	52
Cross finger flap	27	2.7	7.2	23	20	17.6	1.7	87
Dressings	All delayed healing	2.5	3.8	54	6	21.6	2.4	41

Table 6.2 Outcomes of soft tissue reconstruction of the digits

Technique	2PD/mm	Comments	Papers	Highest level of evidence
Dressings alone	3.8	Prolonged healing time	Ma (1982) [2]	III
Skin grafts	6.2 SSG 6.8 FTSG	Poor quality fingertip, scar sensitivity	Ma (1982) [2]	III
VY Flaps	4.3 6 mm	Short advancement	Ma (1982) [2] Lorea (2006) [15]	III IV
Hatchet flap	6.3 mm	Avoids scar on functiona surface	Tuncali (2006) [17]	IV
Kutler/Segmuller	3.9 mm		Ma (1982) [2]	III
Venkatswami/Homodigital advancement island flaps	3–6 mm	Cold intolerance 80 %, extension lag if night splint not used	Lanzetta (1995) [21]	IV
Reverse Homodigital	5.7	Tedious dissection	Yazar (2010) [28]	IV
Cross finger flaps	7.2 7.6	Restoration of tactile gnosis, donor site morbidity	Ma (1982) [2] Nishikawa (1992) [33]	IV
Innervated cross finger	3.6 mm		Lassner (2002) [31]	IV
Free toe pulp for digits other than thumb	13.1 mm	Microsurgical expertise required	Del Pinal (2004) [65] Lin (2007)	IV
Toenail	N/A		Endo (2002) [38]	IV
Turnover adipofascial	8 mm	Not recommended for finger pulp	Laoulakos (2003) [35]	IV

5. Stiffness – Cross finger flaps resulted in the greatest loss of total active range of movement (TAM) of 20°. Kutler flaps resulted in loss of 18° mainly at the DIPJ. Dressings resulted in the least finger stiffness.
6. Power – overall power and pinch grip was comparable in all groups except cross finger flaps. Loss of power was attributed to the prolonged period (2–3 weeks) of immobilization.
7. Return to work and sick leave – although management with dressings took longest for complete wound healing (28 days) they paradoxically returned to work the earliest at 41 days. Cross finger flaps took twice as long to return to work than other methods at 87 days.

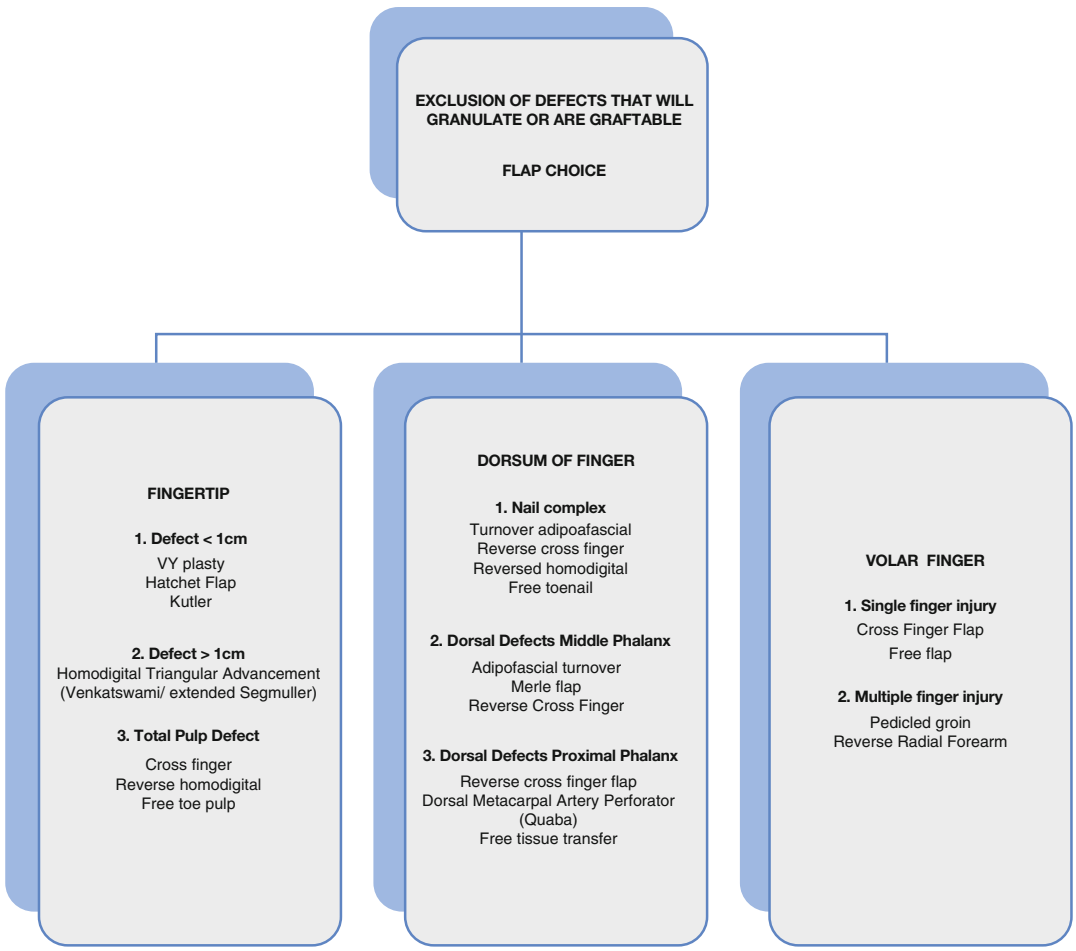
In this series the cross finger flaps resulted in the worst overall outcomes, whilst VY plasty appeared to give the best overall outcomes. Although dressings only also gave good results, the quality of the resultant tip was poor as indicated by the high incidence of scar sensitivity. Skin grafting produced tender fingertips with poor sensation.

Level IV Evidence – Non-comparative Case Series

There are numerous non-comparative retrospective reviews. These are generally unhelpful in isolation, as they do not allow valid comparison of techniques (Table 6.2).

Algorithm for Management

(Flow Chart 6.1)



Flow Chart 6.1 Algorithm for soft tissue defects of the digits

Part 3. The Thumb

Classification

Lister has described four types of thumb deficit requiring reconstruction [47]:

1. Length acceptable, cover is poor (i.e. distal defects)
2. Subtotal, length required
3. Total thumb amputation with intact CMCJ, thenar muscles
4. Total thumb amputation lacking CMCJ and thenar muscles

In this chapter we discuss only options for type 1 deficits. Maintenance of length in such injuries is crucial, as 40 % of hand function is related the thumb, and 50 % of thumb function is lost at amputation at IPJ level [6].

Local Flaps

Homodigital Flaps

Options

I. Palmar Advancement Flaps (Moberg Flap)
II. Switch flap
III. VY advancement flaps (See earlier)
IV. Adipofascial turnover flaps (see earlier)

Palmar Advancement Flaps (Moberg flap)

Indications
Volar defects <2 cm

Technique and Refinements
The palmar advancement flap was originally described by Moberg in 1964, without detachment of the flap base [47]. It has been subject to numerous modifications since, the most significant being the complete islanding of the flap to allow greater advancement and reduce risk of flexion contracture. Other refinements relate predominantly to methods of resurfacing the secondary defect [48, 49]. We prefer to use the VY modification popularized by Elliot (Fig. 6.9).

Limitations
Concerns regarding flexion contracture are not borne out by the literature, however judicious post-operative therapy is required

Advantages
No need for cortical re-orientation, excellent sensory recovery, reliable

Outcomes
Foucher reviewed 12 cases noting a 2PD of 5 mm and grip strength/ROM equivalent to contraleral side and no flexion contractures [50]. Baumiester reviewed 25 cases finding a surgical revision rate of 22 %, predominantly amongst flaps performed by trainees and in larger defects >2 cm. No statistically significant loss of range of movement at the IPJ nor flexion contractures were noted. Eighty-three percent defects were closed without the need for additional bone shortening. Normal sensation noted in 74 % and no loss of grip strength unless thumb length was lost [51].

Switch Flap

Indications
Longitudinal defects of the ulnar thumb pulp

Technique and Refinements
Originally described as an "exchange" flap for use in resurfacing the radial hemi-pulp of the index finger [52], it has been used in the thumb to resurface the functional ulnar surface by Elliot in 2003. The intact radial hemi-pulp is transferred to the ulnar side, with skin grafting to the secondary defect (Fig. 6.10).

Limitations
Violates the radial thumb pulp, therefore caution in certain professions (typists and musicians)

Advantages
Simple, innervated glabrous skin

Outcomes
Elliot reviewed 3 cases. Cortical re-orientation occurred in only 1, but despite this the

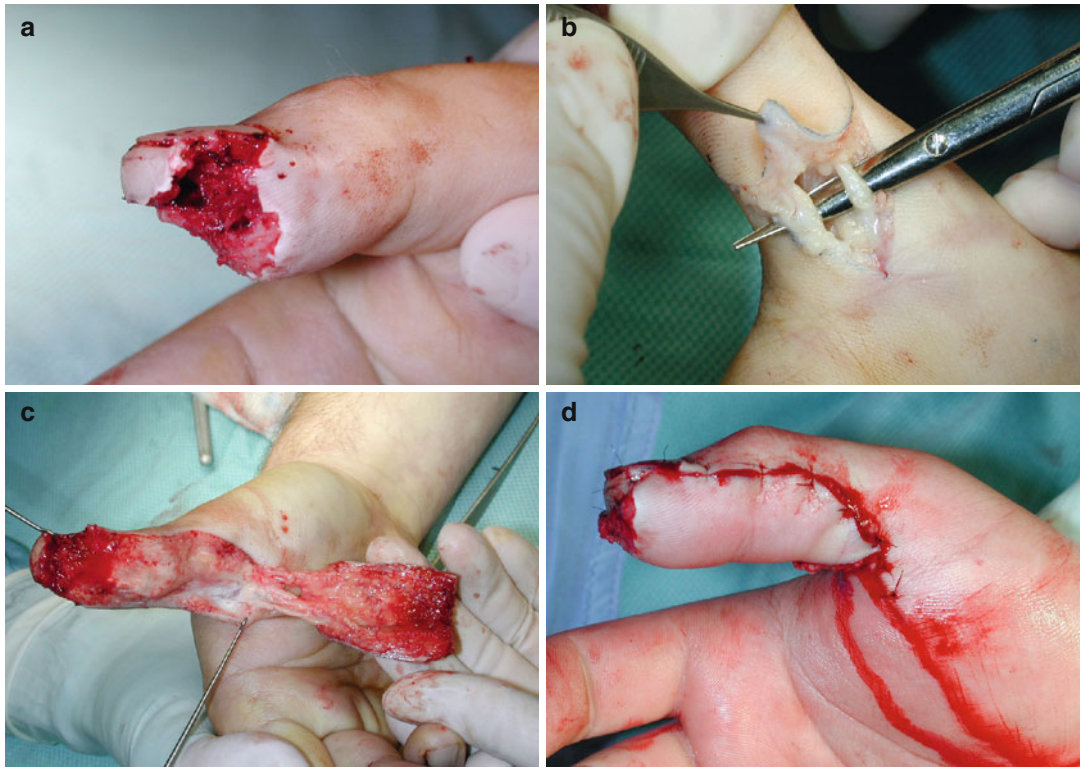


Fig. 6.9 VY modification of Moberg advancement flap. (**a**) Thumb tip defect. (**b**) Neurovascular bundles raised and preserved. (**c**) Flap fully dissected from thumb. (**d**) Inset with VY advancement at base

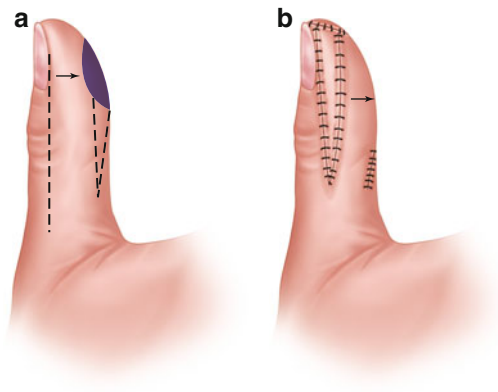


Fig. 6.10 Switch flap. (**a**) Triangulation of ulnar thumb defect. (**b**) Switching the radial pulp to the ulnar side

reconstructed switch flap was used as the "pinch" contact point rather than the more proximal thumb [53].

Heterodigital Flaps

Options

I. Heterodigital Neurovascular Island Flaps (Littler/Buchler Flaps)
II. First Dorsal Metacarpal Artery Flaps (Foucher Flap)
III. Cross finger flaps (See earlier)

Heterodigital Neurovascular Island Flaps (Littler/Buchler Flaps)

Indications

Thumb pulp sensory restoration, as part of osteoplastic thumb reconstruction. Can be used for other digits (not recommended)

Technique and Refinements

One of the landmarks in hand surgery was the development of the neurovascular island flap by

Littler in 1946 [54]. Heterodigital island flaps have generally fallen out of favour, partly due to the deleterious effect on the donor finger but also because long term cortical re-orientation does not occur in the majority of patients, resulting in the dual location phenomenon. Although Foucher described the "dibranchement-rembranchement" technique for local co-aptation, this results in loss of 2PD and therefore sensory discrimination [55]. Furthermore, as a general principle, the use of a normal uninjured finger as a donor site is best avoided.

Buchler described a variation on the Littler flap by dissecting the dorsal branch of the digital nerve from the digital nerve proper, and utilizing a skin island from the dorsum of the middle phalanx, hence terming it the "dorsal middle phalangeal flap". This preserves the digital nerve proper to the fingertip pulp, and also minimizes donor site morbidity [56]. It has also been employed as a retrograde and antegrade pedicled flap, and rarely as a free flap. The versatility of this flap allows it to reach the tip of length preserved thumbs, the fingertips of other digits, and the wrist crease.

Limitations

Cortical re-orientation poor, extensive scarring in hand, violates a normal finger

Advantages

Source of innervated glabrous skin when no local option is available, such as in osteoplastic thumb reconstruction.

Outcomes

Despite near normal tactile gnosis (19 out of 20 cases) cortical re-orientation occurs in only 25 % cases at 10 years [57]. Oka described using local co-aptation ("dibranchement-rembranchement"), which increased the cortical re-orientation rates from 61 to 100 %. In their study they found no significant deterioration in 2PD when local co-aptation was performed [58]. A series of 43 "dorsal middle phalangeal flaps" found a 100 % survival and a static 2PD of 10 mm. Although the donor finger sensibility was preserved completely in 81 % cases, hyperaesthesia was also noted in 12 % [59].

First Dorsal Metacarpal Artery Flap (FDMCA/Foucher Flap)

Indications

Pulp or dorsal defects up to the thumb tip in length preserved thumbs. Large defects >2 cm. The flap will reach the tip of a length preserved thumb

Technique and Refinements

Described initially by Hilgenfeldt in 1950 and Holevitch in 1963, it was popularized by Foucher in 1979 as the "kite" flap, with a modification from a peninsular to an islanded flap [60–62]. The vascular supply of this flap comes from the ulnar branch of the first dorsal metacarpal artery. The radial branch supplies the thumb and an intermediate branch supplies the 1st webspace. In 90 % cases the flap pedicle lies parallel to 2nd MC shaft but in 10 % it lies in the midline of the web. As the relative depth of the pedicle varies, with 57 % suprafascial and 43 % subfascial, the epimyseum of the first dorsal interosseus and the periosteum of the radial half of the second metacarpal shaft should therefore be taken with the pedicle (Fig. 6.11) [63]. Particular care should be taken in the dissection around the extensor hood.

As the 2PD of the dorsum of the index is 12–15 mm, it is at the upper limit of that which is useful for sensory discrimination, and significantly above that required for normal tactile gnosis [62]. In series using superficial branches of the radial nerve for innervation, 2PD averages 10.57 mm [63]. Furthermore, in the original series by Foucher, due to problems with cortical re-orientation, this flap was not recommended for the sensory surfaces of the thumb. Complete re-orientation may be seen in as few as 14 % [63].

Minor refinements include the inclusion of a small dart of skin attached to the skin paddle in order to spatulate the wound closure, and avoiding the raising of flaps distal to the PIPJ of the index finger due to issues of reliability and stiffness of the donor finger.

Limitations

Need for cortical re-orientation limits the usefulness of this flap for sensory restoration of the thumb pulp. 2PD insufficient for tactile gnosis.

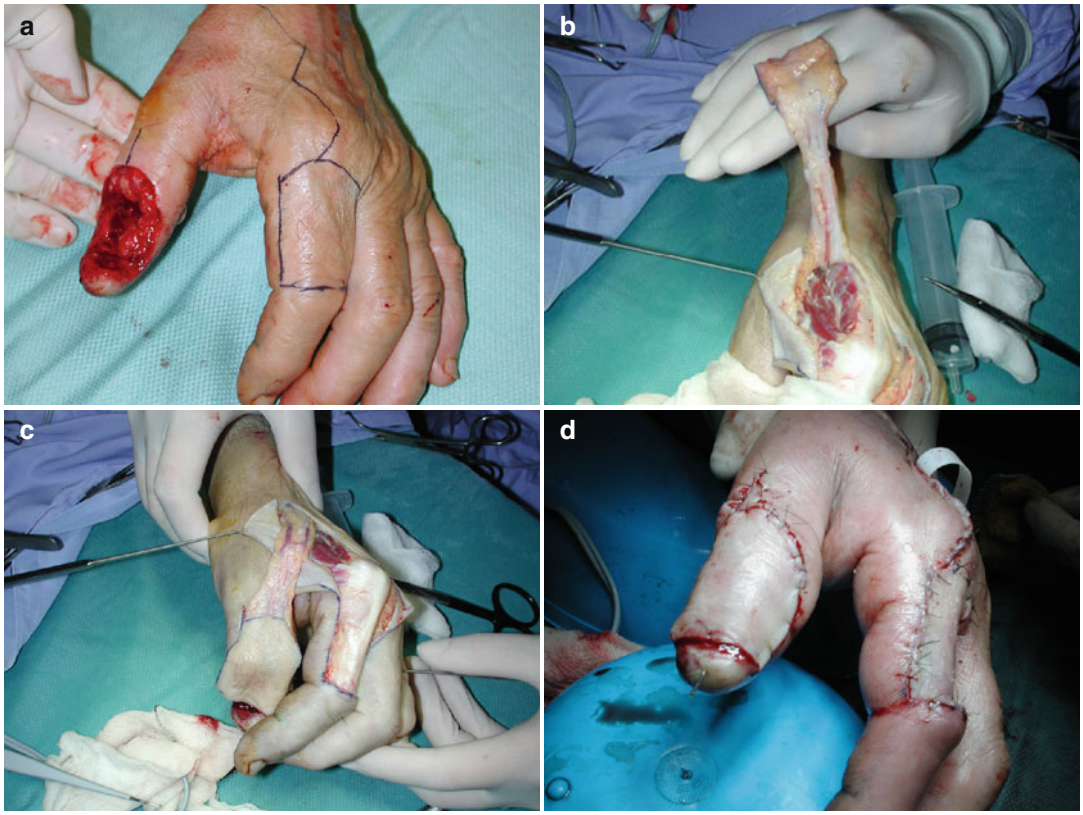


Fig. 6.11 Foucher flap. (**a**) Defect and flap planned over P1 only. (**b**) Flap demonstrates the vascular pedicle. (**c**) Flap reaches easily to tip of thumb. Note the venous plexus preserved dorsally. (**d**) Skin grafted donor site

Advantages

Useful for dorsal thumb defects or defects of the thumb too large for Moberg flap (>2 cm)

Outcomes

In a review of 25 cases of an innervated FDMCA flap, the outcomes at 3 years were static 2PD 10.9 mm, and cortical re-orientation in only 50 %, with no difference between old and young patients. The donor finger was marginally stiffer than the contralateral index with a loss of total active range of motion of 14° [64].

Distant Pedicled Flaps

Distant pedicled flaps are useful for larger defects of the thumb and degloving injuries. There are a multitude of options available including the chest, abdomen, groin and contralateral arm.

Free Flaps

Toe Pulp and Variations (Great Toe Pulp, Tibial Neurocutaneous Flap, Toe Wraparound)

Indications

Total Thumb Pulp Loss with the aim to restore sensation and provide shear resistant pinch grip. Some authors have extended the indications for use in digits other than the thumb [65]

Technique and Refinements

First described by Buncke in 1979 and Foucher in 1980, the great toe pulp microvascular free flap has gained popularity in Microsurgical centres as a method of resurfacing the thumb pulp [66, 67]. The technique of dissection is essentially the same

as that for toe transfer and is not elaborated further here (Fig. 6.12). There are however a number of refinements that can be utilized. Firstly, the short pedicle concept, as described for toenail transfer, allows a more aesthetically respectful donor and recipient site dissection [38]. Secondly, if donor vessels are taken at the Y-junction in the first webspace, this obviates the need for dissection of a plantar or dorsal system. This also entails a more rapid and less damaging dissection [65]. Thirdly, extensive stripping of the arterial adventitia can help prevent vasospasm, which is not uncommon in transfers from the foot [68]. Del Pinal has also described a "Tibial neurocutaneous flap" which takes the medial aspect of the second toe with the digital nerve, rather than from the lateral aspect of the great toe which is usually taken with the digital nerve and deep peroneal nerve. This minimizes first webspace donor site problems. This is performed under axillary and epidural blocks, with an average operating time of 4 h [65].

The toe wraparound flap described by Morrison in 1980, is a variation of the classic great toe flap, utilizing an intercalary bone graft and wrapping the soft tissue transfer around this construct. It is mentioned only briefly here as its

Fig. 6.12 Toe pulp transfer. (**a**) Failed composite graft – total pulp. (**b**) Great toe pulp lateral aspect taken. (**c**, **d**) Dorsal system dissection. (**e**) Inset to digital artery and dorsal veins, rather than snuffbox dissection. (**f**) Donor site

Fig. 6.13 Toe wraparound. (**a**, **b**) Thumb defect after crush injury in machinery, and failed revascularisation. (**c**) Toe wraparound flap. (**d**) Flap in-situ. (**e**) Long term aesthetic result

use is predominantly for loss of thumb length rather than soft tissue cover per se, although in rare instances it may be used in degloving injuries (Fig. 6.13) [69].

Limitations

Classically these have been indicated only for thumb pulp reconstruction, but some authors advocate its use in digits other than the thumb [65, 70]. Donor site problems.

Advantages

Aesthetics, glabrous skin

Outcomes

Lin et al. reviewed 15 toe pulp transfers, with a static 2PD of 13.1 mm, but 3 flaps were able to discriminate 1 point only. Semmes-Weinstein monofilament testing revealed diminished light touch in 40 % and diminished protective sensation in 53 % and loss of protective sensation in 7 % [71].

Outcomes in Distal Thumb Reconstruction

There are no Level I (meta-analysis of RCT) or Level II (Randomised Controlled Trial) studies to support the ideal reconstructive method for soft tissue loss of the thumb.

Level III Evidence – Retrospective Comparative Reviews

Woo et al. published a retrospective comparative review of 5 microvascular techniques used in partial thumb defects [72]. 43 thumb reconstructions were performed with a 100 % survival rate. For thumb pulp defects, 8 cases of lateral great toe pulp transfer were reviewed at 10 month follow-up, with a 2PD of 6 mm, key pinch 95 % of the contralateral side and IPJ ROM of 65°. For dorsal thumb defects, 4 cases of dorsalis pedis flap were assessed, with a 2PD of 15 mm, key pinch 75 % of contralateral side, and IPJ ROM 32°. There were 18 composite partial defects of the thumb, for which partial great toe transfers were done, with a 2PD 9 mm, key pinch 80 % of contralateral side, and IPJ ROM 48°. Additionally there were 10 first webspace and 3 nail complex transfers, but outcomes such as 2PD were not relevant to these cases. Overall, this study indicated that microvascular transfers to restore thumb defects are reliable, with excellent aesthetics and relatively good 2PD in great toe pulp transfers. A 2PD of 6 mm is in fact greater than that which is possible in the toe in its native position. The exact mechanism of this is unknown but may be related to the constant post-operative stimuli. However, not all studies have indicated such excellent sensory results following toe pulp transfer [65, 71].

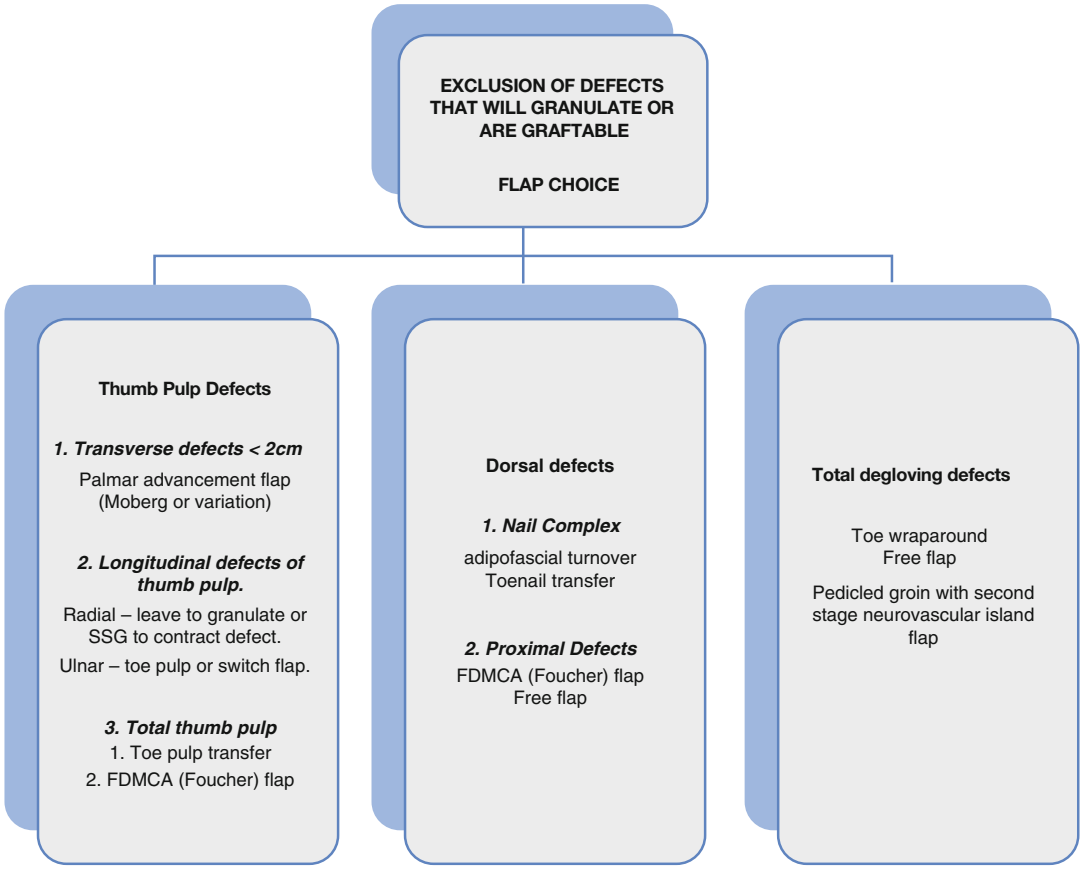
Level IV Evidence – Non-comparative Case Series

The literature strongly favours the Moberg flap as the pedicled flap of choice in thumb tip defects, due to its excellent 2PD and lack of need for cortical re-orientation. However, no Level III outcome data exists to support this. Retrospective cohort reviews by both Foucher and Baumiester confirm excellent restoration of sensory restoration with a 2PD of 5 mm and grip strength/ROM equivalent to the contraleral side. Flexion contractures are not seen when appropriate therapy is instituted. The main advantage over heterotopic flaps is that cortical re-orientation is un-necessary, rendering the thumb functionally useful immediately, thereby facilitating post-operative rehabilitation. Both reviews conclude that the Moberg flap is the ideal flap for defects <2 cm of the thumb pulp [50, 51].

Although technically elegant, the First Dorsal Metacarpal Artery Flap has two major problems for thumb pulp resurfacing. Firstly, the sensory discrimination is poor, and secondly cortical re-orientation occurs in only 50 % [73]. A similar problem exists for heterodigital neurovascular island flaps such as the Littler flap, with only 25 % achieving cortical re-orientation at 10 years [57]. Additionally, one violates a healthy donor finger, a problem that can be partially circumvented by the use of the Dorsal Middle Phalangeal flap. Although problems of cortical re-orientation in heterotopic flaps may be ameliorated by the technique of "dibranchement-rembranchement", there are reservations concerning loss of sensory discrimination with this technique (Table 6.3, Flow Chart 6.2) [55, 58]. Furthermore, this inevitably delays full post-operative rehabilitation and functional use of the hand.

Table 6.3 Outcomes of soft tissue reconstruction of thumb

	2PD	Cortical reorientation	Studies	Level of evidence
Moberg	5 mm	Unnecessary	Foucher (1999)	IV
FDMCA (Foucher)	10.8	50 %	Trankle (2003)	IV
	15		Shi (1994) [85]	IV
Neurovascular island flaps (Littler)	7	25 % at 10 years	Henderson (1980)	IV
	9.4	61–100 %	Oka (2003)	IV
Great Toe Pulp	6	N/A	Woo (1999)	III
	13.1		Lin (2007)	IV
Toe Wraparound	12 mm	N/A	Wei (1994) [86]	III



Flow Chart 6.2 Algorithm for soft-tissue only defects of thumb

Part 4: Small Free Flaps in Digital and Webspace Reconstruction

No list of flaps can be exhaustive, and we include only some of the commoner flaps in use. However, an emphasis has recently been placed on emerging techniques in small free flap reconstruction of the digits. These are particularly useful in rare cases when local reconstructive options are not available, allowing reconstruction of digits that previously may not have been salveagable. Moreover, webspace reconstruction is particularly suited to free flap reconstruction, as the resultant quality of webspace is far superior to that which can be achieved with skin grafts alone.

Free flap reconstruction of the digits has more recently been extended by some authors to include aesthetic considerations alone [65, 70]. Cheng et al. reviewed a series of 80 cases of partial toe, toenail and toe pulp transfers for aesthetic reconstruction of digits (of which 29 were of non-thumb digits), with a 97.5 % survival rate, and 2PD varying from 4 to 10 mm [70]. In a review by Del Pinal, the conventional notions of toe pulp transfer are also challenged and microvascular transfer is performed within the acute trauma period, and is recommended particularly in manual workers [65]. Toe pulp, traditionally used in thumb reconstruction alone, is also recommended for the functional surfaces of digits such as the index and little fingers.

In a review of 18 small free flaps for the digits, a flap necrosis of 11 % and partial flap necrosis rate of 6 % was noted [74]. This series included 10 venous flow through, 1 lateral arm, 1 medial plantar, 2 free Posterior interosseus flaps, 1 toe pulp and 3 first webspace flaps. The majority of the patients regained excellent function with a

quick DASH score of 5.7. All flaps regained protective sensation only, with 2PD of 13–15 mm. Pain and cosmetic deformity were minimal, but donor site morbidity was high in first webspace flaps, with hypertrophic scarring around the donor site in all cases. Endo has described the use of artificial dermis which may obviate some of these problems [38]. This review concludes that the venous flow through flap is the preferred free flap choice for the digits, due to its ease of dissection, versatility of pedicle design and limited donor site morbidity. Although first webspace flaps result in excellent digit reconstruction characteristics, their use is generally avoided due to unacceptably high donor site complications.

Perceived drawbacks of free flap reconstruction of the digits include the lack of sufficient sensation in most flaps for tactile gnosis (except toe pulp), prolonged operating time and the need for relative technical expertise. However, in Specialist Centres the latter two issues are not of concern.

Options

I. Venous Flow Through Flap
II. Posterior Interosseus Flap
III. Medial Plantar
IV. First Webspace
V. Toe Pulp (see earlier)
VI. Toe Nail (see earlier)

Venous Flow Through Flap

Indications

Defects proximal to the fingertip, large finger defects, webspace reconstruction, degloving defects requiring revascularisation and skin cover

Technique and Refinements

Thatte and Thatte have described three variants: Type I – venous unipedicled, Type II – venous-venous and Type III – arterio-venous [75]. Chen has additionally described artery-artery flow through flaps [76]. As the pedicle requires only a single vein or plexus of veins, the donor sites are numerous. For reconstruction of the digits, the volar wrist skin has a number of veins in an "H" configuration, which offer an ideal thin and pliable flap. This site also offers the opportunity to take Palmaris longus or small cutaneous nerve branches such as the palmar cutaneous branch of the median nerve or lateral antebrachial for innervation. The flap is designed after marking the veins and direction of flow, prior to tourniquet inflation (Fig. 6.14). Post-operative flap congestion and oedema may mimic venous compromise. However, normal Doppler signals from the efferent veins and bright red pin-prick bleeding are indicative of healthy perfusion.

Limitations

Perceived high failure rates not supported by large series

Advantages

Thin, pliable flap ideal for small hand defects, does not sacrifice a major artery. Can be used for composite defects requiring tendon or nerve. Good caliber match for small digital arteries and veins.

Outcomes

Large series of type III arteriovenous venous flaps have failure rates of 2–3.6 % [76, 77]. We recommend using arteriovenous or artery-artery flow through flaps in preference to Type II veno-venous flaps, as these have a more questionable reliability [76].

Free Posterior Interosseus Flap

Indications

Defects requiring very thin tissue resurfacing, particularly dorsal hand defects, first webspace and digits.

Technique and Refinements

First described as a pedicled flap by Zancolli and Angrigiani in 1988, this flap is based on the posterior interosseus artery [78]. This in turn is derived from the common interosseus artery, a branch of the ulnar artery, which divides into a posterior and anterior branch. The posterior interosseus artery lies deep to supinator, and its surface landmarks

Fig. 6.14 venous flow through flap. (**a**) Dorsal composite index finger defect. (**b**) Relatively innocuous donor site, note the direction of flow is marked prior to elevation. (**c**, **d**) Type III Arteriovenous flow through flap to the digital vessels

are the junction of the proximal and middle thirds of a line between the lateral epicondyle and the distal radio-ulnar joint. The vessel lies in the septum between extensor compartments 5 and 6. Identification of the correct extensor compartments is easiest to perform at the wrist initially, followed by an approach to the vessel from radial to ulnar (Fig. 6.15). Although the artery carries its own venae comitantes, some authors recommend that a cutaneous vein should be harvested with the flap if it is to be used as a free flap [79].

Limitations

Relatively small caliber pedicle, but good match for digital vessels

Advantages

Thin, can be raised as an fascial flap only, long pedicle

Outcomes

Chen reported 36 cases of free PIA with a success rate of 97 % [79].

Free Medial Plantar Flap

Indications

Small pulp defects or other areas requiring glabrous skin such as the hypothenar aspect of the palm.

Technique and Refinements

The medial plantar flap was described as a free flap for resurfacing the palm by Hidalgo in 1986, and as a method for finger pulp reconstruction by Inoue in 1988 [80, 81]. A number of case series report the successful use of small free medial plantar flaps, with or without reinnervation for finger pulp reconstruction [82, 83].

Fig. 6.15 Free fascial PIA. (**a**) Free PIA to cover traumatic longitudinal finger after DIPJ arthrodesis. (**b**) Split skin graft cover. (**c**, **d**) Final post-operative views

The medial plantar flap has a relatively consistent pedicle between the abductor hallucis brevis and flexor digitorum brevis, although a cutaneous branch of the saphenous vein should additionally be taken for anastamosis when used as a free flap. Reinnervation can be performed to either a cutaneous branch of medial plantar nerve or the terminal cutaneous branch of the saphenous nerve. The medial plantar nerve originates 1–3 cm distal to the medial malleolus, giving off three cutaneous branches to the medial plantar skin. One or more of these branches can be taken with the flap, with intraneural dissection allowing greater length.

Limitations

Relatively easy to raise when taken as a short pedicle transfer. Donor site problems

Advantages

Allows a greater surface area of glabrous skin for resurfacing of larger defects than the toe pulp flap.

Outcomes

Huang described 10 cases for finger reconstruction with a 2PD of 8.8 mm without reinnervation, 20 % donor site problems, 90 % total flap survival, and 10 % partial flap loss [83]. Lee reviewed 6 cases of pulp reconstruction with small medial plantar flaps with reinnervation, with follow up at 2 years indicating a 2PD of 5.2 mm [82].

First Webspace Free Flap/Dorsalis Pedis Free Flap

Indications

Webspace reconstruction, larger finger defects

Technique and Refinements

Described as early as 1977 [84], the anatomy and dissection are essentially the same as that for raising the toe pulp flap. An "extended" flap can be designed, incorporating the first

Fig. 6.16 First Webspace flap. (**a**) Complete volar loss of middle finger. (**b**) Extended 1st webspace flap (or Type III flap according to the classification of Woo 1999). (**c**) Dorsal system. (**d**) Flap in-situ

webspace, the lateral aspect of great toe and the medial aspect of second toe. A flap of up to 7.5 cm width and 14 cm length can thus be designed [68]. The vascular and nerve supply in this area allow great versatility in flap design, with the deep peroneal nerve or digital nerves to both toes, and the dorsal or plantar metatarsal artery and digital branches to both toes, available for inclusion. The first webspace flap has been classified into 4 types based on this versatility [68]:

Type 1: the webspace proper

Type 2: a two island skin flap taken separately from the great and second toes, based on separate digital vessels and nerves

Type 3: fill-up web flap – basically a long dorsalis pedis skin flap (Fig. 6.16)

Type 4: adjuvant web flap – when the first web or adjacent skin is taken in conjunction with a vascularised joint transfer

Limitations

Donor site problems

Advantages

Like for like construction of webspace defects of the hand

Outcomes

In a review of 31 cases a 100 % survival rate and 2PD 8.5 mm was noted [68].

References

1. Mennen U, Wiese A. Fingertip injuries management with semi-occlusive dressing. J Hand Surg Br. 1993; 18(4):416–22.
2. Ma GF, Cheng JC, Chan KT, Chan KM, Leung PC. Finger tip injuries–a prospective study on seven methods of treatment on 200 cases. Ann Acad Med Singapore. 1982;11(2):207–13.

3. Lister G. The hand. Diagnosis and indications. Edinburgh: Churchill Livingstone; 1984.
4. Moiemen NS, Elliot D. Composite graft replacement of digital tips. 2. A study in children. J Hand Surg Br. 1997;22(3):346–52.
5. Heistein JB, Cook PA. Factors affecting composite graft survival in digital tip amputations. Ann Plast Surg. 2003;50(3):299–303.
6. Swanson AB, Göran-Hagert C, de Groot Swanson G. Evaluation of impairment in the upper extremity. J Hand Surg Am. 1987;12(5 Pt 2):896–926.
7. Moberg E. Objective methods for determining the functional value of sensibility in the hand. J Bone Joint Surg. 1958;40B:454–76.
8. Moberg E. Aspects of sensation in reconstructive surgery of the upper extremity. J Bone Joint Sur. 1964;46A:817–25.
9. Dellon AL. Sensibility, re-education of sensation in the hand. Baltimore: Williams & Wilkins; 1981.
10. Lundborg G, Rosen B. The two-point discrimination test: time for a re-appraisal? J Hand Surg (Br). 2004;29:418.
11. Ishikawa K, Ogawa Y, Soeda H, Yoshida Y. A new classification of the amputated level for the distal part of the finger. J Jpn Soc Reconstr Microsurg. 1990;3:54.
12. Tranquilli-Leali E. Ricostruzione dell'apice delle falangi ungue-ali ruediante autoplastica volare peduncolata per scorrimento. Infort Traum Lavaro. 1935;1:186–93.
13. Atasoy E, Ioakimidis E, Kasdan ML, Kutz JE, Kleinert HE. Reconstruction of the amputated fingertip with a triangular volar flap. J Bone Joint Surg. 1970;52A(5):921–6.
14. Elliot D, Moiemen NS, Jigjinni VS. The neurovascular Tranquilli-Leali flap. J Hand Surg Br. 1995;20(6):815–23.
15. Lorea P, Chahidi N, Marchesi S, Ezzedine R, Marin Braun F, Dury M. Reconstruction of fingertip defects with the neurovascular tranquilli-leali flap. J Hand Surg Br. 2006;31(3):280–4.
16. Emmett AJ. The closure of defects by using adjacent tri- angular flaps with subcutaneous pedicles. Plast Reconstr Surg. 1977;59:45.
17. Tuncali D, Barutcu AY, Gokrem S, Terzioglu A, Aslan G. The hatchet flap for reconstruction of fingertip amputations. Plast Reconstr Surg. 2006;117(6): 1933–9.
18. Geissendorfer H. Beitrag zur Fingerkuppenplastik. Zentralbl Chir. 1943;70:1107.
19. Kutler W. A new method for finger tip amputation. JAMA. 1947;133:29.
20. Segmuller G. Modification of the Kutler flap: neurovascular pedicle. Handchirurgie. 1976;8(2):75–6.
21. Lanzetta M, Mastropasqua B, Chollet A, Brisebois N. Versatility of the homodigital triangular neurovascular island flap. J Hand Surg. 1995;20B:824.
22. Smith KL, Elliot D. The extended Segmüller flap. Plast Reconstr Surg. 2000;105(4):1334–46.
23. Venkatswami R, Subramanian N. Oblique triangular flap: a new method of repair for oblique amputations of the fingertip and thumb. Plast Reconstr Surg. 1980;66(2):296–300.
24. Evans DM, Martin DL. Step-advancement island flap for fingertip reconstruction. Br J Plast Surg. 1988; 41(2):105–11.
25. Lai CS, Lin SD, Yang CC. The reverse digital artery flap for fingertip reconstruction. Ann Plast Surg. 1989;22(6):495–500.
26. Kaleli T, Ersözlü S, Öztürk Ç. Double reverse-flow island flaps for two adjacent finger tissue defect [Article in Turkish]. Arch Orthop Trauma Surg. 2004;124:157–60.
27. Moschella F, Cordova A. Reverse homodigital dorsal radi- al flap of the thumb. Plast Reconstr Surg. 2006;117:920–6.
28. Yazar M, Aydın A, Kurt Yazar S, Başaran K, Güven E. Sensory recovery of the reverse homodigital island flap in fingertip reconstruction: a review of 66 cases. Acta Orthop Traumatol Turc. 2010;44(5):345–51.
29. Gurdin M, Pangman WJ. The repair of surface defects of fingers by trans-digital flaps. Plast Reconstr Surg (1946). 1950;5(4):368–71.
30. Heng D, Zhang C, Yao Y, Liu L, Chen YI. Experimental study on early division of cross-finger pedicle flap and its clinical application. Chin J Traumatol. 2000;3(3):159–62.
31. Lassner F, Becker M, Berger A, Pallua N. Sensory reconstruction of the fingertip using the bilaterally innervated sensory cross-finger flap. Plast Reconstr Surg. 2002;109(3):988–93.
32. Atasoy E. Reversed cross-finger subcutaneous flap. J Hand Surg Am. 1982;7(5):481–3.
33. Nishikawa H, Smith PJ. The recovery of sensation and function after cross-finger flaps for fingertip injury. J Hand Surg Br. 1992;17(1):102–7.
34. Paterson P, Titley OG, Nancarrow JD. Donor finger morbidity in cross-finger flaps. Injury. 2000;31(4): 215–8.
35. Laoulakos DH, Tsetsonis CH, Michail AA, Kaxira OS, Papatheodorakis PH. The dorsal reverse adipofascial flap for fingertip reconstruction. Plast Reconstr Surg. 2003;112(1):121–5; discussion 126–8.
36. Foucher G, Nagel D, Briand E. Microvascular great toenail transfer after conventional thumb reconstruction. Plast Reconstr Surg. 1999;103(2):570–6.
37. Koshima I, Soeda S, Takase T, Yamasaki M. Free vascularized nail grafts. J Hand Surg (Am). 1988;13:29.
38. Endo T, Nakayama Y. Microtransfers for nail and fingertip replacement. Hand Clin. 2002;18(4):615–22; discussion 623–4.
39. McCash CR. Free nail grafting. Br J Plast Surg. 1955;8(1):19–33.
40. Voche P, Merle M. The homodigital subcutaneous flap for cover of dorsal finger defects. Br J Plast Surg. 1994;47(6):435–9.
41. Jeffery SL, Pickford MA. Use of the homodigital adipofascial turnover flap for dorsal cover of

distal interphalangeal joint defects. J Hand Surg Br. 1999;24(2):241–4.
42. Quaba AA, Davison PM. The distally-based dorsal hand flap. Br J Plast Surg. 1990;43(1):28–39.
43. Maruyama Y. The reverse dorsal metacarpal flap. Br J Plast Surg. 1990;43(1):24–7.
44. Gregory H, Heitmann C, Germann G. The evolution and refinements of the distally based dorsal metacarpal artery (DMCA) flaps. J Plast Reconstr Aesthet Surg. 2007;60(7):731–9.
45. Lo S, Sebastin S, Tsai L, Pin PY. Reverse radial forearm flap peforator used in digital revascularization. Hand (N Y). 2007;2(3):155–8.
46. Söderberg T, Nyström A, Hallmans G, Hultén J. Treatment of fingertip amputations with bone exposure. A comparative study between surgical and conservative treatment methods. Scand J Plast Reconstr Surg. 1983;17(2):147–52.
47. Lister G. The choice of procedure following thumb amputation. Clin Orthop Relat Res. 1985;195:45–51.
48. O'Brien B. Neurovascular island pedicle flaps for terminal amputations and digital scars. Br J Plast Surg. 1968;21(3):258–61.
49. Elliot D, Wilson Y. V-Y advancement of the entire volar soft tissue of the thumb in distal reconstruction. J Hand Surg Br. 1993;18(3):399–402.
50. Foucher G, Delaere O, Citron N, Molderez A. Long-term outcome of neurovascular palmar advancement flaps for distal thumb injuries. Br J Plast Surg. 1999;52(1):64–8.
51. Baumeister S, Menke H, Wittemann M, Germann G. Functional outcome after the Moberg advancement flap in the thumb. J Hand Surg Am. 2002;27(1):105–14.
52. Littler JW. Principles of reconstructive surgery of the hand. In: Converse JM, editor. Reconstructiue plastic surgery, The hand and upper extremity, vol. 6. 2nd ed. Philadelphia: W. B. Saunders; 1977. p. 3139.
53. Elliot D, Southgate CM, Staiano JJ. A homodigital switch flap to restore sensation to the ulnar border of the thumb tip. J Hand Surg Br. 2003;28(5):409–13.
54. Littler JW. The neurovascular pedicle method of digital transposition for reconstruction of the thumb. Plast Reconstr Surg (1946). 1953;12(5):303–19.
55. Foucher G, Braun FM, Merle M, Michon J. La techniaue de "dibranchement-rembranchement" du lambeau. Ann Chir. 1981;35(4):301–3.
56. Büchler U, Frey HP. The dorsal middle phalangeal finger flap. Handchir Mikrochir Plast Chir. 1988;20(5):239–43.
57. Henderson HP, Reid DA. Long term follow up of neurovascular island flaps. Hand. 1980;12(2):113–22.
58. Oka Y. Sensory function of the neurovascular island flap in thumb reconstruction: comparison of original and modified procedures. J Hand Surg Am. 2000;25(4):637–43. Review.
59. Leupin P, Weil J, Büchler U. The dorsal middle phalangeal finger flap. Mid-term results of 43 cases. J Hand Surg Br. 1997;22(3):362–71.
60. Hilgenfeldt O. Operativer daumenersatz. Stuttgart: Enkeverslag; 1950.
61. Holevich J. A new method of restoring sensibility to the thumb. J Bone Joint Surg. 1963;45B:496–502.
62. Foucher G, Braun JB. A new island flap transfer from the dorsum of the index to the thumb. Plast Reconstr Surg. 1979;63(3):344–9.
63. Muyldermans T, Hierner R. First dorsal metacarpal artery flap for thumb reconstruction: a retrospective clinical study. Strategies Trauma Limb Reconstr. 2009;4(1):27–33.
64. Tränkle M, Sauerbier M, Heitmann C, Germann G. Restoration of thumb sensibility with the innervated first dorsal metacarpal artery island flap. J Hand Surg Am. 2003;28(5):758–66.
65. Del Piñal F. The indications for toe transfer after "minor" finger injuries. J Hand Surg Br. 2004;29(2):120–9.
66. Buncke HJ, Rose EH. Free toe-to-fingertip neurovascular flaps. Plast Reconstr Surg. 1979;63:607.
67. Foucher G, Merle M, Meneanen M, Michon J. Microvascular free partial toe transfer in hand reconstruction: a report of 12 cases. Plast Reconstr Surg. 1980;65:616–8.
68. Woo SH, Choi BC, Oh SJ, Seul JH. Classification of the first web space free flap of the foot and its applications in reconstruction of the hand. Plast Reconstr Surg. 1999;103(2):508–17.
69. Morrison WA, O'Brien BM, MacLeod AM. Thumb reconstruction with a free neurovascular wrap-around flap from the big toe. J Hand Surg Am. 1980;5(6):575–83.
70. Cheng G, Fang G, Hou S, Pan D, Yuan G, Wang Z, Zhang Y, Ding X, Tang H, Yang Z. Aesthetic reconstruction of thumb or finger partial defect with trimmed toe-flap transfer. Microsurgery. 2007;27(2):74–83.
71. Lin CH, Lin YT, Sassu P, Lin CH, Wei FC. Functional assessment of the reconstructed fingertips after free toe pulp transfer. Plast Reconstr Surg. 2007;120(5):1315–21.
72. Woo SH, Kim JS, Kim HH, Seul JH. Microsurgical reconstruction of partial thumb defects. J Hand Surg Br. 1999;24(2):161–9.
73. Trankle M, Germann G, Heitmann C, Sauerbier M. Defect coverage and reconstruction of thumb sensibility with the first dorsal metacarpal artery flap. Article in German. Chirurg. 2004;75(10):996–1002.
74. Turner A, Ragowannsi R, Hanna J, Teo TC, Blair JW, Pickford MA. Microvascular soft tissue reconstruction of the digits. J Plast Reconstr Aesthet Surg. 2006;59(5):441–50.
75. Thatte MR, Thatte RL. Venous flaps. Plast Reconstr Surg. 1993;91(4):747–51. Review.
76. Chen HC, Tang YB, Noordhoff MS. Four types of venous flaps for wound coverage: a clinical appraisal. J Trauma. 1991;31(9):1286–93.
77. Woo SH, Kim KC, Lee GJ, Ha SH, Kim KH, Dhawan V, Lee KS. A retrospective analysis of 154 arterialized venous flaps for hand reconstruction: an 11-year experience. Plast Reconstr Surg. 2007;119(6):1823–38.
78. Zancolli EA, Angrigiani C. Posterior interosseous island forearm flap. J Hand Surg Br. 1988;13(2):130–5.

79. Chen HC, Cheng MH, Schneeberger AG, Cheng TJ, Wei FC, Tang YB. Posterior interosseous flap and its variations for coverage of hand wounds. J Trauma. 1998;45(3):570–4.
80. Hidalgo DA, Shaw WW. Anatomic basis of plantar flap design. Plast Reconstr Surg. 1986;78(5):627–36.
81. Inoue T, Kobayashi M, Harashina T. Finger pulp reconstruction with a free sensory medial plantar flap. Br J Plast Surg. 1988;41(6):657–9.
82. Lee HB, Tark KC, Rah DK, Shin KS. Pulp reconstruction of fingers with very small sensate medial plantar free flap. Plast Reconstr Surg. 1998;101(4):999–1005.
83. Huang SH, Wu SH, Lai CH, Chang CH, Wangchen H, Lai CS, Lin SD, Chang KP. Free medial plantar artery perforator flap for finger pulp reconstruction: report of a series of 10 cases. Microsurgery. 2010;30:118–24.
84. May Jr JW, Chait LA, Cohen BE, O'Brien BM. Free neurovascular flap from the first web of the foot in hand reconstruction. J Hand Surg Am. 1977;2(5):387–93.
85. Shi SM, Lu YP. Island skin flap with neurovascular pedicle from the dorsum of the index finger for reconstruction of the thumb. Microsurgery. 1994;15(2):145–8.
86. Wei FC, Chen HC, Chuang CC, Chen SH. Microsurgical thumb reconstruction with toe transfer: selection of various techniques. Plast Reconstr Surg. 1994;93(2):345–51.

Distal Interphalangeal Joint and Fractures of the Distal Phalanx

7

Subodh Deshmukh and Christopher Armitstead

Keywords
Distal phalanx • DIPJ • Fractures • Dislocation • Amputation • Subungual haematoma • Nail avulsions • Nail bed lacerations • Mallet finger • Jersey finger

Introduction

In this chapter we address injuries to the tips of the fingers and the disorders of the distal interphalangeal joint. It is important to appreciate that trauma to this area produces a variety of injury patterns, not only to the distal phalanx itself, but also to the other surrounding non-bony structures (Fig. 7.1). Injury to the tip of the finger is common and accounts for approximately half of all hand injuries [1] and is usually the result of a sporting, occupational or domestic accident. These injuries are important to recognise and treat appropriately to maintain hand function and prevent permanent disability.

S. Deshmukh, MS, MCh (Orth), FRCS, FRCSG, FRCS (Orth) (✉)
Department of Trauma and Orthopaedic, City Hospital, Dudley Road, Birmingham, B18 7QH, UK
e-mail: subodh.deshmukh@nhs.net

C. Armitstead, MBBS (Lond), MRCS (RCSoE), FRCS (Tr & Orth)
Department of Hand Surgery, Leighton Hospital, Crewe, Cheshire, UK
e-mail: chris-armitstead@doctors.org.uk

Anatomy of the Distal Phalanx

The nail is formed from a flattened sheet of keratinised squamous cells and acts as a shield to protect the underlying bone and dense sensory units of the finger pulp. It is also said that the nail improves two-point discrimination by acting as a counterforce to the tip of the finger when an object is touched [2]. Ninety percent of the nail is formed by the germinal matrix. The most distal edge of the germinal matrix is seen as a white arc under the proximal end of the nail and is called the lunula. The distal, dorsal fold of skin, or eponichyum is responsible for producing the shine on the nail. The nail lies on the sterile matrix, which adds squamous epithelia to

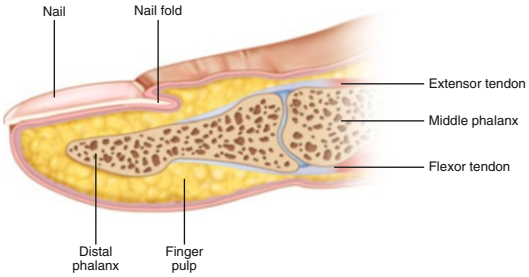


Fig. 7.1 Anatomy of the fingertip

I.A. Trail, A.N.M. Fleming (eds.), *Disorders of the Hand: Volume 1: Hand Injuries*,
DOI 10.1007/978-1-4471-6554-5_7, © Springer-Verlag London 2015

the advancing nail, thickening and adhering the nail to the fingertip. Damage to the germinal matrix stops nail formation, whereas injury to the sterile matrix can lead to nail deformity. The nail is bordered either side by the skin folds of paronychium and the skin distal to the sterile matrix is called the hyponychium. Neural supply to the paronychium comes from the terminal branches of the median and ulnar digital nerves and the vascular supply from the terminal branches of the deep and superficial palmar arch and the terminal branches of the radial artery, before it joins to form the deep palmar arch. The venous drainage from the proximal nail bed and nail fold flow to the dorsal finger along with the lymphatic and then towards the dorsal venous plexus of the hand. The hyponychium is believed to contain the greatest density of lymphatics of any dermal area in order to help prevent infection [3]. The nail growth, colour, shape and texture is affected by many systemic conditions, ranging from chronic anaemia (Koilonychia) to psoriasis and local conditions like ring worm and fungal infection, chronic bacterial infection (paronychia), loss of the distal support (hook nail deformity) as well as previous injuries producing split nail deformity and ridging of the nail.

The distal phalanx is divided into the tuft, shaft and peri-articular portions. The tuft is flattened and widened to help support the nail and has multiple fibrous septa attaching it to the volar skin. Proximally, in the peri-articular region, the extensor mechanism attaches dorsally. The flexor digitorum profundus attaches volarly, distal to the attachment of the volar plate, but over a broader area compared to that of the extensor mechanism.

Injury to the fingertip is associated with bony injury in 50 % of cases, with crushing type injuries being the most common mechanism. Catching the tip of the finger in a door is a common source of injury, as well as being trapped between two objects and lacerations from sharp objects. The most commonly injured digit is the middle finger, probably because of its exposed position, as the longest finger of the hand.

Presentation, Investigation and Treatment Options

Presentation

The patient may present to either the General Practitioner or more commonly, to the Accident and Emergency department. Dominance, occupation, age of the patient, mechanism of injury, where the injury occurred, time since the injury and other associated injuries are factors that should be noted. These features can be important as they may influence treatment.

The mechanism will indicate the severity of the trauma. A sharp object compressing the nail and bone may cause a splitting laceration, a simple nail bed laceration and possible fracture. A blunt mechanism of injury would indicate a wider area of compression, and cause damage ranging from a subungual haematoma to more severe damage to the nail bed, leading to a stellate laceration and even a comminuted fracture of the tuft. The time since injury is important (especially for amputation injuries where revascularisation is being considered) along with the assessment for other injuries.

Investigations

The investigation starts with clinical examination of the digit, which includes assessment of the soft tissues, neurovascular supply and tendon function. X-rays are requested to assess the integrity of the underlying bone and state of the Distal Interphalangeal Joint (DIPJ).

Treatment Options

The initial management should be to clean any wounds with normal saline and to apply a dressing (preferably sterile and non-adherent). This may require some form of analgesia or a digital nerve block.

The Subungual Haematoma

Crush injury of the nail onto the underlying distal phalanx can lead to bleeding beneath the nail. If

the nail is intact, the pressure of the blood within this confined space causes severe throbbing pain. Evacuation of the haematoma using a sterile technique is indicated and performed by trephination.

In the past, the size of the haematoma has been used as an indicator of whether to remove the nail and repair the nail bed or not. Seaberg et al. [4] showed that, regardless of haematoma size, no nail deformity was seen with drainage alone. However, it is commonly felt that if the haematoma is larger than 50 % of the nail, or the nail itself is damaged, the nail bed should be explored.

Avulsion of the Nail

Often the avulsed nail has a section of nail bed still attached, usually distally. In these cases the nail may well have flipped out of the eponychium and, or the paronychium. In children this may indicate a physeal injury and x-rays are advised. If there are significant defects, (greater than 3 mm), the avulsed nail bed should be used as a graft. Larger defects may require split thickness grafts, either from the same finger, an adjacent finger or even a toe. Germinal matrix grafts, however, will usually call for full thickness grafts.

Fractures of the Distal Phalanx

Fractures of the distal phalanx are the most common fractures in the hand. Schneider [5] classified fractures of the distal phalanx into (1) tuft fractures, (2) shaft fractures and (3) articular fractures (Fig. 7.2).

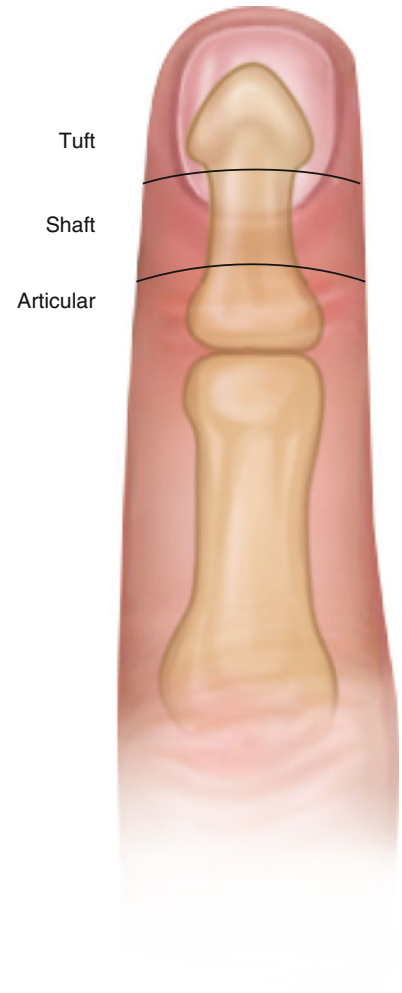


Fig. 7.2 Fractures of the distal phalanx

Clinical Pearls: Spectrum of Fracture Patterns

Tuft fractures	
Simple ↔	Comminuted
Shaft fractures	
Transverse = stable	Unstable = oblique/ longitudinal
Articular fractures	
Volar (rule out profundus accompanying avulsions)	Dorsal (mallet fractures)
Fracture dislocation/subluxation	
Volar	Dorsal

Fractures of the Tuft of the Distal Phalanx

Tuft fractures can be described as either simple or comminuted and are usually caused by a crushing injury. The closed tuft fracture is often associated with a subungual haematoma and is treated by nail trephination. The risk of introducing infection should be covered by prescribing oral antibiotics. These types of fractures rarely require fixation, with more attention being paid to the associated soft tissue injury. Occasionally these fractures go onto a non-union, although usually they are asymptomatic, being stabilised by the new nail.

Fig. 7.3 Non-union of the distal phalanx

Fractures of the Shaft of the Distal Phalanx

Distal phalanx shaft fractures are described as longitudinal or transverse, stable or unstable. Due to the soft tissue envelope stabilisation, the un-displaced fracture can usually be treated with 3–4 weeks of mallet finger splint immobilisation (including the DIP joint), followed by gentle active rehabilitation. Displaced fractures are usually associated with greater soft tissue damage, due to the higher energy of the insult, and as a consequence stability is usually compromised and may result in a non union (Fig. 7.3).

Fractures distal to the nail fold may be associated with subluxation of the nail plate out of the eponychial fold, which can result in germinal or sterile matrix damage. Accurate reduction of the fracture thus avoiding the step in the dorsal cortex minimises abnormal nail growth. Often this cannot be achieved closed and may require surgical intervention to repair the nail bed and accurately reduce displaced fractures using a 1.1 mm longitudinal Kirschner wire with or without transfixing the DIPJ. Hyperflexion injuries to the distal phalanx in children can result in Salter-Harris Type I or occasionally Type II epiphyseal injuries. The position of the tendon insertions dictates the displacement of the fracture. Typically these fractures deform with the apex dorsally. Both the extensor and the flexor of the distal phalanx are inserted on the proximal fragment. Seymore [6] described this fracture of the juxta-articular region in children with the nail subluxed from the eponychium. These were treated conservatively with better results than operative treatment. If repair of the nail bed is performed, Day and Stern [7] advises to avoid pinning of the fracture, as growth disturbances of the physis are not uncommon.

Fig. 7.4 Bony Mallet finger

Intra-articular Fractures of the Distal Phalanx

Intra-articular fractures are either avulsion fractures of the dorsal (Fig. 7.4) or volar lip of the articular surface, or less frequent communited fractures of the base.

The majority are of the “mallet” type, following an axial load with subsequent avulsion of a dorsal bony fragment with the extensor tendon attached to it. Most mallet injuries, with or without bony injury, can be treated conservatively with splinting of the distal phalanx in extension for 6–8 weeks, and 1 month of night splinting. Bony fragments appear to afford better tendon healing [8]. Slight extensor lag may be seen after

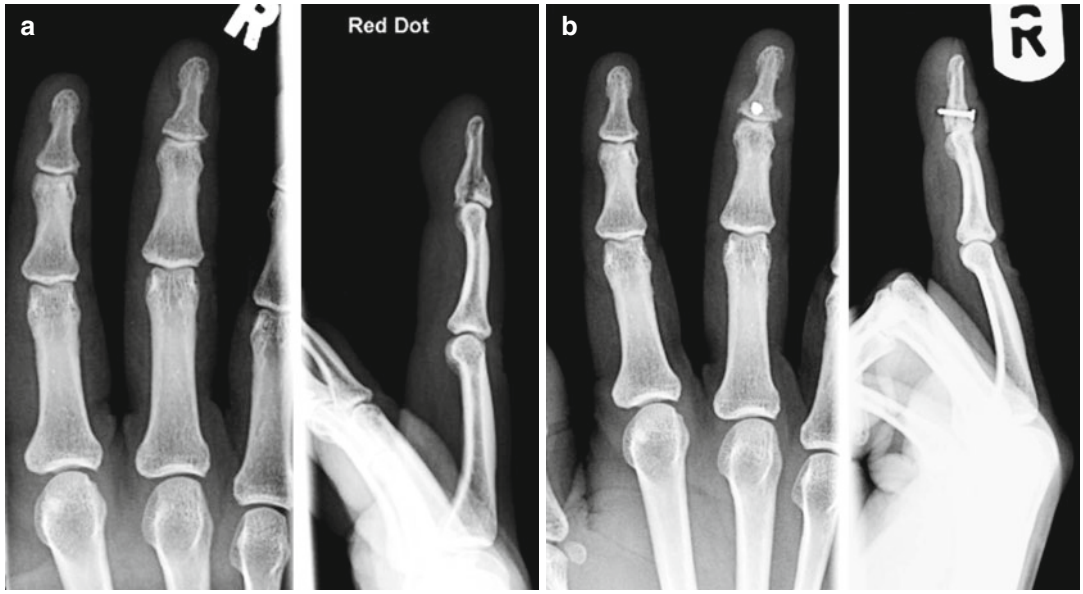


Fig. 7.5 (**a**, **b**) Intra-articular fracture distal phalanx treated by screw fixation

treatment and if the deformity recurs further splintage for 1–2 months may improve the position. A chronic mallet deformity is defined as an injury that occurred more than 3 months ago, however, splinting can still be tried with reasonable results. If there is associated volar subluxation of the distal phalanx, or if the avulsed fragment is greater than a third of the articular surface then it is recommended that these fragments are anatomically reduced and internally fixed either with a dorsal screw (Fig. 7.5a, b) or a hook plate.

Jersey Finger, or Flexor Digitorum Profundus (FDP) Tendon Avulsions

Jersey finger, or Flexor Digitorum Profundus (FDP) tendon avulsions can occur with or without a bony avulsion fragment of the volar articular cartilage. The deformity usually presents when an actively flexed DIP joint is forced into extension and most commonly affects the ring finger [9]. The classic symptom is a sudden loss of the ability to flex at the DIP joint. Classification by Leddy and Packer [10] divided the injury based on how far the tendon retracts, which dictates the proposed management. Type 1 is diagnosed when the tendon is retracted to the palm with disruption to the vincula and as such the blood supply. Because the tendon can become avascular and the musculo-tendinous unit can shorten quickly, any delay in repair can lead to a poor result. Ideally the repair should be performed within 24–48 h. In Type 2 injuries, the most common subtype, the intact vincula hold the tendon at the level of the PIP joint. This means that the blood supply to the tendon is less of an issue and repair can be undertaken up to 6 weeks from the injury. Type 3 injuries have a large bony fragment attached, that prevent the tendon from retracting further than the A4 pulley (Fig. 7.6).

Again, since ischaemia and muscle and tendon shortening are not an issue, a delayed repair is possible. If the avulsed fragment is large it can be fixed back to the distal phalanx with screws. Type 4 [11] injuries have a fracture of the base distal phalynx which is usually a volar lip fracture and the tendon has pulled out from the fragment. The treatment for all types is anatomic repair of the tendon, using either anchors in the distal phalanx or tie-over pull through sutures, tied over the nail with or without fixation of the volar lip fracture of the distal phalynx (Fig. 7.7).

Fig. 7.6 Jersey finger Type 3

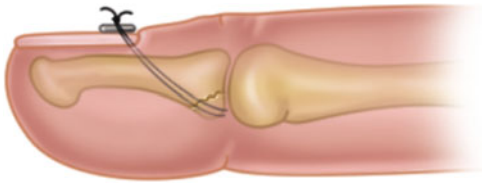


Fig. 7.7 Fixation of volar lip fracture with a pull-through suture

DIP Joint Dislocation

Pure dislocations of the DIP joint are rare. When they do occur, however, commonly they are found to be dorsal, often following a hyperextension injury of the distal phalanx (Fig. 7.8a).

They can also be associated with a volar split type skin wound. The rarity of this injury is thought to be due to the short lever arm of the distal phalanx and the stability provided by the close attachment of the flexor and extensor tendons. Attempted closed reduction, with exaggeration of the direction of initial injury followed by gentle pressure over the base of the distal phalanx, is the initial treatment of choice. If, however, the dislocation is irreducible, open reduction may be necessary followed by either antegrade or retrograde trans-articular Kirschner wire fixation. Open dislocations should be treated with irrigation, debridement and reduction. The position, if stable, can be splinted in extension for 2–3 weeks

Fig. 7.8 (**a**) DIPJ Dorsal dislocation. (**b**) **DIP** joint fracture dislocation

prior to mobilization. Unstable reductions and fracture dislocations (Fig. 7.8b) must be reduced and internally fixed as appropriate. Large bony fragments may be fixed using screws through an additional volar or dorsal approach.

Clinical Pearl: Causes of Irreducible Dislocations

Dorsal dislocation with volar plate entrapment – most common
Entrapped FDP
Entrapped osteo-chondral fragment
Buttonholing of the distal condyles of the middle phalanx through FDP
Chronic dislocation

Amputations of the Fingertip

Treatment is based on (a) the nature of the injury (b) extent of wound contamination and (c) assessment of the extent of crushing of both the remaining proximal tissue and the amputated part.

Very distal amputations (with skin loss of less than 1 cm^2 and with reasonable soft tissue coverage of the bone) can be left to heal with secondary intention. Alternatively, the amputated fragment can be

defatted and used as a full thickness skin graft. As the wound contracts and epithelialisation occurs, well-innervated skin can be pulled into defect, however healing can take up to 2 months [11].

When the bone is exposed, the choice is either to trim the distal phalanx to allow soft tissue coverage or to use local rotation or skin flaps. The nail is left in situ if over 25 % of the nail bed remains intact. However, if less than 25 % or less than 5 mm of sterile matrix is intact, resection of the nail is recommended along with the eponychial fold and germinal matrix. If the resection jeopardises the flexor and extensor insertions and either cannot be left intact, then the distal phalanx should be disarticulated. The tendons are placed under tension, transected and allowed to retract. The tendons should not be sutured together as this will cause tethering of the FDP.

The success rate for re-implantation is variable at the level of the eponychiyum, due to the size of the digital vessels. The digital artery begins to branch at the level of DIPJ and dorsal veins are hard to find.

Composite graft of skin nail bed and nail is used in children [12], generally more successful in children under 3 years. As an alternative or for older children, cap grafts, without the bony fragment, can be effective.

Surgical Techniques and Rehabilitation

Anaesthesia/Analgesia

Most surgery performed on the fingertip can be safely performed under local anaesthetic digital nerve block. The type of local anaesthetic used depends on the surgeon's preference. However, a mixture of short acting Lidocaine and longer acting Bupivacaine is the author's choice. The needle is introduced dorsally, into either of the intermetacarpal spaces deep enough to block both digital nerves and then across the dorsum of the finger to block the dorsal branches of the digit. Small volumes should be used to avoid circumferential pressure and a total of 5–7 ml is generally enough. One can do the same through the volar approach, but the dorsal cutaneous nerves are difficult to block and may require a separate needle insertion point.

The use of adrenaline with local anaesthetic to achieve haemostasis is contentious and historically is thought to be a risk that is worth avoiding. However, more recent studies suggest that the vasoconstriction improves haemostasis and decreases the need for a tourniquet [13, 14]. Chowdhry et al. [15] revisited this topic in 2010, when he retrospectively reviewed 1,111 cases and found no complications in either the plain local anaesthetic group or the group that was injected with local anaesthesia and epinephrine.

Tourniquet

Salem's method, using a single finger from a sterile glove with the tip cut off and rolled to the base of the finger is very useful as is a Penrose drain or paediatric catheter. However, the NHS has recently released a warning statement highlighting the risk of tourniquets used for finger and toe surgery [16]. They suggest that "CE marked digital tourniquets which are labelled and/or brightly coloured should be used, in accordance with manufacturers' instructions. Surgical gloves should not be used as tourniquets."

Subungual Haematoma

Haematoma evacuation should be performed only after thorough surgical preparation. The nail can be breeched using a variety of methods, drills, needles, heated paperclips or cautery needles, as long as the hole is large enough to allow continued drainage of the haematoma. In cases where there are underlying fractures, theoretically as one is converting a closed injury into an open fracture, antibiotic use should be considered.

To Remove the Nail or Not?

As discussed previously, it is believed that if the nail is intact and the haematoma occupies

less than 50 % of the nail, then the nail should be left in situ. If, however, the nail is damaged, or the haematoma occupies more than 50 % of the nail bed, then the nail should be removed to explore and if appropriate repair the sterile matrix.

Nail Removal

Many techniques have been described as to how to remove the nail. Periosteal elevators, blunt forceps, or scissors are the common tools used to lift the nail from the remaining attached sterile matrix. If curved instruments are used, it is recommended that they should be used with the curve pointing towards the nail, to avoid further damage to the nail bed. The nail is teased away from the sterile matrix from the hyponychium to the eponychium.

Fixation Techniques

Kirschner wires can be used to reduce the fracture and in turn support the bone and the nail while healing occurs. In the presence of small nail bed lacerations, the wire is passed retrograde across the fracture from the fingertip. With large nail bed lacerations, after thorough debridement, the wire can be passed antegrade through the fracture into the distal fragment, then retrograde into the proximal fragment under direct vision. The wire is usually placed a few millimetres palmer to the nail plate, as the tip of the distal phalanx lies in the dorsal half of the fingertip. It is also generally advised to avoid crossing the DIP joint, for fear of damaging the articular cartilage and also wire breakage at the level of the joint can be troublesome. A 1.6 mm wire is recommended, which is left proud to facilitate removal in the outpatient setting. Alternative fixation choices with either a single wire or two crossed/parallel wires are dictated by fracture configuration.

Clinical Pearl: Fixation Tips

Use 1.6 mm 'K' wire.

Antegrade then retrograde passage of the wire allows for better apposition of the fracture.

Aim just under nail plate as the tip of distal phalanx lies in the dorsal half of the fingertip.

Avoid crossing the DIP Joint for fear of articular cartilage damage and wire breakage.

Replacing the Nail

The nail should be replaced after fixation for several reasons. Primarily, the nail adds further stability to the distal phalanx fixation; it also keeps the nail fold open and reduces scarring in the germinal and sterile matrix, reducing the possibility of nail deformity. If the natural nail is damaged, a substitute nail can be fashioned from a foil suture packet, a silicon sheet or the reservoir of an IV giving set.

Fixation of the Nail

Once replaced, the nail can be secured using either sutures at each corner, a figure of eight suture (Fig. 7.9), adhesive glue or with steri-strips.

Bony Mallet Finger Fixation Techniques

The preferred techniques are either percutaneous transarticular Kirschner wire fixation, internal fixation with one or occasionally two screws (1.3 or 1.5 mm inter-fragmentary screws) or Birmingham hook plate [17, 18] (Fig. 7.10a, b).

Jersey Finger

Surgical repair using heavy, non-absorbable pull out suture, tied through a button over the nail or alternatively one or two anchors in the distal phalanx are options.

Fig. 7.9 Figure of eight suture fixation of nail

Amputation

A fish mouth or mid-axial skin incision should be used to help preserve the length of the digit, but also to help identify the neuro-vascular bundles. The bony attachments of the FDP and extensor tendons should be carefully preserved as far as possible. The distal phalanx or middle phalangeal condyles are contoured and rounded off with rongeurs. During disarticulation through the DIPJ, the tendons are divided under mild tension and then left to retract. The cutaneous nerves are treated similarly to ensure the ends are kept away from the skin edges. The skin flaps are then fashioned with preservation of the volar skin is preferred. Cutler's flaps or Atasoy's V-Y advancement flaps can be used to bring soft tissues to cover the exposed bone of distal phalanx.

Non-unions of the shaft can be treated by either parallel or crossed K-wires fixation. A recent paper published suggests that interfragmentary screw fixation is also a reliable technique [19].

Outcomes

The outcome following distal phalangeal injuries is dependent upon several factors, including the patient, the nature and extent of the injury, as well as surgical expertise. Poor results have been

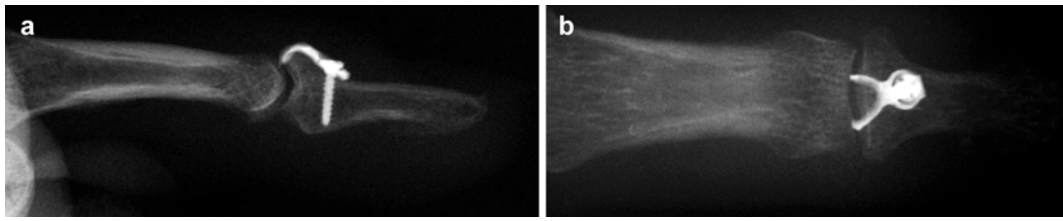


Fig. 7.10 (**a**, **b**) Fixation using Birmingham hook plate (Courtesy of Mr Kanthan Theivendran)

documented with patients older than 50 years and with associated systemic illness. High-energy fractures with comminution and associated extensive soft tissue injury also lead to poorer outcomes. Tendon injury, especially extensor tendon, in association with fracture also compromises results. Factors under the control of the surgeon include selecting the appropriate fixation and assuring that immobilization does not exceed 3 weeks.

DaCruz et al. [20] performed a prospective study of 110 patients with fractures of the distal phalanx and reported that less than one in three patients, with such injuries, recovered after 6 months and non-union rates for shaft fractures were as high as 47 % after 6 months. Factors that carried a poor prognosis included osteolysis of fractured fragments, subungual haematomas and non-union.

Complications of Treatment

Fingertip deformities can be caused by problems of the soft tissue, either the germinal matrix or sterile matrix, or bony complications with uneven dorsal cortex, loss of bony support, non-union or infection. Nail ridges form either longitudinally or transversely as a result of an uneven dorsal cortex, or nail bed scarring. The deformity is generally a cosmetic problem, although in the case of a transverse ridge, the nail can be lifted off the nail bed causing the free nail edge to catch on clothes etc. Lifting the nail and excising the scar, or levelling the dorsal cortex can rectify the problem.

A split nail can occur as the result of a longitudinal ridge or scar in the germinal or sterile matrix, causing an absence of nail production in a small area. The treatment involves nail removal and scar excision. If the residual defect is small (less than 2 mm), it can be closed directly. However, larger defects require a full thickness germinal matrix graft (usually from the toe). For a sterile matrix defect, only a split thickness graft is needed. This can be taken from an adjacent area or toenail bed.

Complete absence of the nail can occur after severe damage to the germinal matrix and treatment can be difficult. Some have tried full thickness or split thickness skin grafts to mimic the nail, others have tried composite grafts to reconstruct. There have also been reports of free micro-vascular transfer of the dorsal tip of the toe, although the surgery is very demanding and success is not guaranteed.

Nail spikes and cysts are common complications when remnants of the germinal matrix from the nail fold are left. Treatment involves complete removal of the germinal matrix.

Eponychial deformities are caused by injury and scarring of the nail fold, thus emphasising the importance of separating the eponychial fold from the underlying germinal matrix, post-operatively using either the original nail or a substitute. A pterygial fold of the eponychium could lead to absence of nail growth or nail splitting. After resection of scar tissue, the nail fold will have to be freed and a composite graft used to reconstruct the nail fold.

Hyponychial deformities can result from too tight a closure of the skin over the tip of the finger resulting in pain and occasionally hooking of the nail (Fig. 7.11). This can also occur with loss of nail support, specifically after distal phalanx shortening. Nail growth follows the sterile matrix and if this curves over the fingertip in a volar

Fig. 7.11 Hook nail deformity due to loss of distal phalangeal support

direction, the nail will follow. Treatment consists of either shortening the nail bed, or increasing the support under the nail.

Paronychia is an acute infection of the lateral nail fold, which requires drainage. Again, however, this can lead to nail deformities. Sometimes partial excision of the nail on the side of the infection might be required. Chronic paronychia is a difficult condition to treat and may require complete excision of the nail.

Complications following amputation include a lumbrical plus finger, when the FDP tendon is released without release of the lumbrical muscle. The un-checked FDP tendon acts on the lumbrical's insertion into the lateral band (which passes dorsally into the triangular ligament), causing the PIP joint to paradoxically extend. Treatment in this case is to release the lumbrical muscle from its insertion. Neuromas can also be problematic following amputation. They express trophic factors in an attempt to regenerate, forming a mass of Schwann cells at the site of injury, producing a painful nodule at the nerve end. Treatments include a variety of methods, including cauterization, nail stripping and excision, or transfer into a muscle, vein or bone. Tight stretched-out or retracted skin over the amputation stump of the distal phalanx can become painful and may require the use of a thimble, or amputation at a more proximal level to allow the skin to be relaxed over the bone.

DIP Joint Fusion

Among the many indications for fusion of the DIP joint, most are related to chronic complications of previously treated joint injuries, for example chronic pain, deformity and functional loss. Joint destruction secondary to post traumatic arthritis, osteoarthritis, rheumatoid arthritis or post infection, together with chronic tendon ruptures and scarring from burns can also be treated with arthrodesis. Fusion should not be contemplated in cases with active infection.

Fixation options include use of Kirchner wires with or without a tension band wire or an intraosseous cerclage wire, headless bone screws (Fig. 7.12) and plates.

Fig. 7.12 DIPJ fusion using the retrograde Herbert screw technique

Generally, the DIP joints of the lesser fingers are fused in slight flexion.

The Technique

The DIP joint is approached using a dorsal H, transverse or Y-incision. The extensor mechanism is exposed and incised transversely, with release of the collateral ligaments if needed. A small oscillating saw is used to flatten the joint surfaces. Depending on the type of fixation, the bone ends are approximated, the reduction is checked with fluoroscopy and the wound is closed in layers. Post-operatively, a light dressing is applied and a protective volar splint is fabricated. At this time active PIP joint movement is encouraged. The finger is X-rayed at 6 weeks and 3 months to check for union. Complications include pin-tract infection, deep infection/osteomyelitis, painful or protruding hardware requiring removal, non-union and cold intolerance.

Clinical Pearl: Fixation Tips for Fusion

Headless bone screw fixation is useful for fixation in full extension

For flexion at the DIP Joint, tension band wire technique is reliable but technically more difficult.

Parallel 'K' wire fixation is adequate in most cases and can help with rotational stability.

Outcomes

Union rates with inter-osseous wiring techniques have been reported at between 80 and 100 %, which are comparable to the use of headless bone screw fixation (95–100 %) [20]. The non-union rates for screw fixation, described in the literature are between 0 and 15 %. However, the 2011 study by Kocak [21] stated that the 'technical simplicity and rigid stability of fixation with compression screw placement makes them the ideal choice in most situations'.

Summary

Injuries to the distal phalanx and DIPJ are both common and potentially disabling. It is the dexterity and sensory perception of the distal phalanx that affords the human hand such excellent function and even a minor injury can potentially be disastrous. When dealing with fingertip injuries the ultimate aim is to achieve a stable, mobile, pain free and sensate distal tip to the digit. However the hand being the second most visible parts of the body (second only to the face), surgeons should always have functional and aesthetic considerations in mind whilst treating these injuries.

References

1. Hove LM. Fractures of the hand. Distribution and relative incidence. Scand J Plast Reconstr Surg Hand Surg. 1993;27(4):317–9.
2. Zook EG. Anatomy and physiology of the perionychium. Hand Clin. 1990;6:1–7.
3. Zook EG. Fingernail injuries. In: Strickland JW, Steichen JB, editors. Difficult problems in hand surgery. St. Louis: CV Mosby; 1982.
4. Seaberg DC, Angelos WJ, Paris PM. Treatment of subungual hematomas with nail trephination: a prospective study. Am J Emerg Med. 1991;9:209–10.
5. Schneider LH. Fractures of the distal phalanx. Hand Clin. 1988;4:537–47.
6. Seymore N. Juxta-epiphyseal fracture of the terminal phalanx of the finger. J Bone Joint Surg (Br). 1966; 48(2):347–9.
7. Day CS, Stern PJ. Chapter 8. Fractures of the metacarpals and phalanges. In: Green's operative hand surgery, vol. I. 6th ed. Elsevier: Churchill Livingstone; Philadelphia 2010. p. 239–90.
8. Hamas RS, Horrell ED, Pierret GP. Treatment of mallet finger due to intra-articular fracture of the distal phalanx. J Hand Surg [Am]. 1978;3:361–3.
9. McCue 3rd FC, Wooten SL. Closed tendon injuries of the hand in athletics. Clin Sports Med. 1986;5:741–55.
10. Leddy JP, Packer JW. Avulsion of the profundus tendon insertion in athletes. J Hand Surg [Am]. 1977;2: 66–9.
11. Henry SL, Katz MA, Green DP. Type IV FDP avulsion: lessons learned clinically and through review of the literature. Hand (N Y). 2009;4(4):357–61.
12. Fox J, Golden G, Rodeheaver G, Edgerton M, Edlich R. Nonoperative management of fingertip pulp amputation by occlusive dressings. Am J Surg. 1977;133(2): 255–6.
13. Moienem NS, Elliot D. Composite graft replacement of digital tips. A study in children. J Hand Surg (Br). 1997;22(3):346–52.
14. Wilhelmi BJ, Blackwell SJ, Miller JH. Do not use epinephrine in digital blocks: myth or truth? Plast Reconstr Surg. 2001;107:393–7.
15. Chowdhry S, Seidenstricker L, Cooney D. Do not use epinephrine in digital blocks: myth or truth? Part II. A retrospective review of 1111 cases. Plast Reconstr Surg. 2010;126(6):2031–4.
16. NHS Rapid Response Report NPSA/2009/RRR007: reducing risks of tourniquets left on after finger and toe surgery, December 2009
17. Teoh LC, Lee JYL. Mallet fractures: a novel approach to internal fixation using a hookplate. J Hand Surg (Br & Eur). 2007;32(1):24–30.
18. Theivendran K, Mahon A, Rajaratnam V. A novel hook plate fixation technique for the treatment of mallet fractures. Ann Plast Surg. 2007;58(1):112–5.
19. Chim H, Teoh LC, Yong FC. Open reduction and interfragmentary screw fixation for symptomatic nonunion of distal phalangeal fractures. J Hand Surg (Eur). 2008;33(1):71–6.
20. DaCruz DJ, Slade RJ, Malone W. Fractures of the distalphalanges. J Hand Surg (Br). 1988;13:350–2.
21. Kocak E, Carruthers KH, Kobus R. Distal interphalangeal joint arthrodesis with the Herbert headless compression screw: outcomes and complications in 64 consecutively treated joints. Hand (N Y). 2011;6(1):56–9.

Proximal Interphalangeal Joint Injuries

8

Grey Giddins and Lawrence Moulton

Keywords
Clinical presentation • Investigation • Soft tissue injuries • Proximal phalangeal fractures • Extra and intra articular • Middle phalangeal fractures • Fracture dislocation • Surgical treatment • Outcome

Introduction

These are common injuries, particularly to the base of the middle phalanx. The PIP joints are the most important joint in the fingers, so pain and stiffness can be very limiting particularly on the ulnar side of the hand. Fortunately, most injuries are mild and can be treated successfully with early mobilisation. Conversely complex injuries, particularly of the base of the middle phalanx, can be very challenging to treat.

G. Giddins, BA, MBBCh, FRCS(Orth) FRCSEd (✉)
Department of Orthopaedic, The Royal United Hospital, Combe Park, Bath BA1 3NG, UK
e-mail: greygiddins@thehandclinic.co.uk

L. Moulton, MBChB(Hons), MRCS(Ed)
ST5 Trauma and Orthopaedics, All Wales Rotation, Morriston Hospital, Heol Maes Eglwys, Morriston, Swansea SA6 6NL, UK

University Hospital of Wales, ST6 Trauma and Orthopaedics, Heath Park, Cardiff CF14 4XW, UK
e-mail: lawrence.moulton@doctors.net.uk

Background/Aetiology

Injuries to the PIP joint typically follow either a fall on an outstretched hand, where the fingers have not got out of the way in time, or as a result of a localised injury to a single finger, such as the missed catch of a ball. Other mechanisms include traction injuries, such as a pull on a finger through a rope or a lead twisted around the finger.

The mechanism of injury is typically a hyperextension injury, although oblique, lateral and volar loading can occur. The deforming forces of the injury are resisted primarily by the ligamentous structures around the joint (Fig. 8.1). Some lateral stability is conferred by the condylar shape of the joints but the majority of the deforming forces are resisted by the volar plate and collateral ligaments. Volar deforming forces are typically accommodated by the natural flexion of the joint with the tendons, in particular the central slip insertion, resisting hyperflexion. Otherwise the tendons around the joint do not confer much stability. Once there is instability the tendons provide the forces that dictate the residual deformity. In particular, the flexor digitorum superficialis tendon insertion into the middle phalanx and the central slip insertion

I.A. Trail, A.N.M. Fleming (eds.), *Disorders of the Hand: Volume 1: Hand Injuries*,
DOI 10.1007/978-1-4471-6554-5_8, © Springer-Verlag London 2015

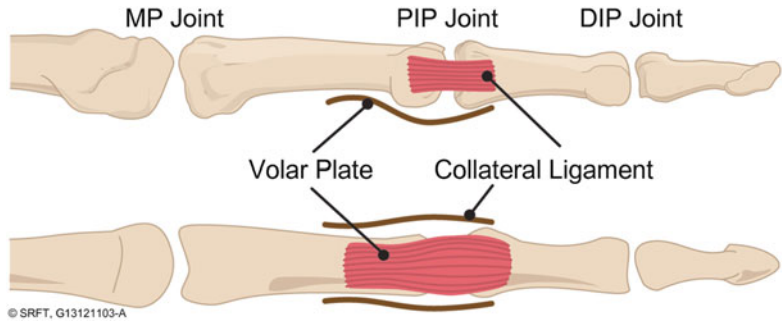


Fig. 8.1 Side and volar views of the PIP joint showing the normal ligamentous and tendinous restraints

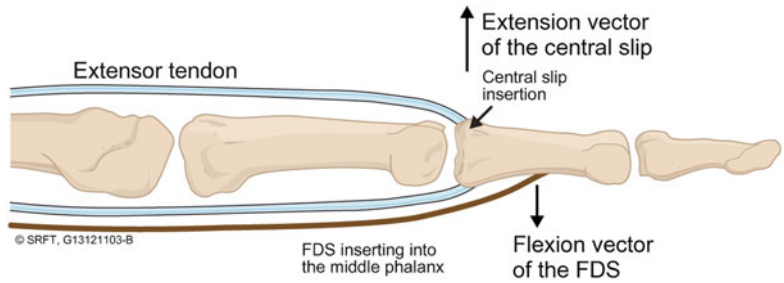


Fig. 8.2 Diagram of the forces leading to PIP joint dorsal subluxation following a volar base of middle phalanx fracture

Combined vector at the base of the middle phalanx resisted by the volar plate & volar lip of the base of the middle phalanx

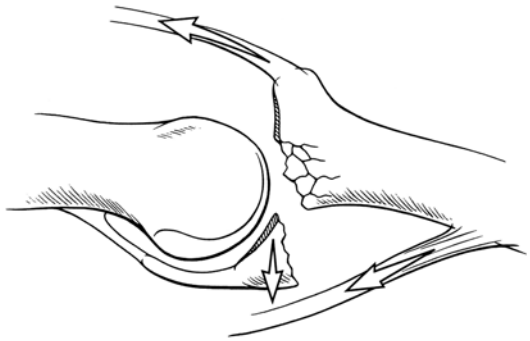


Fig. 8.3 Diagram of a dorsal dislocation of the middle on the proximal phalanx

dorsally to the base of the middle phalanx lead to a flexion and a subluxation vector (Fig. 8.2). This is normally resisted by the bony anatomy, particularly the collateral ligaments and especially the volar plate. If these fail then the base of the middle phalanx will often sublux and even dislocate dorsally relative to the head of the proximal phalanx (Fig. 8.3). If the bony anatomy is in tact or largely in tact then the joint congruency provides enough early stabilisation, provided the joint is reduced correctly. If there is sufficient damage to the bony anatomy, however, then there will inevitably be instability in the joint, typically with malalignment.

Finally, it is important to remember that concave joint surfaces, such as the base of the middle phalanx, are generally more tolerant of some malalignment than convex joint surfaces. This is especially true of the PIP joint, but is also seen in the wrist, knee and ankle. The response to injury i.e. deforming forces is also different: concave joint surfaces tend to fragment; whereas convex joint surfaces tend to have a single split or T-shaped fracture in more severe injuries.

Presentation, Investigation and Treatment Options

Patients will normally present with a reasonably clear-cut history of injury. It is usually an isolated injury to a singel PIP joint. However, it is important to remember that PIP joint injuries can easily be overlooked in patients with multiple injuries, such as a fall from a height. The patient may also give a history of marked deformity in the joint, compatible with dislocation, which may have been reduced at the site of injury. On presentation they will report pain, swelling and stiffness centred around the PIP joint and possibly some altered sensibility in the finger.

Fig. 8.4 PIP joint swelling following injury

On examination, the PIP joint is normally swollen and may be deformed, particularly if there has been a dislocation or substantial damage to the bony anatomy (Fig. 8.4). On palpation there will be tenderness with a reduced range of motion with a particular reluctance to actively move the joint. There may be a volar laceration if there has been an open dislocation of the PIP joint (Fig. 8.5). This typically follows a fall where there is a hyperextension injury driving the middle phalanx dorsally where the head of the proximal phalanx tears through the volar skin typically to one side or other of the midline. Rarely there will be gross contamination of the PIP joint. Sensibility in the finger is normally a little reduced, but it would be rare for there to be significant nerve or arterial injury.

Fig. 8.5 Volar split laceration following an open PIP joint dislocation

Fig. 8.6 Radiograph of a concomitant DIP joint injury as well as a PIP joint injury

It is also important to seek out injuries elsewhere in the injured finger in particular at the distal interphalangeal joint or in other fingers (Fig. 8.6). These are easily overlooked when focusing upon the main injury.

Fig. 8.7 PA and oblique radiographs of the hand do not show the PIP joint subluxation well (**a**, **b**). It is very clear on a true lateral radiograph (**c**)

The primary investigations are plain radiographs. These should be centred on the injured joint and not simply be hand radiographs. Hand radiographs normally include a postero-anterior and oblique views. It is easy to overlook significant PIP joint injuries on these radiographs, in particular dorsal subluxation of the middle phalanx (Fig. 8.7). Occasionally plain radiographs will not be sufficient and a CT scan may be necessary, especially to assess complex partial condylar fractures of the head of the proximal phalanx.

The treatment of these injuries very much depends upon their severity. In most cases reassurance and early mobilisation will suffice.

Soft Tissue Injuries

Volar Plate and Collateral Ligament Injuries

These typically occur following hyperextension injuries (tearing the volar plate and volar part of the collateral ligaments) or lateral/oblique injuries primarily tearing one collateral ligament but also part of the volar plate. There may be small bony avulsions, although these are only indicators of injury and not of anatomical consequence (Fig. 8.8). Because the underlying bony anatomy is intact, early mobilisation is the best treatment [1–3]. Some support for comfort may be needed, but most patients do best with early restoration of function. Although there may have been a hyperextension injury these joints are rarely unstable in extension, except in patients with a tendency to some pre-existing volar ligament laxity, which is normally seen on assessing their other digits. In these cases prevention of hyperextension may be necessary, but most patients are best encouraged to regain full extension, which is much more easily lost then flexion. About 5 % of patients develop sufficient pain that they progress slowly. This is normally evident at around 6–8 weeks following injury. In these cases a steroid injection into the PIP joint can help overcome the pain and help with early mobilisation. Patients who do not have this

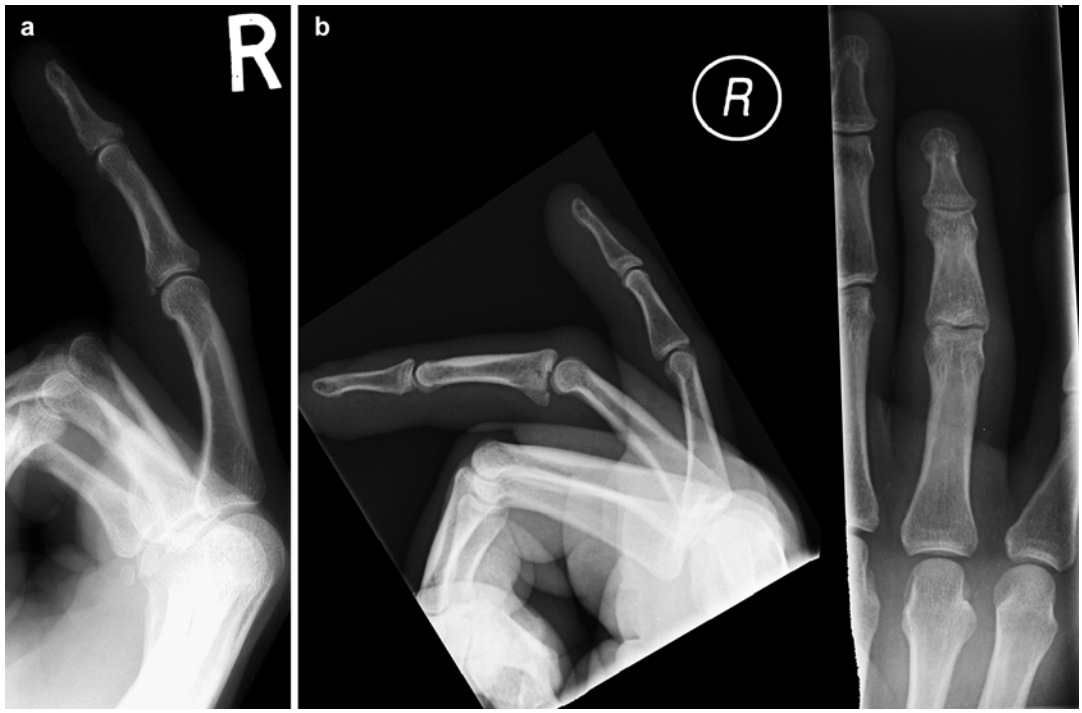


Fig. 8.8 Volar bony avulsion following a hyperextension (volar plate) injury (**a**, **b**)

injection will typically regain most of their pre-injury range of motion over a number of months. Even in patients who do well, there may be a little bit of discomfort in the PIP joint for 6–12 months.

Acute Boutonniere injuries are uncommon and sometimes difficult to diagnose. There will clearly have been a PIP joint injury with swelling and loss of flexion and some extension. It is easy to confuse these with the more common volar plate injuries. Patients may well be able to maintain some useful active PIP joint extension through the functioning lateral bands. The typical secondary deformity of DIP joint hyperextension does not normally occur immediately. Suspicions are raised by the mechanism of injury, (which is often a little unusual and not typically a hyperextension injury); tenderness, particularly dorsally at the base of the middle phalanx, some weakness of extension and possible radiographic changes dorsally at the base of the middle phalanx (Fig. 8.9). Patients with radiographic evidence of some bony abnormality often do very well with a shorter period of immobilisation of the PIP joint, typically for only two to three weeks. Where there is a suspicion or certainty of a Boutonniere injury then they are best treated with 6 weeks of splintage in full extension of the PIP joint, whilst encouraging DIP joint flexion and then gradually increasing mobilisation of the PIP joint with intermittent splintage over 2–3 weeks [4]. Most patients achieve a very good result, although there may be a mild extensor lag of perhaps 10–20°, although this is not normally of functional consequence. Patients who present a little later, often after a few weeks, with some fixed flexion deformity, are best treated with stretching of the PIP joint into full extension and once that is achieved then splinting as above [5]. Surgical treatment should almost always be avoided, as it is fraught with complications and unreliable outcomes.

Dislocation of the PIP joint is common, as noted above. Typically, following a closed reduction, the PIP joint will be perfectly congruent and stable (Fig. 8.10). This should be confirmed radiologically. If the PIP joint is not perfectly congruent on well centred radiographs, then it must be

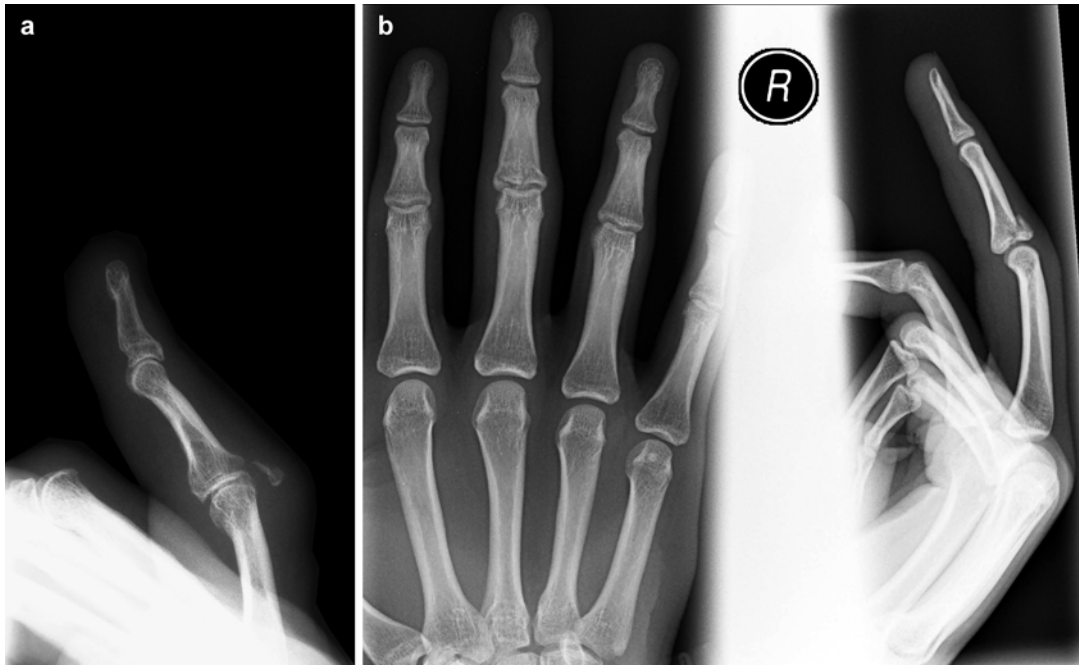


Fig. 8.9 Radiographs of bony boutonniere injuries (**a**, **b**)

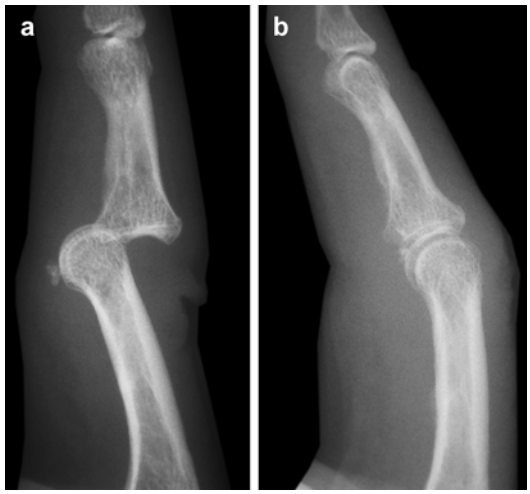


Fig. 8.10 PIP joint dislocation (**a**) with anatomical reduction (**b**)

assumed that there is some soft tissue interposed in the joint which, if left, will typically result in a poor outcome. Careful experienced assessment is needed. Further investigations are not typically of value. If there is soft tissue interposed between the proximal and middle phalanges it is typically one of the lateral bands leading to a rotational malalignment of the middle phalanx, relative to the proximal phalanx. This almost always requires open removal of the soft tissue after which the joint is typically stable and can be mobilised as for a typical PIP joint dislocation.

Open PIP joint dislocations, where the head of the proximal phalanx has torn through the volar skin, can be missed as the significance of the volar tear is overlooked. Although theoretically a severe injury with risk of infection, this injury will often have a very good outcome if treated with appropriate washout and early mobilisation. Many authors have suggested that the patient needs to be taken to the operating theatre as an emergency for washout of the joint [6]. This is a perfectly reasonable treatment plan, although washout under a good local anaesthetic ring block in the Emergency Department is almost always sufficient in a cooperative patient. They should also be given a short course of antibiotics to treat common bacteria such as staphylococci and streptococci. Obviously if there has been gross contamination such as a fall in a farm environment then formal

washout in the operating theatre may be appropriate. In the main the outcome is similar, although not always as good as closed dislocations, as this is typically a higher energy injury often in a slightly older age group.

Clinical Pearl
Injuries to the proximal interphalangeal joint are relatively common. Fortunately most are mild and resolve with conservative treatment. In the most severe cases the identification of significant structural damage, that is to the collateral ligaments or volar plate will require detailed and often repeated examination. Again, however, these can respond well to appropriate therapy.

Bone

The two sides of the joint need to be considered separately, the proximal side being convex and distal the concave side.

Fig. 8.11 Radiograph of a condylar fracture of the proximal phalanx

Proximal Phalanx

Extra-articular Fractures

Extra-articular fractures of the head of the proximal phalanx are typically at the neck i.e. just proximal to the condyles. This ususally follows a hyperextension injury with dorsal translation or tilting of the distal fragment (Fig. 8.11). If this is mildly displaced then a period of immobilisation in a plaster or splint for 3–4 weeks and then mobilisation as normal suffices. If more markedly displaced then this should be reduced [7, 8]. This can typically be performed under local anaesthetic in the Emergency Department or Outpatient Clinic. Following reduction this may be stable, in which case plaster/splintage for 3–4 weeks should suffice. If this is unstable then the reduction should be held with one or two oblique K-wires passed through the side of the head of the proximal phalanx into the distal shaft of the proximal phalanx. The wires are typically left outside the skin. As with almost all k-wiring, the construct should be supported in a splint or plaster for 3–4 weeks. The wires can be removed around 4½–5 weeks following injury and mobilisation encouraged. As with most PIP joint injuries of any significance, there is likely to be some long-term stiffness, particularly a lack of the last 20–30° of full extension. Flexion is generally very good.

Intra-articular Fractures

Intra-articular fractures of the head of the proximal phalanx are uncommon but well recognised. There is usually a unicondylar fracture i.e. one fracture is displaced off typically following some form of lateral tilting injury (Fig. 8.12). If the condyle is minimally displaced i.e. less than 1 mm, then immobilisation in a splint or plaster for 3–4

Fig. 8.12 Radiographs of a unicondylar fracture of the head of the proximal phalanx

weeks should suffice. As there is a risk of these injuries displacing within the first 2 weeks then these patients should be reviewed for at least 2 weeks with up-to-date radiographs, preferably out of the plaster or splint, as the judgement about displacement can be quite fine. If the condyle is displaced, typically either proximally or rotated forwards, then it needs to be reduced [9]. These are unstable injuries and almost always require some form of surgical stabilisation. This can be performed closed with k-wires, typically passing 1–3 K wires between one condyle and the other. Surgery can also be performed open with screw fixation allowing for early mobilisation. However, this is not always technically easy (see below).

Base of Middle Phalanx Injuries

Volar Plate Bony Avulsion From the Volar Base of the Middle Phalanx

This is essentially a volar plate injury and should be treated with early mobilisation as noted above.

Dorsal Fracture Subluxation

Dorsal fracture subluxation is essentially a volar plate injury with a large bony fragment that has been avulsed from the volar base of the middle phalanx leading to instability. Exactly how much of the volar base of the middle phalanx needs to be avulsed to lead to instability is not clear and there is no absolute cut-off. Assessment is normally made on a lateral radiograph of the PIP joint. If there is less than 30 % of the arc of the base of the middle phalanx avulsed then typically the joint will be stable. Between 30 and 40 % it may be stable and above 40 % typically the joint is unstable. The key to stability is looking for perfect congruence or otherwise of the base of the back of the middle phalanx relative to the proximal phalanx. If there is even slight incongruency there will be a triangle between the two bones (Fig. 8.13). If there is no incongruity then these injuries can be treated with early mobilisation as for volar plate injuries. If there is incongruence this needs to be reduced. The simplest way is to flex the PIP joint around 45°. Further lateral radiographs taken in this position may

Fig. 8.13 Radiograph of subtle dorsal subluxation of the middle phalanx

well show congruence. If so, the finger can be splinted, preventing extension beyond 45° but allowing flexion. Gradually over the following weeks the degree of extension block can be reduced by about 10° per week starting 2 weeks from injury until around 4 weeks from injury when the extension block can be removed. At each visit the congruence needs to be confirmed on a lateral radiograph. Thereafter the patient can mobilise and will typically gain a good result with a fixed flexion deformity of no more than 20° and good, if not full, flexion, with some mild aching with heavy use or in the cold. If the joint is not reduced satisfactorily without more flexion, then surgical stabilisation is warranted. Various options include a K-wire holding the joint reduced [10] a dynamic external fixator [11] or even open reduction and internal fixation [12], although the latter is fraught with complications and failures.

Pilon Fracture

A pilon fracture is typically the most severe injury to the base of the middle phalanx. It normally follows a high energy fall onto the end of the finger or high energy blow to the end of the finger, such as with the miss-catch of a hard ball. There is disruption of the base of the middle phalanx with a breach in the volar cortex and at least one of the lateral cortices. In addition, there is often dorsal cortex and central impaction of bone. In a small number of patients, perhaps 10 %, there will be good early mobilisation within a few days of injury (Fig. 8.14). These patients will typically do very well with careful supervised mobilisation, although they still require careful follow-up in the Outpatient Clinic for at least 2 weeks, with check radiographs to make sure that the joint is not collapsing further. The majority of patients, however, require surgery. As this is a longitudinally unstable injury simple K-wiring will not usually suffice. The best treatment is a dynamic external fixator [13]. Again some surgeons recommend open reduction and internal fixation, although this is technically challenging and fraught with complications [14]. With successful treatment, most patients can gain a functional range of motion from around 10° flexing to about 90°, but again with some aching with heavier use and in cold weather.

Surgical Techniques and Rehabilitation

Proximal Phalanx

Extra-articular Fracture

There are few indications for open reduction and internal fixation. Typically one or two oblique K-wires will suffice. Having reduced the fracture, it is usually reasonably stable. A K-wire can be inserted obliquely into the head of the proximal phalanx, just proximal to the articular surface. This passes through the collateral ligament, although this does not normally give any problems. Under careful image intensifier review the 1.1 (1.0–1.2) mm K-wire is advanced into the proximal phalanx. Ideally, the wire should purchase the cortex to the

Fig. 8.14 Radiographs of a pilon fracture with good early mobilisation

side of the proximal phalanx, but not penetrate. If the wire fully penetrates then this will potentially allow for wire migration, whereas if the wire just purchases with perhaps just the very tip of the wire going through the cortex, then the wire should not migrate distally. One K-wire may suffice (Fig. 8.15). A second K-wire is often difficult to place on the same side. Instead, a contralateral K-wire is needed if two are required. The problem with this is that it can lead to some malrotation of the distal fracture fragment, as one K-wire passes the other in the distal end of the proximal phalanx, applying a torque force. This should be obvious radiologically but also clinically as it should be possible to flex the finger down into the palm to assess for malrotation. As with almost all K-wires, this construct needs supporting in a plaster or splint for 3–4 weeks with the wires removed at around 4½–5 weeks.

Fig. 8.15 Radiographs of an extra-articular distal proximal phalanx fracture treated with crossed K wiring. (**a**) lateral, (**b**) anteroposterior

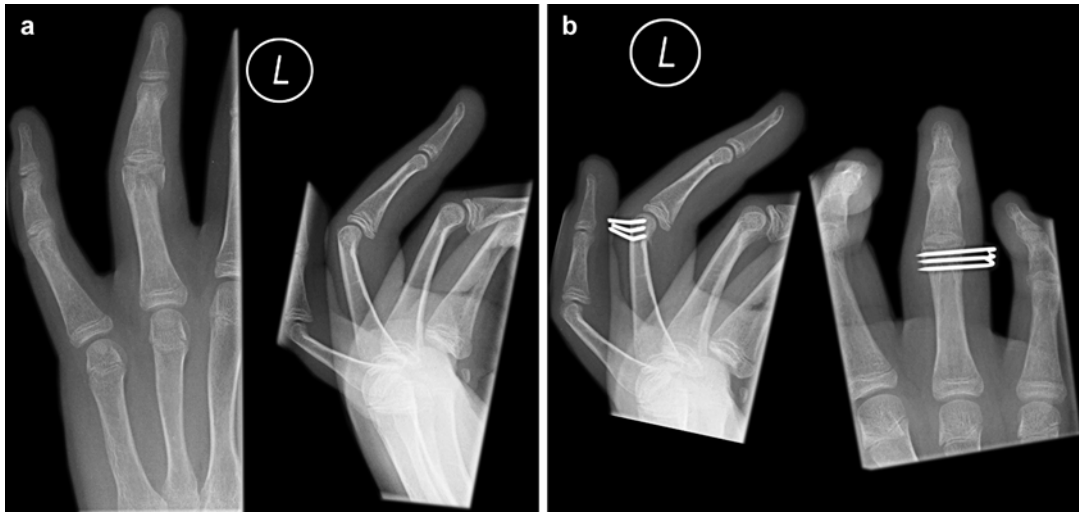


Fig. 8.16 Radiographs of closed latereal K wiring of a unicondylar fracture. (**a**) preop, (**b**) post.op

Intra-articular Fracture

These can be stabilised surgically with closed K-wiring or open screw fixation. Open K-wiring can be used, but this has the disadvantage of a further significant insult, without gaining stability allowing for early mobilisation.

Closed k-wiring: The condylar fracture may reduce with simple traction. It can be difficult to provide enough traction however by simply pulling on the finger and it is common for the surgeons fingers to be in the field of the radiographic beam. A simple technique to avoid this is to pass a K-wire (1.1 mm) transversally across the distal end of the middle phalanx. The wire is then bent at a right angle at both ends and traction applied using a surgical instrument. This can provide significant traction at the fracture site, which can be held by a surgical assistant. The condyle will normally reduce well. 1–3 transverse K-wires are then passed across the fracture, again through the non-articular side of the condyle through the collateral ligament out to the other side of the head of the proximal phalanx (Fig. 8.16). Again it is important to try to avoid the K-wire perforating the far cortex of the head of the proximal phalanx to reduce the risk of migration. If one or more K-wires have perforated, then having been bent over outside the skin these can be taped together with sterile surgical tape such as a steristrip to help keep the construct a little more stable. This again may reduce the risk of migration. Again, as with most K-wire constructs, this needs to be supported in plaster or a splint for 3–4 weeks. The wires need to be removed at 4½–5 weeks from injury unfortunately. It is sometimes not possible to get an absolutely perfect reduction, but provided the reduction is within about 1 mm then this is normally very well tolerated and does not merit open reduction.

Open reduction and internal fixation An alternative to closed reduction is to open the joint. This should allow for perfect alignment of the condyles. Although theoretically easy, in practice this is not always the case. It should be possible to pass a small screw transversely between the two condyles, typically a screw with a diameter of 1.2 mm. This has to be inserted very carefully, as it is easy to over tighten the screw and shatter the head of the proximal phalanx, or if there is any comminution press the two heads together, narrowing the joint, which would then lead to incongruency and stiffness. Sometimes it is possible to place two screws. The construct should be stable enough for early mobilisation. However, this is not always the case and a period of immobilisation for 1–2 weeks may be necessary, which unfortunately can compromise the outcome of

treatment. Again the likely outcome is a fixed flexion deformity of around 20–30° with flexion to over 90° and good function.

Base of Middle Phalanx

Fracture Subluxation

Manipulation and K wiring. The K-wire can be passed as a dorsal block directly into the head of the proximal phalanx, holding the middle phalanx in approximately 30° of flexion. This allows some early flexion of the joint and is a good, relatively straight forward technique. The risk is of introducing infection directly into the PIP joint. The middle phalanx is flexed out of the way and a 1.1 mm k-wire is passed centrally between the condyles longitudinally from the dorsum across to the volar cortex of the proximal phalanx, again engaging, but not perforating, the cortex. An alternative technique is to pass the K-wire into the dorsal base of the middle phalanx, securing its reduction to the proximal phalanx in about 10–20° to flexion. If there is a reasonable bulk of the base of the middle phalanx i.e. 50 % or more on the lateral radiograph then this is relatively straightforward. The wire passes from dorsal distally into the base of the middle phalanx, across the middle of the PIP joint, between the two condyles and into the volar cortex at the distal end of the proximal phalanx. If there is less than 50 % of the base of the middle phalanx then it is sometimes difficult to get adequate purchase on the bone, which may lead to an inadequate reduction. In these circumstances consideration should be given to an external fixator.

Dynamic external fixator. The principal of the dynamic external fixator is to provide a traction force across the PIP joint distracting it to largely, if not completely, realign the proximal and middle phalanges. By passing the proximal wire through the centre of rotation of the head of the proximal phalanx the joint can rotate about that wire whilst keeping the joint space distracted. This allows for early mobilisation and restoration of some articular or fibrous cartilage to the base of the middle phalanx. This is typically a very forgiving technique, which avoids opening up the joint with all its attendant complications. Under appropriate anaesthetic (typically LA) two K-wires are passed under an image intensifier control transversely, one across the distal end of the middle phalanx and one across the head of the proximal phalanx aiming for the centre of rotation or just proximally (Fig. 8.17). The wires are then linked together. There are various constructs, either bending the wires physically, holding the wires together with wire or using a rubber band construct (Figs. 8.18 and 8.19). The ultimate outcomes appear to be similar. There are pros and cons of each technique. Limited dressing needs to be applied and the patient carefully followed up in the outpatient Clinic, with up-to-date radiographs for at least two weeks, aiming to remove the k-wires at around 4½–5 weeks from injury and thereafter mobilising freely. Some patients find early mobilisation difficult and the insertion of local anaesthetic in the Outpatient

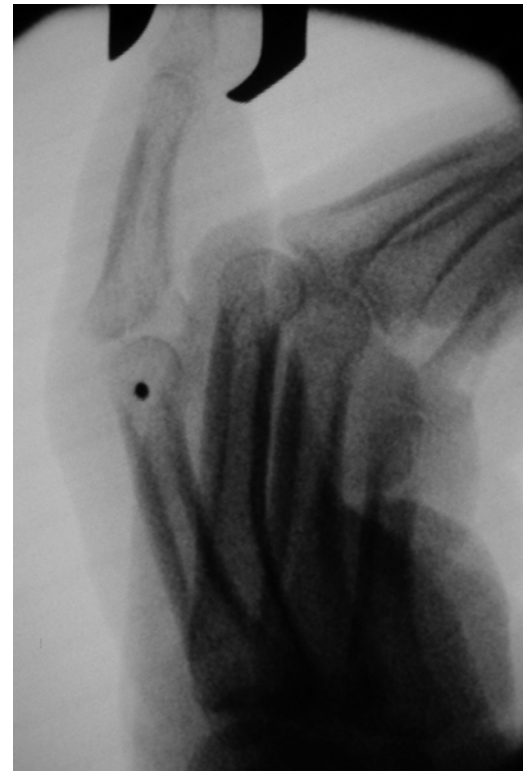


Fig. 8.17 Perioperative insertion of the proximal K wire of s dynamic external fixation. Note that the wire placed at the centre of rotation of the PIP joint

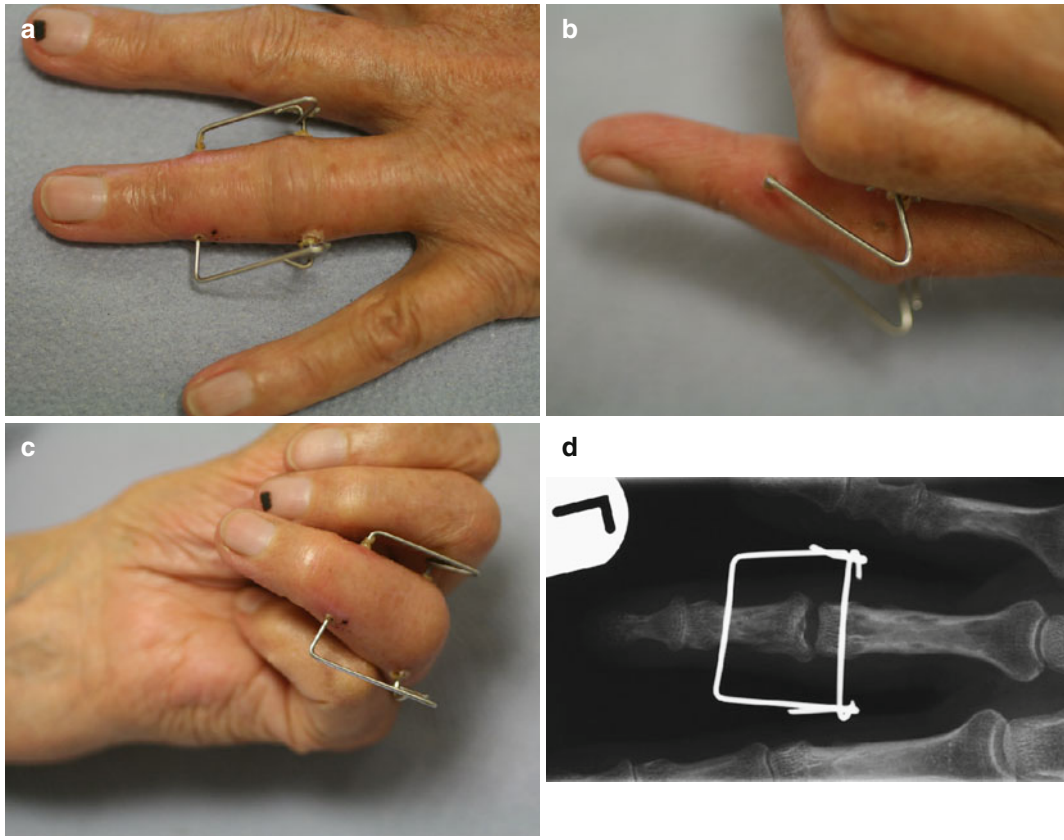


Fig. 8.18 Clinical photographs and a radiograph of a K wire dynamic external fixator. (**a**, **b**, **c**) Clinical images, (**d**) x-ray appearance

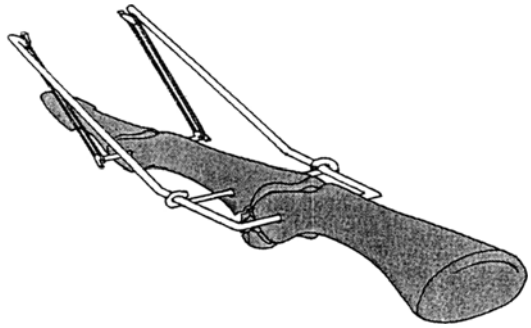


Fig. 8.19 Alternative design of dynamic external fixator; the Suzuki frame

Clinic and encouraging mobilisation on one or more occasions can substantially improve their outcome. Most patients will achieve a fixed flexion deformity of 10° or less with flexion to 90° or more with good DIP joint movement.

Open reduction and internal fixation. This is technically quite a difficult operation. It is best suited when there is one large volar fragment which can be linked to the main proximal phalanx fragment. The fracture may reduce relatively easily with traction and a single screw can be placed from the dorsal into the volar fragment. When this works this can give very good congruence and allow for early mobilisation with a good outcome. It is, however, technically demanding and it is easy to fragment the volar fragment, which may already be comminuted. In the main this should only be undertaken by very experienced surgeons.

Pilon Fracture

As noted above, a longitudinally unstable injury is not suited to simple K-wiring. It is an ideal situation for using a dynamic external fixator

with the technique as described above. Open reduction and internal fixation is even more difficult and should probably never be used, except again by very experienced surgeons.

Outcome Including Literature Review

The literature on the outcome of injuries around the PIP joint is best described as extensive, but superficial. Most of the studies report on a mix of injuries to the base of the middle phalanx. There are usually fewer than 10 cases with follow-up for around 1–2 years with variable outcomes. What can typically be established is the type of injury, the treatment given, the number of patients, the mean follow-up the length of treatment (particularly for application of K-wires or an external fixator), the average arc (and thus range) of motion and any complications. Less complete but frequently reported data include grip strength and pain.

This data is presented as a table below. It is important to recognise that these are typically from good units with an interest in treating these problems. Thus their results are likely to be better than those achieved in standard hand surgical practice. The results in standard practice are likely to be at least 25 % worse. In our experience the ROM will on average be only 75 % of that reported and the patients will have the next grade up of pain. In summary:

Head of Proximal Phalanx

(i) Extra-articular – most of these occur in children and one would expect an excellent recovery with minimal fixed flexion deformity, flexion >90° and little if any long-term pain.

(ii) Intra-articular – With a simple (non-comminuted) fracture of one condyle, one would expect very good recovery with a fixed flexion deformity up to 10–20°, flexion >90° and only mild if any long-term pain. If there is comminution then this is much harder to treat and a poor result is likely, not least as the initial reduction and fixation of the fracture is usually less optimal. Thus there may be more pain with aching in the cold and with heavy use and in less successful cases with a background continuous ache.

Base of Middle Phalanx

Soft Tissue Injuries

(i) Volar plate injury – typically there will be full recovery of function, but as noted above around 5 % of patients have persisting discomfort and stiffness, which typically resolves over 3–6 months, but may require a steroid injection into the joint to optimise the outcome. In a very few cases there will be a marked fixed flexion deformity, which will require a surgical release.

(ii) Open dislocation – this is primarily a soft tissue injury. Provided no infection occurs, then, as for the volar plate injuries, outcome should be very good if not a full recovery of function with around 5–10 % of patients having persisting discomfort and stiffness which typically resolves over 3–6 months. Again in a few cases there will be a marked fixed flexion deformity.

(iii) Boutonniere injury – The key to successful treatment is early diagnosis. Provided the injury is appreciated and treated within 2 weeks or so then there should be an excellent outcome, although some patients have a persisting mild extensor lag or fixed flexion deformity of 10–20° with full flexion and minimal if any discomfort. The longer treatment is delayed then the greater the likelihood of a more significant fixed flexion deformity. Specifically whilst the extensor mechanism is not working, the tendons are imbalanced and a fixed flexion deformity often develops at the PIP joint, which may be difficult to treat non-operatively or operatively.

Bone Injuries

(iv) Dorsal fracture subluxation – With a small volar fragment typically <25–30 % this should be a stable injury and so treated as for a volar plate injury, with comparable outcomes.

With a larger volar fragment, up to c. 50 % of the articular surface on the lateral radiograph, then the injury may be stable or if unstable is usually amenable to relatively simple treatment, such as immobilisation in flexion, single K-wiring or a dynamic external fixator. There should be a good to very good outcome, with only mild pain, a range of movement from 10° to at least 90°, although often not full movement. With increasing severity of injury comes an increased risk of complications and poorer outcomes. Around 5–10 % will have less good outcomes, with 1–2 % having marked stiffness and pain.

With a volar fragment >50 % then surgery is almost inevitable. Single K-wiring is usually unreliable, so dynamic fixation is usually required or ORIF, although this is technically demanding. With increasing severity comes an increased risk of a poorer outcome.

(v) Pilon fracture – Occasionally the fracture will be stable enough to allow early movement and non-operative treatment will result in a good to very good outcome, with little or no pain and an arc of movement of c.10–90°

Most however, need operative treatment. The best results seem to be with dynamic external fixation, which typically gives little or no pain and an arc of movement of c.10–90° but again an increasing risk of poorer outcomes in c. 10–15 %.

Clinical Pearl

Bony injuries to the proximal interphalangeal joint are generally easily identified by plain x-ray. Management and indeed outcome, however, depend predominantly on joint congruence. If this is maintained then a good outcome can be achieved. Surgery is confined to restoring articular congruence and ideally should only be undertaken by surgeons experienced with these techniques.

Prognosis

Typically symptoms will improve for up to 1–2 years from injury. Thereafter, they will largely remain static for the long term. With more severe injuries there will inevitably be some incongruence in the PIP joint and thus, in due course, radiological evidence of degenerative change. Because these joints are not weight bearing, unlike in the lower limb they seem to tolerate incongruence and degenerative changes rather better. Thus most patients do not become more symptomatic, other than experiencing a little more stiffness.

Complications

The main complication of non-operative treatment is pain, stiffness and reduced strength following malunion.

Following surgery there are the standard complications of infection, nerve injury, stiffness and CRPS. The particular complications are osteomyelitis from pin track infection or following ORIF and malunion with potentially very poor outcomes. Some patients will have marked problems requiring salvage surgery, including PIP joint release, which tends only to be of value if the joint is reasonably congruent; arthrodesis, arthroplasty and even amputation.

Conclusion

PIP joint injuries are common. They are usually relatively mild (volar plate injuries) with good outcomes following early supervised mobilisation. As with most joint injuries the key is joint congruency. If this is maintained

then a good outcome is usually achieved. If not, then treatment is needed to restore joint congruence. There are many techniques to achieve this, however, some form of K-wiring usually suffices. ORIF should only be undertaken by very experienced surgeons as it is technically demanding with typically poorer outcomes when compared to simpler techniques.

Fracture dislocation

Authors	Surgical procedure	Mean follow-up	Active/effective range of movement (arc)	Complications
Bain et al. [15]	ORIF or VPA and then monolateral ex-fix	222 days	65.6°	1 septic arthritis 2 pin track infection
Deshmukh et al. [16]	Pins and rubber traction system	34 months	85°	2 pin track infections
Badia et al. [17]	Dynamic ex-fix	24 months	84°	2 pin tract infections
Calfee et al. [18]	Hemi-hamate arthroplasty	4.5 years	70°	1 fracture 1 flexor pulley insufficiency 2 revisions
Deitch et al. [12]	Volar plate arthroplasty ORIF	46 months 46 months	72° 60°	6 dislocations 2 deep infections 2 capsulotomies and tenolysis
Ellis et al. [19]	Dynamic ex-fix	26 months	69° 88°	1 pin track infection
Hamilton et al. [20]	ORIF	42 months	70°	
Inanami et al. [21]	Dynamic ex fix	Not stated	95°	1 × recurrent subluxation
Morgan et al. [22]	Dynamic ex fix	24 months	89°	6 pin site infection 5 pin loosening
Rosenstadt et al. [23]	Closed reduction and percutaneous K-wire transfixing PIPJ ORIF	55 months	91° (results reported together)	Fracture displacement 2 × residual subluxation
Rutland et al. [11]	Dynamic ex fix	16 months	89°	8 pin track infections
Waris et al. [10]	Dorsal blocking wire	5 years	83°	None reported
Williams et al. [24]	Hemi-hamate autograft	16 months	85°	2 recurrent subluxation
Durhan-Smith et al. [25]	Volar plate arthroplasty	Minimum 6 months	95°	3 dorsal skin necrosis 1 pin track infection
Hamer et al. [26]	Extension block splint	20.6 months	87°	3 treatment failure requiring surgery
Newington et al. [27]	Dorsal blocking wire	16 years	85°	2 pin track infection
Aladin et al. [28]	Percutaneous K-wire (dorsal blocking wire) ORIF (lag screw) ORIF (circlage wire)	7 years	75° 73° 48°	 1 postop infection causing PIPJ fusion 2 secondary tenolysis
Grant et al. [29]	ORIF	39 months	94°	2 subluxations 1 dislocation needing further surgery
Lee et al. [30]	ORIF	8.7 months	85°	Additional external fixator required in one case
Houshian et al. [31]	Ex fix	20 months	79°	
Afendras et al. [32]	Hemi hamate osteochondral graft	60 months	67°	1 Flexor tenolysis

Fracture dislocation (continued)

Authors	Surgical procedure	Mean follow-up	Active/effective range of movement (arc)	Complications
Debus et al. [33]	Pins and rubbers	53	56.5°	3 pin track infections 4 additional surgical procedures 1 osteomyelitis
Keramidas et al. [34]	Suzuki dynamic ex Fix	18 months	91°	2 infections
Agawal et al. [35]	Suzuki dynamic ex fix	12.8 months	71°	2 pin site infection
Patel et al. [36]	Mini hand distractors	12 months	79°	1 pin site infection
Tekkis et al. [37]	ORIF	16–18 months	95°	Nil
De Smet et al. [38]	Suzuki dynamic ex fix	16.5 months	82°	2 pin tract infection
Duteille et al. [39]	Dynamic ex fix (pins and rubbers)	18 months	91.25°	1 did not tolerate it 1 pin track infection
Weiss [40]	ORIF – Circlage Wire	2.1 years	89°	Nil
Krakauer et al. [41]	ORIF then external device	11 months	73°	1 lost position 1 persistent subluxation 1 pin track infection
		14 months	56°	3 persistent subluxation 1 bone resorption 1 pin tract infection

Fracture

Authors	Implant	Mean follow-up	Active/effective range of movement (arc)	Complications
Bain et al. [15]	ORIF or VPA and then monolateral ex-fix	92 days	90°	1 pin track infection
Ruland et al. [11]	Dynamic ex fix	16 months (reported with fracture dislocations above)	87°	8 pin track infections Swan neck deformity
Sarris et al. [42]	ORIF and then dynamic traction splint	29 months	94°	1 pin site infection
Wolfe et al. [43]	ORIF	21 months	97°	Prominent K-wires Neurapraxia
Hynes et al. [13]	Dynamic ex fix	6–12 months	76°	2 pin site infection
Syed et al. [44]	Dynamic ex fix	2.2 years	79°	2 uncoupling of fixator
Keramidas et al. [34]	Suzuki dynamic ex fix	18 months	95.5°	2 infections
Agawal et al. [35]	Suzuki dynamic ex fix	12.8 months	66°	5 pin site infection 1 corrective osteotomy
Khan et al. [14]	ORIF	61 months	100°	Nil
Korting et al. [45]	Dynamic ex fix	10 months	56.5°	2 cases required additional fixation 1 Fracture displacement 1 PIPJ infection
Duteille et al. [39]	Dynamic Ex Fix (pins and rubbers)	18 months	105°	1 did not tolerate it 1 pin track infection
Lahav et al. [46]	Percutaneous K-wire fixation	36 months	96°	1 pin site infection
Thievendran et al. [47]	Dynamic ex fix	24 weeks	64°	2 pin track infection 1 needed reapplication

Dislocation

Authors	Implant	Mean follow-up	Active/effective range of movement (arc)	Complications
Bain et al. [15]	ORIF or VPA and then monolateral ex-fix	417 days	60.5°	1 × redislocation
Arora et al. [3]	Static splinting in flexion	Not reported	Not reported	
	Early active motion with dorsal block splinting	Not reported	Not reported	

Mixed patients

Authors	Patients	Implant	Mean follow-up	Active/effective range of movement (arc)	Complications
Majumder et al. [48]	Fracture +/– dislocation	Dynamic ex fix	20 months	74°	3 pin site infection 1 osteomyelitis
Suzuki et al. [49]	Fracture +/– dislocation also involving other joints	Pins and rubbers	13.1 months	80°	
De Soras et al. [50]	Proximal and middle phalanx fractures around the PIPJ	Dynamic ex fix (pins and rubbers)	9.7 months	Reported as 84 % of the normal joint	5 × pin site inflammation 1 × osteitis
Finsen [51]	Fractures and fracture dislocations	Suzuki ex fix	49 months	72°	3 pin site infections 2 slipped wires

References

1. Gaine WJ, Beardsmore J, Fahmy N. Early active mobilisation of volar plate avulsion fractures. Injury. 1998;29(8):589–91.
2. Phair IC, Quinton DN, Allen MJ. The conservative management of volar avulsion fractures of the PIP joint. J Hand Surg (Br Vol). 1989;14B:168–70.
3. Arora R, Lutz M, Fritz D, Zimmerman R, et al. Dorsolateral dislocation of the proximal interphalangeal joint: closed reduction and early active motion or splinting or static splinting; a retrospective study. Arch Orthop Trauma Surg. 2004;124(7):486–8.
4. Souter WA. The boutonniere deformity: a review of 101 patients with division of the central slip of the extensor expansion in the fingers. J Bone Joint Surg (Br Vol). 1967;49B(4):710–21.
5. To P, Watson JT. Boutonniere deformity. J Hand Surg (Am Vol). 2011;36A(1):139–42.
6. Henry MH. Management of hand injuries. In: Bucholz RW, Heckman JD, Court-Brown CM, editors. Rockwood and Green's fractures in adults, 6th ed. Lippincott Williams and Wilkins: Philadelphia; 2005. p. 802.
7. Singh J, Jain K, Mruthyunjaya, Ravishankar R. Outcome of closed proximal phalangeal fractures of the hand. Indian J Orthop. 2011;45(5):432–8.
8. Burkhalter WE. Closed treatment of hand fractures. J Hand Surg (Am Vol). 1989;14A(2):390–3.
9. Weiss APC, Hastings H. Distal unicondylar fractures of the proximal phalanx. J Hand Surg (Am Vol). 1993;18A(4):594–99.
10. Waris E, Alanen V. Percutaneous, intramedullary fracture reduction and extension block pinning for dorsal proximal interphalangeal fracture-dislocations. J Hand Surg (Am Vol). 2010;35A(12):2046–52.
11. Ruland RT, Hogan CJ, Cannon DL, Slade JF. Use of dynamic distraction external fixation for unstable fracture-dislocations of the proximal interphalangeal joint. J Hand Surg (Am Vol). 2008;33A(1):19–25.
12. Deitch MA, Kiefhaber TR, Comisar BR, Stern PJ. Dorsal fracture dislocations of the proximal interphalangeal joint: surgical complications and long-term results. J Hand Surg (Am Vol). 1999;24A(5):914–23.
13. Hynes MC, Giddins GE. Dynamic external fixation for pilon fractures of the interphalangeal joints. J Hand Surg (Br Vol). 2001;26B(2):122–4.
14. Khan W, Agarwal M, Muir L. Management of intra-articular fractures of the proximal interphalangeal joint by internal fixation and bone grafting. Arch Orthop Trauma Surg. 2004;124(10):688–91.
15. Bain GI, Mehta JA, Heptinstall RJ, Bria M. Dynamic external fixation for injuries of the proximal interphalangeal joint. J Bone Joint Surg (Br Vol). 1998;80B(6):1014–9.

16. Deshmukh SC, Kumar D, Mathur K, Thomas B. Complex fracture-dislocation of the proximal interphalangeal joint of the hand. Results of a modified pins and rubbers traction system. J Bone Joint Surg (Br Vol). 2004;86B(3):406–12.
17. Badia A, Riano F, Ravikoff J, Khouri R, Gonzalez-Hernandez E, Orbay JL. Dynamic intradigital external fixation for proximal interphalangeal joint fracture dislocations. J Hand Surg (Am Vol). 2005;30A(1): 154–60.
18. Calfee RP, Kiefhaber TR, Sommerkamp TG, Stern PJ. Hemi-hamate arthroplasty provides functional reconstruction of acute and chronic proximal interphalangeal fracture-dislocations. J Hand Surg (Am Vol). 2009;34A(7):1232–41.
19. Ellis SJ, Cheng R, Prokopis P, Chetboun A, Wolfe SW, Athanasian EA, Weiland AJ. Treatment of proximal interphalangeal dorsal fracture-dislocation injuries with dynamic external fixation: a pins and rubber band system. J Hand Surg (Am Vol). 2007;32A(8):1242–50.
20. Hamilton SC, Stern PJ, Fassler PR, Kiefhaber TR. Mini-screw fixation for the treatment of proximal interphalangeal joint dorsal fracture-dislocations. J Hand Surg (Am Vol). 2006;31A(8):1349–54.
21. Inanami H, Ninomiya S, Okutsu I, Tarui T. Dynamic external finger fixator for fracture dislocation of the proximal interphalangeal joint. J Hand Surg (Am Vol). 1993;18A(1):160–4.
22. Morgan JP, Gordon DA, Klug MS, Perry PE, Barre PS. Dynamic digital traction for unstable comminuted intra-articular fracture-dislocations of the proximal interphalangeal joint. J Hand Surg (Am Vol). 1995;20A(4): 565–73.
23. Rosenstadt BE, Glickel SZ, Lane LB, Kaplan SJ. Palmar fracture dislocation of the proximal interphalangeal joint. J Hand Surg (Am Vol). 1998;23A(5): 811–20.
24. Williams RM, Kiefhaber TR, Sommerkamp TG, Stern PJ. Treatment of unstable dorsal proximal interphalangeal fracture/dislocations using a hemi-hamate autograft. J Hand Surg (Am Vol). 2003;28A(5):856–65.
25. Durham-Smith G, McCarten GM. Volar plate arthroplasty for closed proximal interphalangeal joint injuries. J Hand Surg (Br Vol). 1992;17B(4):422–8.
26. Hamer DW, Quinton DN. Dorsal fracture subluxation of the proximal interphalangeal joints treated by extension block splintage. J Hand Surg (Br). 1992; 17B(5):586–90.
27. Newington DP, Davis TR, Barton NJ. The treatment of dorsal fracture-dislocation of the proximal interphalangeal joint by closed reduction and Kirschner wire fixation: a 16-year follow up. J Hand Surg (Br Vol). 2001;26B(6):537–40.
28. Aladin A, Davis TR. Dorsal fracture-dislocation of the proximal interphalangeal joint: a comparative study of percutaneous Kirschner wire fixation versus open reduction and internal fixation. J Hand Surg (Br Vol). 2005;30B(2):120–8.
29. Grant I, Berger AC, Tham SK. Internal fixation of unstable fracture dislocations of the proximal interphalangeal joint. J Hand Surg (Br Vol). 2005; 30B(5):492–8.
30. Lee JY, Teoh LC. Dorsal fracture dislocations of the proximal interphalangeal joint treated by open reduction and interfragmentary screw fixation: indications, approaches and results. J Hand Surg (Br Vol). 2006;31B(2):138–46.
31. Houshian S, Ghani A, Chikkamuniyappa C, Sakka SA. Single-stage distraction correction for neglected dorsal fracture dislocations of the proximal interphalangeal joint: a report of eight cases. J Hand Surg Eur Vol. 2008;33E(3):345–9.
32. Afendras G, Abramo A, Mrkonjic A, Geijer M, Kopylov P, Tägil M. Hemi-hamate osteochondral transplantation in proximal interphalangeal dorsal fracture dislocations: a minimum 4 year follow-up in eight patients. J Hand Surg Eur Vol. 2010;35E(8): 627–31.
33. Debus G, Courvoisier A, Wimsey S, Pradel P, Moutet F. Pins and rubber traction system for intra-articular proximal interphalangeal joint fractures revisited. J Hand Surg Eur Vol. 2010;35E(5):396–401.
34. Keramidas E, Solomos M, Page RE, Miller G. The Suzuki frame for complex intra-articular fractures of the proximal interphalangeal joint of the fingers. Ann Plast Surg. 2007;58(5):484–8.
35. Agarwal AK, Karri V, Pickford MA. Avoiding pitfalls of the pins and rubbers traction technique for fractures of the proximal interphalangeal joint. Ann Plast Surg. 2007;58(5):489–95.
36. Patel MR, Joshi BB. Distraction method for chronic dorsal fracture dislocation of the proximal interphalangeal joint. Hand Clin. 1994;10(2):327–37.
37. Tekkis PP, Kessaris N, Gavalas M, Mani GV. The role of mini-fragment screw fixation in volar dislocations of the proximal interphalangeal joint. Arch Orthop Trauma Surg. 2001;121(1–2):121–2.
38. De Smet L, Boone P. Treatment of fracture-dislocation of the proximal interphalangeal joint using the Suzuki external fixator. J Orthop Trauma. 2002;16(9): 668–71.
39. Duteille F, Pasquier P, Lim A, Dautel G. Treatment of complex interphalangeal joint fractures with dynamic external traction: a series of 20 cases. Plast Reconstr Surg. 2003;111(5):1623–9.
40. Weiss AP. Cerclage fixation for fracture dislocation of the proximal interphalangeal joint. Clin Orthop Relat Res. 1996;327:21–8.
41. Krakauer JD, Stern PJ. Hinged device for fractures involving the proximal interphalangeal joint. Clin Orthop Relat Res. 1996;327:29–37.
42. Sarris I, Goitz RJ, Sotereanos DG. Dynamic traction and minimal internal fixation for thumb and digital pilon fractures. J Hand Surg (Am Vol). 2004;29A(1): 39–43.
43. Wolfe SW, Katz LD. Intra-articular impaction fractures of the phalanges. J Hand Surg (Am Vol). 1995; 20A(2):327–33.
44. Syed AA, Agarwal M, Boome R. Dynamic external fixator for pilon fractures of the proximal

interphalangeal joints: a simple fixator for a complex fracture. J Hand Surg (Br Vol). 2003;28B(2):137–41.
45. Körting O, Facca S, Diaconu M, Liverneaux P. Treatment of complex proximal interphalangeal joint fractures using a new dynamic external fixator: 15 cases. Chir Main. 2009;28(3):153–7.
46. Lahav A, Teplitz GA, McCormack Jr RR. Percutaneous reduction and Kirschner-wire fixation of impacted intra-articular fractures and volar lip fractures of the proximal interphalangeal joint. Am J Orthop. 2005;34(2):62–5.
47. Theivendran K, Pollock J, Rajaratnam V. Proximal interphalangeal joint fractures of the hand: treatment with an external dynamic traction device. Ann Plast Surg. 2007;58(6):625–9.
48. Majumder S, Peck F, Watson JS, Lees VC. Lessons learned from the management of complex intra-articular fractures at the base of the middle phalanges of fingers. J Hand Surg (Br Vol). 2003;28B(6):559–65.
49. Suzuki Y, Matsunaga T, Sato S, Yokoi T. The pins and rubbers traction system for treatment of comminuted intraarticular fractures and fracture-dislocations in the hand. J Hand Surg (Br Vol). 1994;19B(1):98–107.
50. De Soras X, de Mourges P, Guinard D, Moutet F. Pins and rubbers traction system. J Hand Surg (Br Vol). 1997;22B(6):730–5.
51. Finsen V. Suzuki's pins and rubber traction for fractures of the base of the middle phalanx. J Plast Surg Hand Surg. 2010;44(4–5):209–13.

Fractures of the Proximal Phalanx and Those Involving the Metacarpo-Phalangeal Joint

9

David J. Shewring

Keywords
Fracture • Phalanx • Avulsion • Condyle • K-wire • Lag screw • Nonunion • Malunion • Osteotomy • Internal fixation

Introduction

Fractures of the proximal phalanx are amongst the most common affecting the hand. In a large study by Stanton et al. [1], the proximal phalanx accounted for 17 % of 691 hand fractures studied. Although many of these fractures can be treated non-operatively [2], they can be unforgiving, particularly those affecting the phalangeal shaft [3].

Treated poorly, with a lack of respect and if the wrong tools are used in the wrong way and in the wrong hands, then the results may be catastrophic and irretrievable (Fig. 9.1).

Good or perfect results can be obtained, but these may be hard won, entailing careful planning, with consideration of all the various methods in the surgical armamentarium.

As the best and often only chance of obtaining a good result is at the first surgical intervention, these fractures should not be delegated to unsupervised more junior members of the surgical team. There is also no place for the surgical treatment of these fractures by the occasional hand surgeon. It is better to delay fixation by a few days until the best expertise is available.

At the cornerstone of treatment should be the services of skilled specialised hand therapists. For most proximal phalangeal fractures their involvement should commence at the earliest opportunity. During conservative treatment, they can, if necessary, splint injured digits leaving non-injured ones free. They can give patients advice on mobilisation and correct care of the injured digit and their involvement will free up time for clinicians in busy clinics. Ideally, the therapists should be present in the trauma clinic so that plans can be formulated jointly and treatment plans discussed with surgeon, therapist and patient present. If complex fixations are being attempted, then post-operative supervision by a specialised hand therapist is absolutely essential. If the therapists are encouraged to check matters at crucial junctures and bring patients back if they are concerned then this will allow early discharge to their care.

D.J. Shewring, MB BCh, FRCS(Orth), Dip Hand Surg(Eur)
Department of Trauma and Orthopaedics,
University Hospital of Wales,
Heath Park, Cardiff CF14 4XW, UK
e-mail: davidshewring@me.com

I.A. Trail, A.N.M. Fleming (eds.), *Disorders of the Hand: Volume 1: Hand Injuries*,
DOI 10.1007/978-1-4471-6554-5_9, © Springer-Verlag London 2015

Fig. 9.1 An irretrievable result from a poorly fixed fracture. Several attempts have been made to fix this fracture with an inappropriate implant. There is a snapped off drill bit. The result is an infected non–union

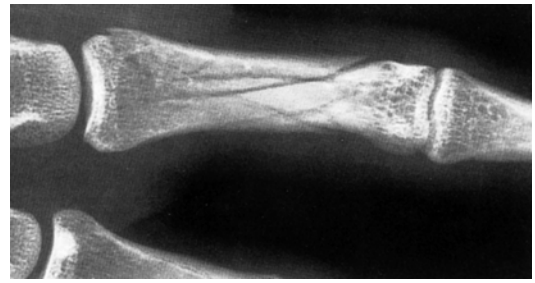


Fig. 9.2 A crush injury may result in a comminuted but relatively stable fracture as the periosteum is intact

Fig. 9.3 Transverse fractures will result from a direct blow. These fractures are unstable and may be open

Assessment

The **background** to the injury is important and numerous factors will influence how the patient is treated. The age, occupation, demands, priorities, co-morbidities and potential compliance of the patient must be established. Many of these patients have a tendency towards irresponsibility, which must be taken into account.

It is important to establish the exact amount of time that has elapsed between the date of the injury and the first consultation with the clinician who is going to provide the definitive treatment. There will often have been delays, due to late presentation by the patient or tardy referral by the accident service.

The **mechanism** of the injury will dictate the configuration of the fracture and will often have a bearing on how the fracture is treated.

A crush injury (Fig. 9.2) may result in a comminuted but un-displaced fracture.

The periosteum is likely to be intact and, despite the comminution, the fracture may therefore be relatively stable. This will allow early mobilisation after initial symptomatic treatment. Crush injuries may, however, be associated with more severe soft tissue damage and a higher incidence of Chronic Regional Pain Syndrome and this will also influence management.

A direct blow to a finger may result in a transverse fracture (Fig. 9.3). The degree of displacement will reflect the energy of the injury. Such fractures tend to be unstable and management of these is particularly challenging.

In the case of a spiral fracture (Fig. 9.4), the trauma will have been indirect. The shape of the fracture echoes the rotational nature of the force involved. These fractures often occur as the result of a fall, assaults, injury during contact sport or as the result of the finger having

Fig. 9.4 The configuration of a spiral fractures echoes the nature of the force applied

become entangled in a dog lead or similar. Displaced spiral fractures tend to be unstable, due to tearing of the periostium and the configuration of the fracture.

Although a spiral fracture may appear undisplaced, this is relative as the very fact that the fracture is visible on a radiograph will indicate some displacement.

The finger will need to be inspected carefully for signs of mal-rotation. This can be done by either inspecting the relative orientation of the fingernails, or by gently flexing the digits, which will make any malrotation more obvious. If malrotation is present it must be corrected or significant morbidity will result, as well as potential litigation.

It is essential that all strapping is removed **before** the finger is examined or x-rayed. Failure to do so will not permit adequate examination and radiographs taken with strapping in place will also be inadequate.

Initial radiographs taken in the accident unit are often inappropriate. All too often a request for "X-ray hand" has been made and the resulting images include the whole hand rather than specific views of the relevant digit or joint.

It is important that a specific request is made and that AP and true lateral views of the relevant digit are obtained.

The advent of computerised radiology with the ability to expand, rotate and change the contrast of images as well as accurate measurement of angles, has made planning the treatment of small bone fractures significantly easier.

Pearls

Most fractures can be treated non-operatively.

The best and often only chance of obtaining a good result is at the first surgical intervention.

It may be better to delay fixation by a few days until the best expertise is available.

A cornerstone of treatment should be the services of skilled specialised hand therapists.

It is important to establish the age of the fracture, mechanism of injury and the nature of the patient.

Ensure adequate radiographs have been taken.

Diaphyseal Fractures

Undisplaced

For many undisplaced diaphyseal fractures, simple "buddy-taping" to the adjacent digit may suffice. Fractures may, however, have an unstable configuration. If considered vulnerable to displacement and particularly if the patient has a tendency towards irresponsibility, it may be prudent to augment this with a protective splint, at least for the initial 1–2 weeks. The digit should be splinted in the "intrinsic-plus" position with the MCP joint flexed and the IP joints in extension [4]. A custom-made, thermoplastic splint can be formulated by the hand therapists for this (Fig. 9.5). When in a safe environment and at night, the splint should be removed and the

Fig. 9.5 A custom made thermoplastic splint can be made by the hand therapists to protect the fractured digit

finger allowed to mobilise. During conservative treatment, care has to be taken not to "over-splint", as stiffness will readily ensue.

Displaced

If **fixation** is indicated, then there is a spectrum of techniques which are available and which can be tailored to each fracture.

At one extreme would be a strong, robust fixation, such as a plate (Fig. 9.6).

Although this would have the advantage of more stability and reliability, the increased trauma to the digit involved in its application could potentially compromise the result.

At the other end of the spectrum would be a minimal fixation such as a single wire, inserted closed (Fig. 9.7). Although this would have the advantage of having inflicted minimal added trauma to the digit, the fixation may not be robust enough to allow vigorous mobilisation.

A study by Horton et al. [3] showed that there was no significant difference in the functional recovery rates or in the pain scores for two groups of patients with spiral proximal phalangeal fractures treated either with closed K-wire fixation or with open reduction and fixation with lag screws. X-rays showed similar rates of mal-union and there were no statistically significant differences in range of movement or grip strength. They made the point that surgeons treating these fractures should favour the method with which they are most familiar and competent. Consideration should also be given as to which technique utilizes the least health care resources.

Fig. 9.6 Plate fixation of the proximal phalanx

Ideally we want to achieve a **precise restoration of the anatomy** with a stable and strong fixation that allows immediate mobilisation, so that the fracture can be effectively ignored and the soft tissues rehabilitated.

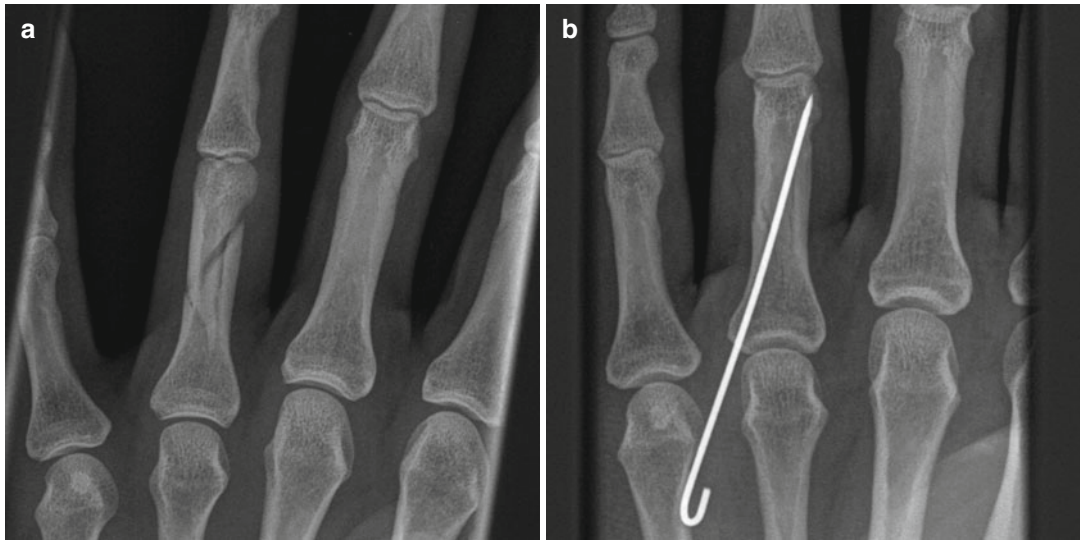


Fig. 9.7 (**a**, **b**) Closed K-wire fixation will maintain the integrity of the soft tissue envelope

Pearls

During conservative treatment, care has to be taken not to "over-splint", as stiffness will readily ensue.

If **fixation** is indicated, then there is a spectrum of techniques which are available and which can be tailored to each fracture.

Treating surgeons should favour the method with which they are most familiar and competent.

K-wires

K-wires do not provide this stability of fixation – but they can be used as what are effectively **bone sutures.** They will provide augmentation of conservative treatment, making it more reliable.

They can be used to maintain length and rotation to enable effective splintage and allow some movement [5–7].

K-wires have some disadvantages. They may protrude – causing soft tissue interference. The pin site is vulnerable to infection and will need regular cleaning.

They do not provide a stable enough fixation for vigorous mobilisation and they generally need to be removed, although this can be done in clinic if they are left protruding through the skin.

But, they are cheap and can be inserted quickly. When inserted closed the integrity of the soft tissue envelope can be preserved.

If, however, the fracture is associated with significant soft tissue damage, as in the case of a crush injury, then the option to provide stability without the further soft tissue injury consequent to dissection is invaluable (Fig. 9.8).

Ideally, protrusion of the wires through the distal end of the phalanx should be avoided (Fig. 9.9). The PIP joint is intolerant of injury and any interference or transfixion of the collateral ligaments of that joint invites trouble.

It is preferable and easier to insert the wires from the base of the phalanx.

The wire can be guided between the metacarpal heads.

The rim of the base of the phalanx can be felt with the wire and the wire inserted up the medullary canal, either up to the subchondral bone or to engage the lateral cortex (Fig. 9.7).

Alternatively, the wire can be inserted into the distal end of the phalanx and then exit the

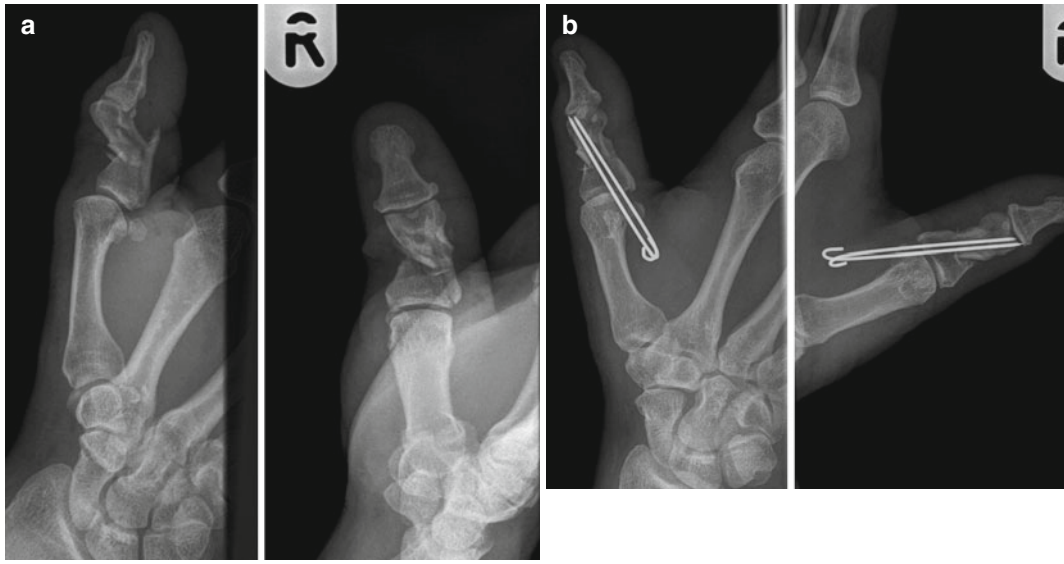


Fig. 9.8 (**a**, **b**) In the presence of severe soft tissue injury, K-wires will provide stability without the need for further dissection

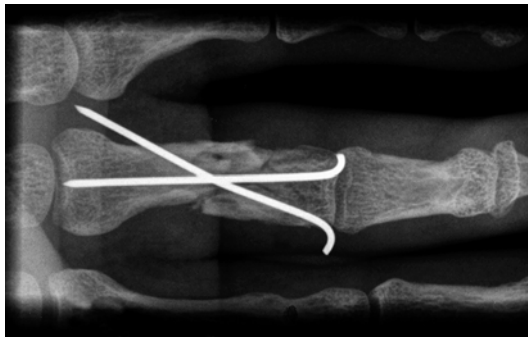


Fig. 9.9 Poor use of K-wires. The wires cross at the fracture site predisposing to non-union. The wires protrude at the PIP joint, transfixing the collateral ligaments and predisposing to stiffness

shaft cortex. The wire can then be withdrawn to leave the trailing end in the distal segment, so as not to interfere with the collateral ligament (Fig. 9.10). A single ended wire is best for this.

It is not necessary to use a wire bigger than 1.2 mm for phalangeal fractures.

The wires usually remain in situ for approximately 3 ½ weeks, although further protection with "buddy-taping" may be necessary for several further weeks.

Fig. 9.10 A single ended wire can be left trailing in the fragment, providing stability but without transfixion of the collateral ligament

Fig. 9.11 (**a**, **b**) The combination of a precise reduction of the inter-digitations of a fresh fracture and compression with lag screws will provide a very stable fixation of a spiral fracture permitting immediate mobilisation

Lag Screws

Over the last 20 years there has been a considerable improvement in the technology of implants used for internal fixation in the hand. Screws are now smaller and self-tapping. The screw heads are lower profile and the instrumentation for insertion has become more refined [8, 9]. As a result of this, as well as there being more widespread expertise in the operative management of hand fractures, the scope for internal fixation has increased.

Metalwork should ideally be kept to a minimum, so that there is minimal interference with the soft tissues and for a displaced spiral fracture with no or minimal comminution, fixation with one or two lag screws is ideal (Fig. 9.11a, b).

However, the reduction must be absolutely perfect and accurate. If this is not achieved then the stability will not be enough for mobilisation and the patient may be left with the worst of all options.

Plates

The use of plates is indicated only occasionally for proximal phalangeal fractures. Although the implants have become lower profile in recent years, they are still relatively bulky and it may be difficult to close the periosteum over the implant. Application of a plate necessitates significant soft tissue stripping, which is tolerated less over the phalanges than in the metacarpal region. As the phalanx has an elliptical shape in cross section they are more easily applied to the dorsal aspect of the bone through a dorsal approach. The "mini condylar plate" is a particularly unforgiving implant.

Pearls

K wires can be used as bone sutures.

They provide augmentation of conservative treatment, making it more reliable.

The PIP joint is intolerant; interference or transfixion of the collateral ligaments of that joint invites trouble.

It is not necessary to use a wire bigger than 1.2 mm for phalangeal fractures.

Metalwork should ideally be kept to a minimum

When using lag screws the reduction must be perfect and accurate

Use of plates is indicated only occasionally for proximal phalangeal fractures

Specific Fractures

Spiral Fractures

Long spiral fractures of the phalanges are common. For minimally displaced fractures that reduce easily, an intra-medullary K-wire inserted through the base of the phalanx and engaging the lateral cortex may suffice (Fig. 9.7a, b). Although gentle mobilisation will be possible, this will need to be augmented by splintage. Alternatively, the combination of a precise reduction of the inter-digitations of a fresh fracture and compression with lag screws will provide a very stable fixation and will permit immediate mobilisation.

Any collateral damage to the soft tissues from surgery should be minimised – so the surgical approach for internal fixation needs to be thought about carefully.

Approaches

A **lateral** approach for this type of fracture (Fig. 9.12a–e) is more difficult than a dorsal approach. The lateral approach does, however, avoid violation of the plane between the extensor mechanism and the periosteum which is a potent cause of adherence and stiffness. For uncomplicated spiral factures, this is therefore recommended.

When deciding whether to select a radial or an ulnar approach to a fracture, there are various factors to consider. An ulnar approach will necessitate an extra assistant to keep the arm rotated. Access to the site of most difficult reduction will be required, usually where there is most likely to be soft tissue interposition. This usually occurs distally, if the fracture line involves the area of attachment of the collateral ligament.

The extensor and underlying periosteum are identified and any dorsal branches of the digital nerve protected. The periosteum is incised and elevated with a Mitchel's trimmer.

Taking care to avoid the plane between periosteum and extensor, the phalanx is exposed.

The collateral ligament is identified and carefully preserved and the fracture is opened up, to clean it of fibrinous material, which would prevent reduction

The fracture is reduced and held. The fracture can be held reduced with a reduction forceps or an artery clip prior to insertion of the first screw. It is worth taking extra time to make sure that it is as perfect as possible. Two lag screw are usually adequate. The drill reamings can be collected to use as graft which can be smeared into the fracture site. This is particularly useful if there has been some slight comminution of the fracture edges.

The periostium and skin are closed and the rotation checked.

The finger can then be mobilised early. It is adequate to dress the finger and bandage it to an adjacent finger, without plaster.

Transverse Fractures

Transverse fractures of the mid shaft are relatively uncommon, but pose their own particular challenges. This configuration of fracture is often caused by a direct blow to the finger and so these fractures are often open. They may also be comminuted. The surgical approach, if necessary, may be determined by the position of the open wound, which is often dorsal.

Either an intra-osseous "Lister" wire loop or a plate may be necessary. Both provide a robust

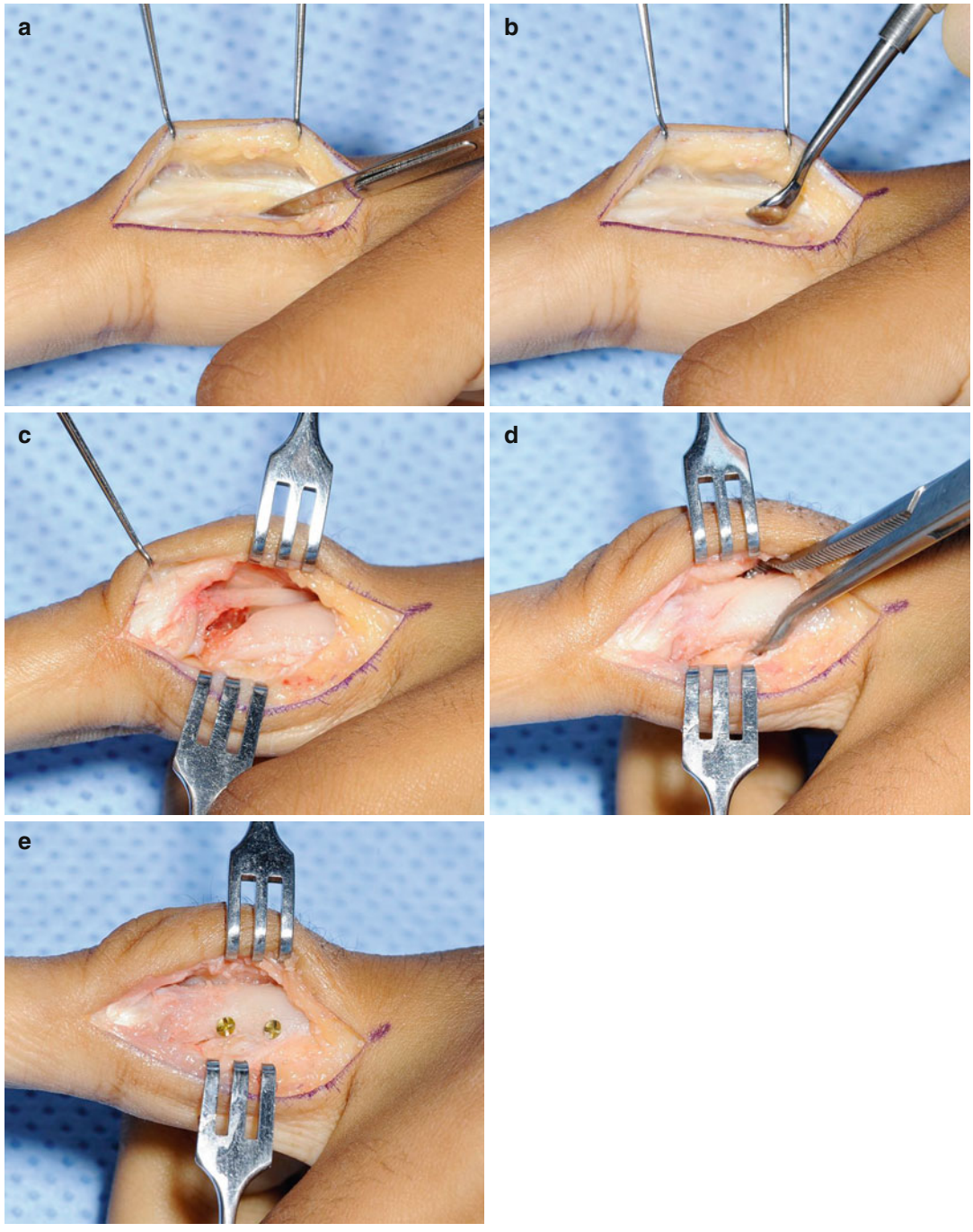


Fig. 9.12 A lateral approach to the spiral fracture of the proximal phalanx. The periosteum is incised (**a**) and elevated (**b**). The fracture is opened (**c**), curetted and reduced (**d**). Fixation with two lag screws (**e**)

fixation. The Lister loop technique [10] is likely to involve more extensive dissection and soft tissue stripping and is technically more exacting. A plate will require a dorsal approach and acts as a "tension band" type device (Fig. 9.6). The dorsal approach will allow better access if the

Fig. 9.13 Fracture through the metaphyseal region at the base of the phalanx

fracture is comminuted. For either technique the surgeon, therapist and patient should be prepared for a period of stiffness, with extension lag of the proximal inter-phalangeal joint, which although usually temporary, may be protracted.

Metaphyseal Fractures

Fractures at the base of the proximal phalanx, through metaphyseal bone, are common, particularly in the middle aged and elderly (Fig. 9.13). They usually result from a simple fall and due to their configuration present their own specific problems.

Due to the pull of the intrinsic muscles, these fractures will tend to angulate with the distal segment being pulled into dorsiflexion. The angulation can be readily corrected, but conservative treatment is generally unsuccessful, as the pull of the intrinsics tends to cause re-angulation of the fracture. If the fracture is allowed to heal in such a position, then the normal action of the intrinsics will be altered.

Normally, the pull of the lumbrical tendons passes palmer to the axis of rotation of the metacarpophalangeal joint and they therefore have a **flexor** influence on that joint (Fig. 9.14).

If the fracture is allowed to heal in dorsal angulation, then the line of pull of the intrinsics is shifted dorsally and becomes dorsal to the axis of rotation of the metacarpophalangeal joint. They will then have an **extensor** influence on the

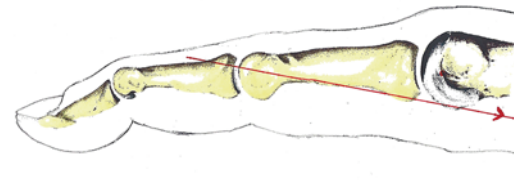


Fig. 9.14 The normal line of pull of the lumbrical tendon, palmar to the axis of rotation of the metacarpophalangeal joint

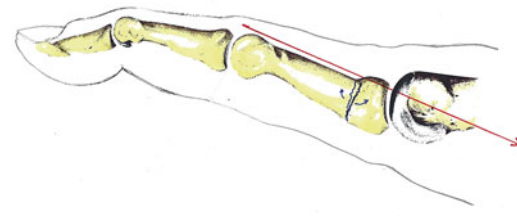


Fig. 9.15 If the fracture heals in dorsal angulation then the line of pull of the intrinsics becomes dorsal to the axis of rotation of the metacarpophalangeal joint and their action is reversed

metacarpophalangeal joint, their action having been reversed (Fig. 9.15).

The consequence of this will be significant loss of flexion at the metacarpophalangeal joint, which no amount of therapy will overcome. An osteotomy may then be necessary.

These fractures can be effectively treated with a single K-wire.

The wire is inserted between the metacarpal heads and then through the rim of the proximal phalangeal base, with the metacarpophalangeal joint fully flexed, avoiding transfixion of the collateral ligaments. It is driven up through the base of the phalanx, through the medullary canal up to either the to subchondral bone, or to engage the opposite cortex (Fig. 9.16). This can readily be done under a local anaesthetic block.

The wire is retained for 3 ½ weeks and during this time the pin-site is cleaned regularly.

With a thermoplastic splint, the metacarpophalangeal joints are immobilised in full flexion, to prevent irritation of the skin by the wire. The interphalangeal joints are mobilised under supervision by the hand therapists. The wire is removed in clinic and the hand is then fully mobilised.

Fig. 9.16 Intramedullary K-wire fixation of the base of the proximal phalanx

Pearls

Think carefully about the approach to the fracture – minimise collateral damage to the soft tissues from surgery

The combination of a precise reduction of the inter-digitations of a fresh fracture and compression with lag screws will provide a very stable fixation.

Use the drill reamings as graft.

Transverse fractures may require a wire loop or plating through a dorsal approach

Metaphyseal fractures are unstable. The intrinsics will pull the distal segment dorsally resulting in loss of flexion and power.

Neck Fractures

Neck or "sub-condylar" fractures are uncommon [11]. They occur most often in younger children in whom they may result from a crush injury typically in the hinge of a door [12]. A true lateral radiograph of the digit is essential for accurate evaluation. If these fractures heal with dorsal displacement of the condyles, then a palmar spike of bone will block flexion of the joint (Fig. 9.17). These fractures tend to be unstable and may remodel poorly due to the distance of the fracture from the physis.

Fig. 9.17 Subcondylar fracture of the phalangeal neck. These fractures remodel poorly due to the remoteness of the physis

They can readily be reduced closed, with a combination of flexion at the proximal interphalangeal joint and pressure over the dorsum of the head of the phalanx. If the fracture is several weeks old, a K-wire can be introduced into the fracture site from the dorsal aspect and, in conjunction with the above manoeuvres, used as a "joystick" to reduce the fracture. Even when reduced, these fractures are unstable.

The fracture can be held reduced with a K-wire, introduced through one of the condyles and then withdrawn through the side of the shaft, leaving the training end of the wire in the condyle, level with the cortex (Fig. 9.18). A single ended 0.9 mm wire is best for this.

The wire can then be removed at 2 ½ weeks.

Although open reduction is advocated for severely displaced fractures [12], this is not necessary for the majority. The dorsal periosteum is usually intact and these fractures tend to reduce readily and accurately. An open reduction would inflict further unnecessary damage on the digit.

Late Presentation

Occasionally these fractures will present at a late stage, when the fracture has effectively united (Fig. 9.19). These cases usually present with inability of the child to fully flex the proximal interphalangeal joint, due to the palmar boney spike impinging at the base of the middle phalanx and preventing full flexion. Flexion can be

Fig. 9.18 Fixation of the fracture with a single ended wire with the training end of the wire in the condylar fragment

Fig. 9.19 Mal-union of a phalangeal neck fracture. There is a palmar spike of bone, which will block flexion of the joint if left uncorrected

improved by excising the palmar spike. This is best done via a lateral approach. To obtain maximum flexion it is also necessary to re-create the "retro-condylar fossa", to allow full excursion of the base of the middle phalanx. This can be done using a high speed dental burr.

Condylar Fractures

Condylar fractures of the proximal phalanges are relatively common.

These may occur when a lateral force is applied to the digit resulting in an avulsion fracture through the collateral ligament. In these cases the condyle is unlikely to be deformed or comminuted and accurate open reduction will be possible.

They may also occur as the result of axial loading of the fingertip when catching a cricket or baseball or when tripping whilst ascending stairs. If as the result of such an axial force they may be bicondylar. The articular surface may be buckled and the subchondral cancellous bone compressed, making accurate reduction difficult.

Most condylar fractures will require stabilisation [13], since the oblique fracture pattern of the typical condylar fracture renders them inherently unstable and they will have a tendency to displace. Articular incongruity will ensue, and, even if this remodels, there may be significant deformity.

If the fracture presents in an un-displaced position and non-operative treatment is initially embarked upon, close monitoring will be required with weekly radiographs, as these fractures will frequently displace. Un-displaced condylar fractures in children may be more stable, due to the thickness of the periosteum, which may be intact, but vigilance is still required.

Displaced condylar fractures will require open reduction and internal fixation (Fig. 9.20).

If there is marked displacement at presentation then there will be significant soft tissue disruption, with tearing of the periosteum and this will herald instability. Even if it were possible to reduce such a fracture by closed means, it is unlikely that an acceptable result would be obtained by non-operative means and internal fixation is the method of choice for these fractures.

Method of Fixation

K-wires do not really give enough stability for immediate mobilisation. Furthermore, they tend to interfere with the soft tissues and in particular the collateral ligament. Repeated attempts to place the wire may comminute the fragment (Fig. 9.21) and when a K-wire is inserted into a small fragment, without the benefit of a pre-drilled hole, there is a risk of explosion of the fragment.

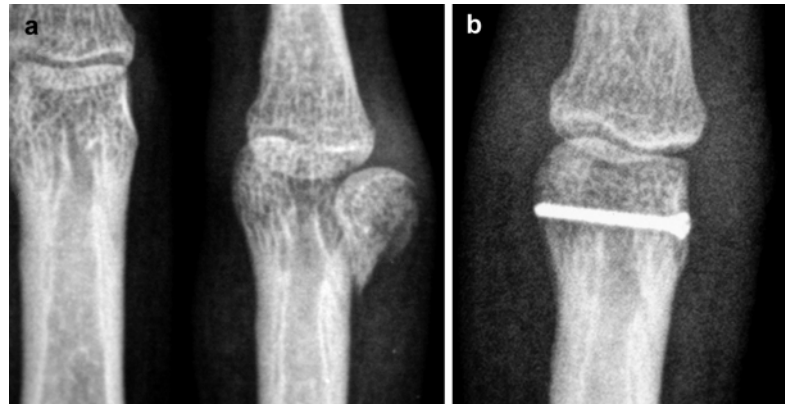


Fig. 9.20 Displaced condylar fracture (**a**) fixed with a single lag screw (**b**)

Fig. 9.21 Repeated attempts to fix a condylar fracture with K-wires may comminute the fragment

Other ways of fixing these fractures are available, such as the "bone tie" [14]. This method, however, seems unnecessarily complex. The device is relatively bulky and may impinge on the collateral ligament. It may also be difficult to maintain an accurate reduction during application. Application of the device may involve significant soft tissue stripping and a bilateral approach.

The simplest, safest and most effective method to fix these fractures is by using a small self-tapping lag screw. The screw can be inserted quickly and easily. The conical head of the screw will countersink into the softer cancellous bone of the condyle, so that there is no interference with the collateral ligament.

The screw provides a strong fixation, with compression, to maintain the reduction and permit immediate mobilisation.

Although some authorities have recommended a minimum of two screws [15] this is usually not necessary. As long as the fracture is relatively fresh, the interdigitations of the fracture surface will provide rotational stability and there is usually no need for a second screw.

Surgical Approaches

Dorsal

A dorsal approach can be used, between the central slip and lateral band of the extensor apparatus [15]. The screw is inserted dorsal to the collateral ligament.

Lateral

A lateral approach (Fig. 9.22a–d) can also be used, elevating the periostium and extensors in one layer. This will give better access to the fragment and avoids violating the plane between periosteum and extensor tendon. The collateral ligament, attached to the fragment, is identified and carefully preserved. The condyle can be flipped out into the wound and the fracture surfaces cleaned.

The fracture is then reduced. The collateral ligament is elevated slightly from the condyle, in order to place the screw beneath it. A little dent can be created in the surface of the condyle to stop the drill bit skiving off, aiding accurate placement of the screw.

After 2–3 days of rest and elevation, the dressings can be reduced and mobilisation commenced under supervision of the therapists.

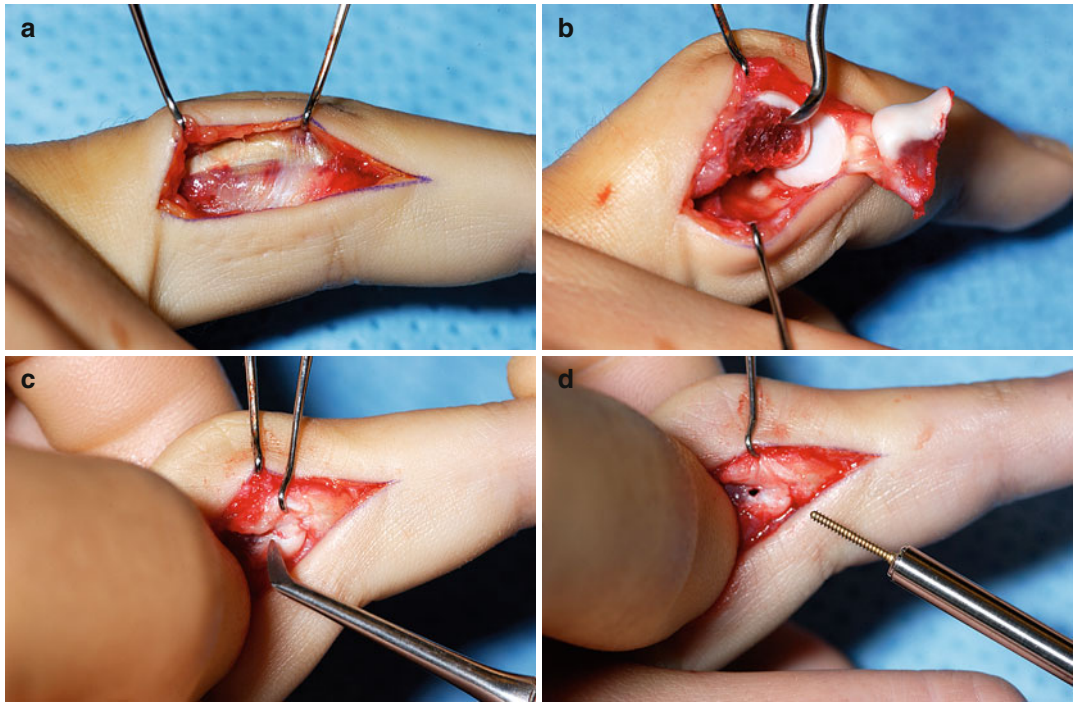


Fig. 9.22 Lateral approach to a condylar fracture (**a**). After incising and elevating the periosteum the condyle can be flipped out (**b**) and cleaned. The collateral ligament is elevated (**c**) and a lag screw inserted (**d**)

Timing of Surgery

Although it is easier to fix these fractures at an earlier stage, a few days delay makes no difference to the outcome and it is certainly preferable to do these semi-electively on a designated list. The procedure can be planned and performed on a day surgery unit for maximum service efficiency and minimum patient inconvenience. The appropriate level of expertise and supervision can be present and training opportunities can be utilised.

Peripheral centres should be encouraged to send these promptly to surgeons who perform this surgery regularly and who have the appropriate therapy available for post-operative rehabilitation, before attempting fixation.

These fractures, if presenting at a relatively late stage, can be still unpicked even after the passage of several weeks. With a combination of sharp dissection and gentle indirect traction, condylar fractures may be taken down up to three months after injury. The reduction becomes more difficult and less stable with the passage of time, as the fracture inter-digitations become less pronounced and tend to round off. This, however, may be preferable to the prospect of an intercondylar osteotomy for a healed mal-union [16], which would be a significant technical challenge.

Pearls

Condylar fractures are inherently unstable.

Undisplaced condylar fractures require close monitoring

Displaced condylar fractures require stabilisation.

A lag screw provides a strong fixation, with compression, to maintain the reduction and permit immediate mobilisation.

The interdigitations of the fracture surface will provide rotational stability and there is usually no need for a second screw.

As a few days delay makes no difference to the outcome condylar fractures can be dealt with semi-electively with the appropriate level of expertise.

If presenting at a relatively late stage, condylar fractures can be still unpicked even after the passage of several weeks and up to three months after injury.

Problems and Particular Challenges

Comminuted Fractures

Occasionally, very difficult fractures will be encountered. These fractures may be comminuted and may present or be referred late. A good position may not be achievable by non-operative means and surgical fixation may have the significant potential for producing a worse situation. The combination of an extensive dissection of an already compromised digit and a poorly fixed fracture, that can't be mobilised, would leave the patient significantly worse off. It may be preferable to accept the less than perfect position that can be achieved by less interventional means; problems which occur as the result of the imperfect reduction can be dealt with later. An example of this would be a 4-week old fracture with a bony spike blocking flexion of the base of the middle phalanx and thus limiting flexion of the proximal inter-phalangeal joint (Fig. 9.23). Rather than embarking on a difficult and extensive dissection, it may be more prudent to wait for consolidation of the fracture and then restore flexion to the joint by excising the palmar spike

Fig. 9.23 A 4 weeks old comminuted fracture with a palmar boney spike which is blocking flexion

of bone and re-creating the retro-condylar fossa. This can be done through a lateral approach from the side of the bony spike and using a dental burr to recreate the retro-condylar fossa; an easier and far less hazardous procedure.

Union

Union and subsequent consolidation of a phalangeal fracture will depend on numerous factors; age of the patient, concurrent disease and nutrition, type and severity of fracture and method of treatment. All will influence time to union. A minimally or un-displaced, uncomplicated shaft fracture will usually be stable enough to mobilise without adjuvant support at 3 ½ weeks. K-wires can therefore be removed at this stage, although some protection, such as buddy taping, may still be necessary for some activities. The fracture will not be consolidated, with complete bony healing, until 5 months [17]. This will have to be taken into account with regards to the level of protection required for return to work and contact sport.

Non-union

Non-union of the phalanges is uncommon and is particularly rare following closed phalangeal fractures treated non-operatively. When it occurs, there will usually be additional complicating factors such as infection, soft tissue compromise and stiffness. It may follow poorly executed attempts at internal fixation (Figs. 9.1 and 9.9), or open fractures with significant soft tissue damage.

If non-union is to be treated operatively, then infection must be eradicated and any bony deficit will require bone graft. A robust fixation, such as a plate, may be necessary to allow more vigorous post-operative mobilisation [18].

Even if union is achieved by operative means, the digit may have poor function and a stiff, painful digit may compromise the function of the rest of the hand. If this is the case, then amputation of the digit may be a more pragmatic option and may restore the patient to normal activities and working capability sooner.

Malunion

Rotational malunion may occasionally occur and this may result in considerable inconvenience to the patient. A de-rotational osteotomy may be necessary [19].

When considering an osteotomy a number of factors have to be taken into account.

The **timing** of the osteotomy is important. Ideally, the digit should have as good a range of movement as possible prior to surgery and the improvement of this since injury should have reached a plateau.

The **site** of the osteotomy has been the source of some debate. Metacarpal osteotomy has been advocated, as it is thought to be less hazardous [20]. It makes sense, however, to perform the correction as close to the site of deformity as possible. If the osteotomy is performed through cancellous bone at the base of the phalanx (Fig. 9.24), then bony healing should not be a problem. The surgery is well away from the proximal interphalangeal joint and the central slip, which should minimise potential problems.

Pearls

- Non-union is uncommon and is usually accompanied by additional complicating factors.
- A stiff, painful digit may compromise the function of the rest of the hand. Amputation may be a more pragmatic option and may restore working capability.
- Rotational malunion may result in considerable inconvenience.
- The digit should have as full a range of movement as possible prior to osteotomy.

In conclusion, the challenges of the proximal phalangeal shaft fracture are many and late problems are all too common.

If they are given due respect and treatment is carefully planned, then good results can be obtained.

Fig. 9.24 De-rotational osteotomy performed through the metaphyseal region of the phalanx

Fractures Around the Metacarpophalageal Joint

Avulsion Fractures

A fragment of bone can be avulsed through the collateral ligament, either from the base of the proximal phalanx, or less commonly, from the head of the metacarpal. These injuries occur as the result of the application of an adduction or abduction force to a digit, usually during a fall onto the outstretched hand. The metacarpophalangeal joint is rendered unstable by detachment of the collateral ligament. Non-union of these fractures is common and, if this occurs, instability will persist [9].

Although both injuries presumably occur by a similar mechanism, it is unclear as to why some patients should sustain avulsion fractures from the base of the phalanx and others from the metacarpal head. Consideration of the role of the palmar plate in the aetiology of these injuries may provide an explanation. The palmar plate shares an attachment with the collateral ligament at the base of the proximal phalanx, at the point where the avulsion occurs. If the injury occurs with the joint in extension, the collateral ligament would be slack, but the palmar plate is taut and would contribute to the avulsion force, thereby predisposing to avulsion from the base of the proximal phalanx. With the metacarpophalangeal joint in flexion, the palmar plate would not be under tension but the collateral ligament would, so avulsion of the collateral ligament from the weaker, cancellous non-articular bone of the metacarpal head would occur. Since the fingers are more likely to be extended during a fall onto the outstretched hand, this may also explain the relative frequencies of the two injuries, avulsions from the base of proximal phalanx being commoner. For both these type of injuries the ulnar side of the index and radial side of the little finger are more commonly affected.

The high rate of non-union may be explained by the repeated pull of the collateral ligament on the fragment during daily movements of the joint.

Fig. 9.25 An avulsion fracture from the base of the proximal phalanx (**a**). Fixation (**b**) with a lag screw through a palmar approach (**c**)

Flexion of the metacarpophalangeal joint tightens the collateral ligament and may lead to displacement of the fragment, compromising union. This precludes early vigorous mobilization of the metacarpophalangeal joint, if conservative treatment is used.

When the fractures initially treated conservatively were later secured with a lag screw, union occurred without the need for bone graft, indicating that these fractures will unite readily if adequate stability is provided. If patients are warned of the possibility of non-union and the consequent need for surgery, then some may prefer immediate surgery in the hope of avoiding a more extended period of incapacity.

When a fragment is avulsed from the **base of the proximal phalanx,** the fragment may involve a significant amount of articular surface, leading to joint incongruity if the fragment remains displaced. Conservative treatment results in high rates of non-union and ongoing symptoms, despite immobilization of the metacarpophalangeal joint for up to 8 weeks. Internal fixation is therefore the treatment of choice for these fractures when displaced (Fig. 9.25). Although some authors have advocated a dorsal tendon splitting incision to approach these fractures [13], a palmar approach (Fig. 9.26) allows far better visualization and easier reduction of the fracture fragment. Insertion of a lag screw is straightforward and optimal placement of the screw, into what is often a very small fragment, is possible. Union occurs readily, even when there has been some delay before fixation, which does not appear to affect the outcome [9].

When the avulsion fragment is from the **metacarpal head** (Fig. 9.27), union may occur in

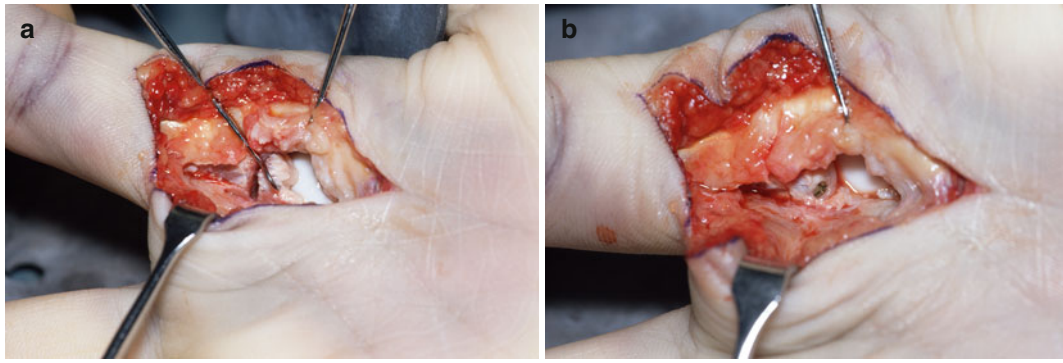


Fig. 9.26 Through a palmar approach, the flexor mechanism is reflected, elevating the palmar plate and A1 pulley to expose the fragment (**a**), which has then been fixed with a lag screw (**b**)

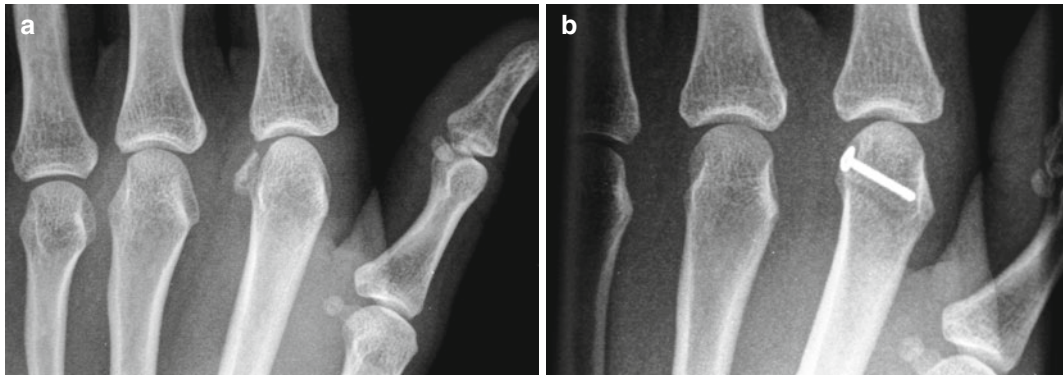


Fig. 9.27 An avulsion fracture from the metacarpal head (**a**). Fixation (**b**) with a lag screw through a dorsal approach. A single hole plate has been used as a washer

un-displaced fractures, but the rate of non-union is still high [21]. Delay does not appear to affect the outcome and it would seem reasonable, therefore, that undisplaced fractures are given a trial period of conservative treatment, which may avoid the need for surgery. Although the non-union rate is not as high as for the avulsion fractures from the base of the proximal phalanges, this is still a problem for some patients after conservative management, even with undisplaced fractures.

Fixation is achieved through a dorsal approach (Fig. 9.28) and, as with avulsions from the base of the proximal phalanx, delay in fixation does not appear to affect the outcome. If the fragment is substantial a single lag screw can be used for fixation. This can be augmented with the single hole of a plate used as a washer. If the fragment is small or comminuted then the collateral ligament with its attached fragment of bone can be secured with a nylon suture passed through drill holes in the metacarpal head. Fine drill holes can be made using a 1.0 mm K-wire and the suture passed through a needle inserted through the holes.

Metacarpal Head

Fractures of the metacarpal head are uncommon. They usually occur as the result of a direct axial force to the head of the metacarpal with the joint flexed. They may occur as the result of a fall or an object striking the hand. Since punching injuries, which are common, usually result in metacarpal neck fractures, they presumably occur when a tight fist is not formed. As the metacarpal head is

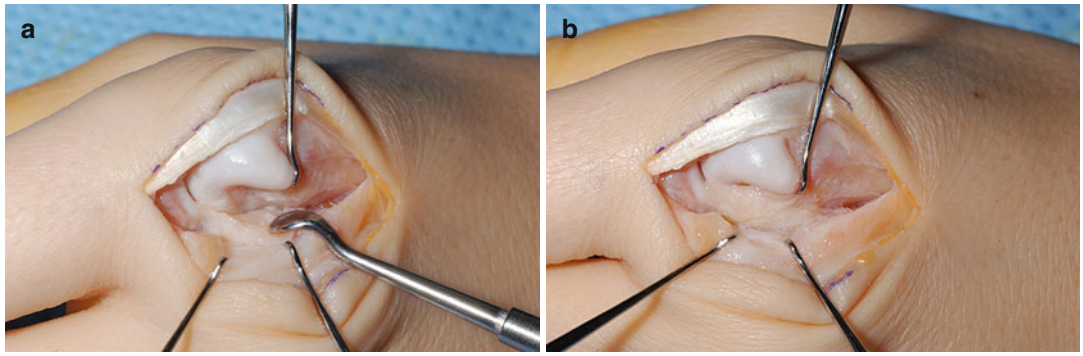


Fig. 9.28 Fixation through a dorsal tendon splitting approach (**a**). After freshening the fracture surfaces the fracture can be reduced (**b**) and fixed

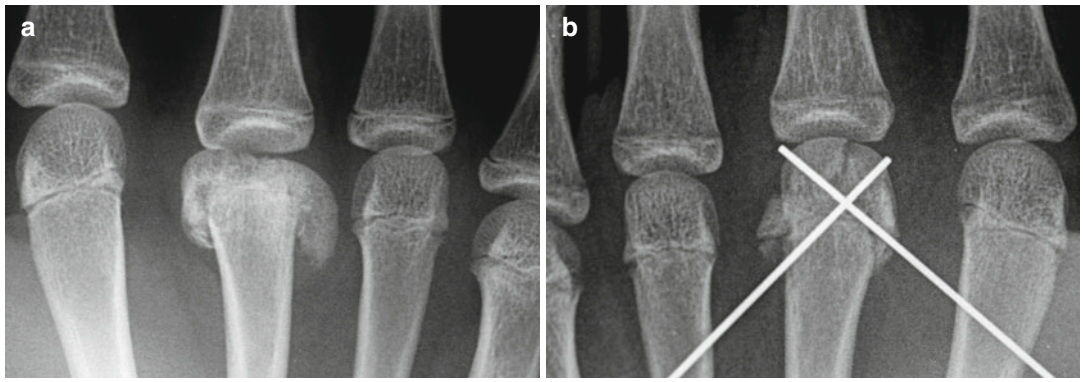


Fig. 9.29 (**a**) Closed traction on the digit will occasionally result in a reduction amenable to fixation by closed K-wiring. (**b**) The trailing ends of the single ended wires have been left in the fragments

cancellous, these fractures can be highly comminuted. Simple closed traction on the digit may reduce these fractures, which can then be stabilised with K-wires (Fig. 9.29). If the fracture cannot be reduced closed then open reduction may be necessary using a dorsal approach (Fig. 9.30). Good access can be obtained through a dorsal approach. The extensor tendon is split longitudinally, avoiding damage to the sagittal bands.

Conclusions and Summary

Fractures of the proximal phalanx are common, diverse and can be unforgiving. The aim for the outcome of these fractures is to obtain a timely uncomplicated union, with the best achievable range of movement of the finger. There is a range of available options for treatment of each type of fracture and all factors must be taken into account when planning treatment, including the method that yields the best results in the hands of the treating surgeon and the qualities of the patient attached to the finger.

If internal fixation is to be used there is no place for the "occasional surgeon". The best and often only chance of obtaining a good result is at the first surgical intervention. Many of these fractures do not have to be treated immediately, but can wait a few days so that the best expertise and facilities are available.

When particularly difficult fractures of the proximal phalanx are encountered it should be remembered that there can be significant potential

Fig. 9.30 A highly comminuted fracture of the metacarpal head (**a**). Fixation with a T-shaped plate (**b**)

to make the situation worse. It may be more judicious to accept a less than perfect situation as some problems can be dealt with in a safer way at a later date when union has occurred.

In conclusion, the challenges of the proximal phalangeal fracture are many and late problems are all too common. If, however, they are given due respect and treatment is carefully planned, then good results can be obtained.

The involvement of appropriately trained, dedicated hand therapists will improve the results of all treatments of these fractures considerably and will free up surgical time for clinicians in busy clinics.

References

1. Stanton JS, Dias JJ, Burke FD. Fractures of the tubular bones of the hand. J Hand Surg Eur. 2007;32(6): 626–36.
2. Barton N. Internal fixation of hand fractures. J Hand Surg. 1989;14B(2):139–42.
3. Horton TC, Hatton M, Davis TRC. A prospective randomized controlled study of fixation of long oblique and spiral shaft fractures of the proximal phalanx: closed reduction and percutaneous Kirschner wiring versus open reduction and lag screw fixation. J Hand Surg Eur. 2003;28(1):5–9.
4. James JI. Fractures of the proximal and middle phalanges of the fingers. Acta Orthop Scand. 1962;32: 401–12.
5. Belsky MR, Eaton RG, Lane LB. Closed reduction and internal fixation of proximal phalangeal fractures. J Hand Surg. 1984;9A:725–9.
6. Green DP, Anderson JR. Closed reduction and percutaneous pin fixation of fractured phalanges. J Bone Joint Surg. 1973;55A:1651–3.
7. Hornbach EE, Cohen MS. Closed reduction and percutaneous pinning of fractures of the proximal phalanx. J Hand Surg. 2001;26B(1):45–9.
8. Ford DJ, El-Hadidi S, Lunn PG, Burke FD. Fractures of the phalanges: results of internal fixation using 1.5 mm and 2 mm A.O. screws. J Hand Surg. 1987; 12B(1):28–33.
9. Shewring DJ, Thomas RH. Avulsion fractures from the base of the proximal phalanges of the fingers. J Hand Surg Eur. 2003;28(1):10–4.
10. Lister GD. Intra-osseous wiring of the digital skeleton. J Hand Surg. 1978;3:427–35.
11. Dixon GL, Moon NF. Roational supracondylar fractures of the proximal phalanx in children. Clin Orthop Relat Res. 1972;188:120–30.
12. Newington DP, Craigen MA, Bennet GC. Children's proximal phalangeal neck fractures with 180 rotation deformity. J Hand Surg. 1995;20B(3):353–6.
13. Hastings 2nd H, Carroll CT. Treatment of closed articular fractures of the metacarpophalangeal and proximal interphalangeal joints. Hand Clin. 1988; 4:503–27.
14. Sammut D, Evans D. The bone tie: a new device for interfragmentary fixation. J Hand Surg. 1999;24B(1): 64–9.
15. Day CS, Stern PJ. Chapter 8. Fractures of the metacarpals and phalanges. In: Wolfe SW, Hotchkiss RN, Pederson WC, Kozin SH, editors. Green's operative hand surgery, vol. 1. 6th ed. Philadelphia: Elsevier/ Churchill Livingstone; 2011. p. 239–90.

16. Teoh LC, Yong FC, Chong KC. Advancement osteotomy for correcting condylar malunion of the finger. J Hand Surg. 2002;26B(1):31–5.
17. Smith FL, Ryder DL. A study of the healing of one hundred consecutive phalangeal fractures. J Bone Joint Surg. 1935;17A:91–109.
18. Jupiter JB, Koniuch MP, Smith RJ. The management of delayed union and nonunion of the metacarpals and phalanges. J Hand Surg. 1985;10A:457–66.
19. Buchler U, Gupta A, Ruf S. Corrective osteotomy for post traumatic malunion of the phalanges in the hand. J Hand Surg. 1996;21B(1):33–42.
20. Menon J. Correction of rotary malunion of the fingers by metacarpal rotational osteotomy. Orthopaedics. 1990;13:197–200.
21. Shewring DJ, Thomas RH. Collateral ligament avulsion fractures from the heads of the metacarpals of the fingers. J Hand Surg Eur. 2006;31(5):537–41.

Injury-Fractures-Carpo-Metacarpal Joint and Metacarpal

10

Chye Yew Ng and Michael J. Hayton

Keywords
Metacarpal • Carpometacarpal joint • Trapeziometacarpal joint • Fracture • Fracture-dislocation • Joint dislocation • Rotational deformity • Intra-articular • Bennett's fracture • Rolando's fracture • Malunion

Introduction

Metacarpal fractures are extremely common and, after distal radial fractures, are the second most common fracture treated by an orthopaedic surgeon [1]. The annual incidence has been estimated at $130/10^5$ population/year, with a male to female ratio of 85:15 [1]. The majority of fractures do not require surgical intervention and can be treated adequately with immobilisation or protective splinting. Irreducible or unstable fracture patterns warrant special attention and fixation to prevent long-term problems. As these fractures present with an average age of 29.9 years [1], any morbidity and loss of function as a consequence of inadequate treatment or surgical complications could have a significant socioeconomic impact.

C.Y. 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 WN6 9EP, UK
e-mail: chyeng@gmail.com

M.J. Hayton, BSc (Honours), MBChB, FRCS (Tr&Orth), FFSEM (UK)
Consultant Orthopaedic Hand Surgeon, Upper Limb Unit, Wrightington Hospital, Hall Lane, Appley Bridge, Wigan, Lancashire WN6 9EP, UK
e-mail: mikehayton@gmail.com

Functional Anatomy

The metacarpals are bowed with a dorsal apex. The volar cortex is consistently 20 % thicker than the dorsal cortex [2]. They form the longitudinal and transverse arches of the hand, provide attachment for the interrosei and form an articulated system of levers between the flexor and extensor tendons [3]. The intrinsic muscles and the relatively stronger extrinsic flexor tendons create deforming forces on the metacarpal shaft, leading to typical apex dorsal angulation, when a fracture occurs. This deforming force is further exaggerated with a greater lever arm, when a fracture is located more proximally. Fractures to the metacarpals thus could disturb

I.A. Trail, A.N.M. Fleming (eds.), *Disorders of the Hand: Volume 1: Hand Injuries*,
DOI 10.1007/978-1-4471-6554-5_10, © Springer-Verlag London 2015

the equilibrium of the system, resulting in impairment of hand function.

The metacarpals are attached to each other by stout interosseous ligaments at their bases and by the deep transverse inter-metacarpal ligaments distally. The intervening connections contribute to the stability of the transverse arches of the hand and limit the degree of shortening of isolated metacarpal fracture. Cadaveric study has demonstrated that, for every 2 mm of metacarpal shortening, there would be an average of 7° of extensor lag at the metacarpophalangeal (MCP) joint [4]. The extensor lag may be compensated partially by the inherent ability for hyperextension at the MCP joint [4]. This is confirmed by a clinical study showing recovery of the extensor lag with time [5]. In addition to the extensor lag, shortening of more than 3 mm was shown to be associated with a decrease in the long flexor and extensor forces in another cadaveric experiment [6].

The thumb metacarpal is thicker and shorter than the finger metacarpals. It has a saddle-shaped facet proximally for the trapezial articulation. The finger metacarpals have expanded bases, which articulate proximally with the distal row of the carpus and on the sides with their neighbouring metacarpals. Articulations between the index and middle finger metacarpals, with the trapezoid and capitate, permit little movement. In contrast, articulations between the ring and little finger metacarpals with the hamate allow considerable flexion-extension and slight rotation. This variation in carpometacarpal (CMC) movement has implications on the degree of angulation that is tolerable in metacarpal fractures. As a guide, the hand can tolerate 10–15° of dorsal angulation, more than the available CMC joint movement of the involved digit [7]. Hence, only 10–15° of angulation can be accepted in the index and middle finger metacarpal fractures, whilst 30–35° is tolerable in the ring finger and up to 50° in the little finger. A number of biomechanical studies have consistently identified angulation of 30° as being the threshold, beyond which there would be a decrease in the flexor tendon efficiency [6, 8]. However, these did not take into account the compensatory mechanism present in living subjects.

Mechanisms of Injury

Metacarpals may fracture by direct or indirect force. The fracture pattern that occurs will be dependant upon the direction and amount of force that is applied. Energy applied with an axial load and rotation may cause a spiral fracture. The presence of a butterfly fragment reflects greater energy dissipation. With direct impact, the fracture pattern is more likely to be transverse. The majority of metacarpal fractures are caused by punching-type injuries. With the MCP joint maximally flexed, the impact strikes the metacarpal head and energy is transmitted along the shaft. Thumb fractures are usually caused by a fall or twisting type injury. Punching type injuries do occasionally occur and are caused by the abducted thumb becoming caught.

Clinical Assessment

In addition to basic information regarding the patient's age, hand dominance and pre-injury level of function, history-taking should focus on the mechanism of injury, potential exposure to a foreign body, such as glass and symptoms of pain and clicking. Clinical examination of the injured hand begins with inspection for swelling, ecchymosis and the presence of an open wound, particularly those caused by a human tooth. This would constitute the fight-bite injury and require urgent surgical washout and debridement. Palpation is then performed to localise areas of tenderness and crepitation. The neurovascular status should be assessed and documented carefully. Further examination should be performed to establish if there is rotational, angulation or shortening deformity of the injured digit. Whilst the patients may have some discomfort, they are asked to close the hand and make a fist. The fingers should all flex in line and generally all point towards the scaphoid tubercle. This can, however, vary between individuals and one should compare with the opposite non-injured hand. If there is restricted movement due to fracture, rotational deformity may be assessed by an end-on view of the nail plates [9]. It has been estimated that 5° of metacarpal shaft rotation would result

Fig. 10.1 Appearance of dropped knuckles

in 1.5 cm overlap of the digits [10]. However, mild degrees of rotational deformity (up to 10°) can sometimes be tolerated by the patient [9] and may be preferable to the potential risks associated with surgery. If a greater degree of rotational deformity is observed, the fingers may start to cross over one another, resulting in scissoring. In an anaesthetised patient, the tenodesis effect may be demonstrated by moving the wrist backwards and forwards, which would produce adequate flexion and extension of the digits allowing rotational alignment to be judged.

As the fracture deforms with apex dorsal angulation, the metacarpal head becomes more prominent in the palm of the hand. This is sometimes called "dropped knuckle" (Fig. 10.1). If allowed to heal in this position, the metacarpal head may become painful on gripping or holding objects in the palm [11]. The displaced metacarpal head can usually be palpated in the acute situation. If readily palpable, it may be prudent to consider either manipulation or fixation.

Shortening of the digit can be observed by placing both hands flat, with the digits extended, and recording the length of the injured digit in relation to its neighbouring digits. This is compared to the opposite hand, to reveal the shortening, if present. Generally 3–4 mm of shortening is well tolerated in all digits.

Clinical Pearl

Decision making

The majority of metacarpal fractures do not require surgical intervention and can be treated adequately with immobilisation or protective splinting.

When assessing for rotational deformity:

- Firstly, ask the patient to make a fist. The fingers should all flex in line and generally all point towards the scaphoid tubercle.
- Secondly, look at the digits end-on and the nail plates should be in line.
- Thirdly, repeat examination weekly during the first 3 weeks, as the deformity may evolve as the swelling subsides.
- Fourthly, in an anaesthetised patient, use the tenodesis effect to observe flexion and extension of the digits.

When assessing for shortening:

- Place both hands flat and observe the length of the injured digit in relation to its neighbouring digits.
- Make a fist and look for a dropped knuckle.

When assessing metacarpal neck fractures (particularly little finger), palpate for the displaced metacarpal head. If it is prominent, it may result in a painful grip.

10–15° of angulation can be accepted in the index and middle finger metacarpal fractures, whilst 30–35° is tolerable in the ring finger and up to 50° in the little finger.

Angulation deformity in the sagittal or coronal planes, of up to 30°, may be tolerable in the thumb metacarpal.

Fight-bite injury warrants urgent surgical washout and debridement.

CMC joint dislocations are easily missed and CT scan is recommended if there is any clinical suspicion.

Investigations

If there is clinical suspicion of a metacarpal fracture or dislocation, plain radiographs should be requested, including the posteroanterior (PA), lateral and oblique views. Specialised views may be useful to delineate the pattern of injury in selected cases. A 30° pronated lateral view is particularly useful for index and middle metacarpal fractures, whilst a 30° supinated lateral view outlines ring and little metacarpal fractures well. The Brewerton view [12], classically used in the rheumatoid hand, may visualise the collateral recesses which are the sites of insertion of the collateral ligaments. This view is taken with the wrist supinated, the MCP joint flexed at 65° and the dorsum of the proximal phalanges lying flat against the radiograph cassette, the beam is then angled at 15° ulnar to radial (Fig. 10.2). Subtle avulsion fractures of the collateral ligaments, that may have been missed on standard views, are often identified. The skyline view, taken with the MCP joint fully flexed, may show impaction fractures of the metacarpal head, otherwise not seen in other projections [13] (Fig. 10.3). This is particularly useful in human fight-bite injuries.

CMC joint dislocations can be easily missed [14]. Care must be taken to course the joint line of each joint individually on each of the standard films. The 'metacarpal cascade lines' drawn on a PA film may increase its diagnostic yield [15]. In a normal hand, all the lines superimposed along the central axis of the finger metacarpals, should converge onto a point 2 cm proximal to the distal

Fig. 10.2 Positioning for Brewerton view

Fig. 10.3 Positioning for skyline view

Fig. 10.4 Positioning for Mehara and Bhan view

Fig. 10.5 Sagittal CT scan showing a CMC joint dislocation

radial articular surface. Divergence of the lines suggests possible disruption of the CMC joint, which should prompt closer examination of other views. A true lateral radiograph will often demonstrate the dislocation, which is typically in a dorsal direction [14]. In addition, Mehara and Bhan described a view for evaluating possible dislocation of the index CMC joint [16] (Fig. 10.4).

Increasingly, computed tomography (CT) scan is employed if there is any doubt as to whether a joint is congruent, following injury (Fig. 10.5). It can help to delineate the fracture pattern and also to plan the method of fixation, if required.

Treatment Options and Outcomes

Optimal management of metacarpal fractures relies on prompt identification of injuries, which would require surgical intervention, as well as appropriate application of conservative treatment for the majority of fractures. The fractures may be classified according to location (head, neck, shaft or base); configuration (transverse, oblique, spiral or comminuted); and whether it is open or closed. The indications for fixation are summarised in Table 10.1. A key determining factor for intervention is the stability of the fracture. This is, in turn, determined by the nature of injury, fracture configuration, number of metacarpals involved and amount of soft tissue destruction.

Table 10.1 The indications for fixation are listed below

Significant shortening >4–5 mm
Significant angulation (apex dorsal)
Angulation less well tolerated moving from ulnar to radial metacarpals
Index and Middle <10–15° is tolerated
Ring <30–35° is tolerated
Little <50° is tolerated
Rotational deformity
Open fractures
Multiple digit fractures
Displaced intra-articular fracture
Severe soft tissue injury including associated tendon lacerations

Nonoperative Treatment

An externally applied splint or cast effects indirect control over the fracture alignment, using the principles of three-point fixation, ligamentotaxis and neutralisation of myotendinous deforming forces. The surface area of contact should be as broad as possible, to reduce the risk of skin necrosis. Padding should be limited to one or two layers of soft wool, to allow for further swelling, but also to ensure adequate pressure application. If the padding is too thick, the cast would become loose as the swelling settles, causing the consequent loss of effective contact areas. This is an aspect that is often overlooked during the follow-up of conservatively treated fractures. Radiographs not only delineate the bony structures, but also outline the cast, splint and the thickness of padding.

Generally, immobilisation longer than 3 weeks is not required and interphalangeal joint movement should begin immediately following trauma. Patient's understanding and compliance with the regime are paramount to the success of the treatment.

Operative Treatment

The various fixation devices (wire, plate, screw and nail) are a means that can be utilised to maintain a reduction, or to revert an unstable fracture configuration, allowing healing in a favourable position. If a deformity is deemed unacceptable, closed reduction under anaesthesia, combined with percutaneous pinning, should be attempted first, in the majority of cases. This has the advantage of being minimally invasive, thereby preserving the soft tissue envelope. When satisfactory reduction cannot be achieved by closed means, such as an intra-articular fracture or inherently unstable multiple fractures, open reduction is indicated. The choice of fixation device is dictated by the fracture configuration, availability of implants, costs and, perhaps most importantly, familiarity of the surgeon with a particular technique. Rigid fixation, afforded by screw and plate, may allow early mobilisation, although this is compounded by the potential complications of stiffness, non-union, plate prominence, infection and tendon rupture [17].

Clinical Pearl

Non-operative treatment

When applying a splint or cast, keep the surface area of contact as broad as possible to reduce the risk of skin necrosis.

Padding should be limited to one or two layers of soft wool to allow for further swelling, but also to ensure adequate pressure application.

Radiographs not only delineate the bony structures, but also outline the cast, splint and the thickness of padding.

Generally, immobilisation longer than 3 weeks is not required and interphalangeal joint movement should begin immediately following trauma.

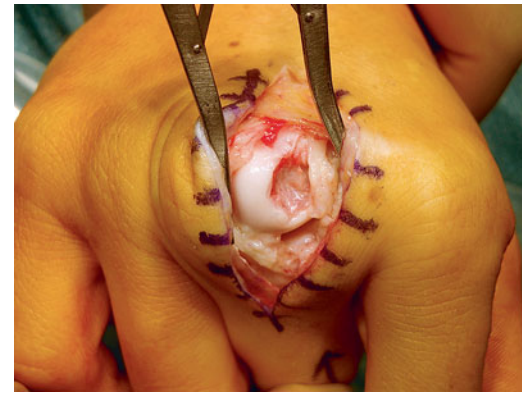


Fig. 10.6 An example of osteochondral damage to the metacarpal head

Head Fractures

McElfresh and Dobyns described ten categories of the metacarpal head fractures in a series involving 100 patients [18]. In order of decreasing frequency, they were comminuted, oblique sagittal, ligament avulsions, osteochondral, loss of substance, Salter-Harris III, occult compression with avascular necrosis, vertical coronal, transverse horizontal and boxer's fractures with extension into the joint (Fig. 10.6). A variety of treatment strategies thus are required to restore a congruous metacarpal head and to facilitate early mobilisation of the joint.

The surgical approach is through a longitudinal curvilinear incision around the MCP joint, which prevents a tender scar being placed directly over the joint. With the skin flaps retracted, the extensor hood is incised longitudinally. The tissue plane between the tendon and the capsule is developed and a longitudinal capsulotomy is performed to expose the joint and fracture. Head splitting fractures can be fixed with countersunk screws (Fig. 10.7a, b). If the fracture propagates down the shaft then additional fixation with a plate may be needed. Avulsion fractures are

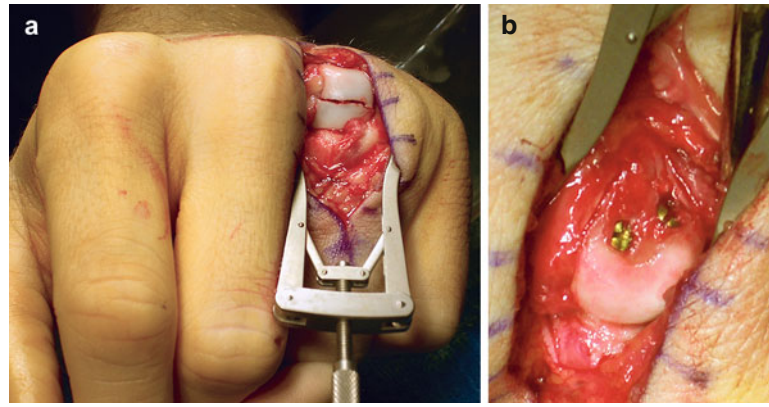


Fig. 10.7 (**a**) A coronal vertical fracture of the metacarpal head. (**b**) Dorsal view showing the fracture fixed with two countersunk screws

prone to non-union, when treated conservatively [19]. In the case of ligament avulsion which is too small for screw fixation, suture anchor repair can be a useful technique.

Neck Fractures

The little finger is the commonest metacarpal to be fractured, accounting for 27 % of total hand fractures in a recent population study [20]. This is often termed the 'boxer's fracture' and has been shown to be associated with social deprivation in men [20]. Whilst there is general agreement regarding the acceptable dorsal angulation for fractures of the index, middle and ring fingers, the tolerable angulation for the little finger remains controversial, with a range of 20–70° reported in the literature [21–26]. This is at least partially due to the high inter-observer and intra-observer variability associated with radiographic assessment of the angle [27].

Apart from the threshold for intervention, the need for immobilisation [25, 28] and even follow-up [29] have been questioned. In a series of 100 metacarpal neck or shaft fractures, which were randomised to receive either a dorsal/ulnar cast immobilising the wrist and the joints of the involved digits or a functional cast allowing motion of the wrist and the digits, the latter group achieved better radiographic alignment and returned to work earlier [30]. In a similar prospective randomised trial of 50 patients with little finger metacarpal fractures, ulnar gutter cast was compared to a functional tape [31]. The tape group showed a quicker functional recovery, but the results were similar at 6 months. Harding et al. prospectively randomised 73 patients with little finger metacarpal neck fractures (<40°) to treatment with a metacarpal brace or neighbour strapping [32]. The former group reported less pain, better range of movement and earlier return to work.

For the fractures with unacceptable deformity, manipulation with external splintage is of doubtful value, as the reduction achieved is difficult to maintain [31, 33]. Fixation with percutaneous transverse Kirschner wires [34, 35] or intramedullary wires [36] are both well-described techniques (Fig. 10.8). Wong et al. compared the two techniques in a non-randomised trial of 59 patients with boxer's fractures [37]. At a mean follow-up of 24 months, both groups showed similar radiological and functional outcomes. In another prospective randomised trial involving 36 patients, both techniques produced functional range of movement of the hand, but the group treated with intramedullary pinning consistently recorded higher total active motion [38]. All fractures in the study united and no infection was reported. Facca et al. compared locking plates and intramedullary wiring in 38 fractures [39]. The use of locking plates not only incurred extra cost, but also resulted in poorer motion and was associated with complications of stiffness, head necrosis and delayed union [39].

Strub et al. performed a prospective trial where 40 patients with boxer's fractures were

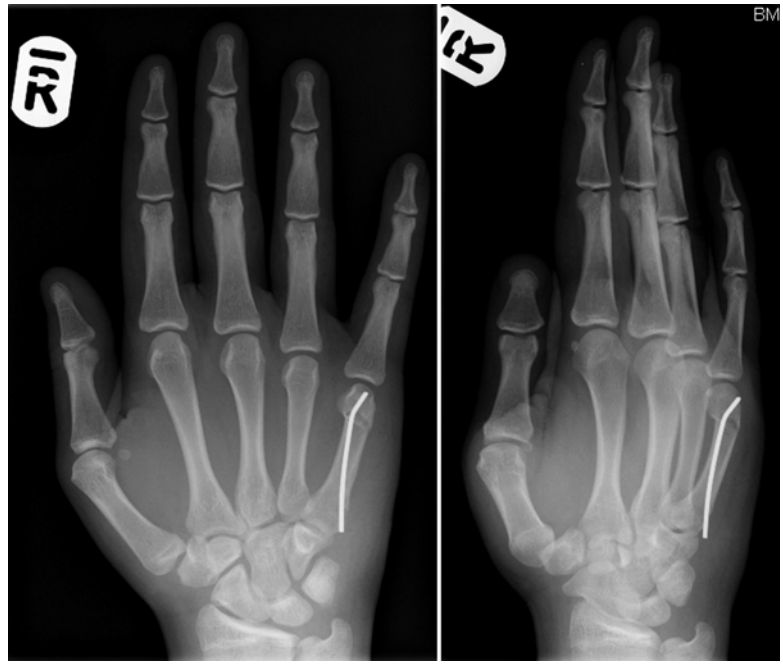


Fig. 10.8 Intramedullary wiring for a fifth metacarpal fracture

treated with intramedullary splinting or conservatively without attempt at reduction [40]. At 12 months, the surgically-treated group reported higher satisfaction and better appearance of the hand. However, no statistically significant differences in the range of motion and grip strength were found between the study groups.

Shaft Fractures

As discussed previously, most uncomplicated isolated metacarpal shaft fractures are amenable to conservative treatment. Viegas et al. described the use of a functional brace, providing 3-point fixation, whilst allowing motion of the wrist and digits, in treating metacarpal fractures of index to little fingers [41]. They reported less residual angulation when compared to an ulnar gutter splint. However, one has to be aware of the potential risk of causing skin necrosis with the brace use [42]. In a prospective trial, 42 patients with isolated stable metacarpal shaft fractures were randomly allocated to treatment with immobilisation in a plaster slab or immediate mobilisation in a compression glove [43]. The glove group regained motion quicker during the initial recovery period and avoided the loss of function associated with plaster immobilisation. When commencing early mobilisation, the patient may notice that the hand clicks on flexion of the digits. They should be reassured that the clicking usually stops as the fracture becomes sticky, progressing to union in 3–4 weeks. Regular review at a fracture clinic is required to ensure that, as the swelling settles, the fracture does not displace further, or surgical intervention may be indicated.

A number of surgical techniques have been described for treating metacarpal shaft fractures, including open or closed reduction methods and fixation with screws and plates [44], Steinmann pin [45], wires or specialised intramedullary devices. Dental wires can be employed as an interosseous loop [46], a cerclage [47], or combination of the two configurations [48]. Kirschner wires may be used to provide transverse fixation [34, 35] or intramedullary splinting [49, 50]. Furthermore, the transverse wires could be linked with acrylic resin [51] or methylmethacrylate rods [52] functioning as a true external fixator. In

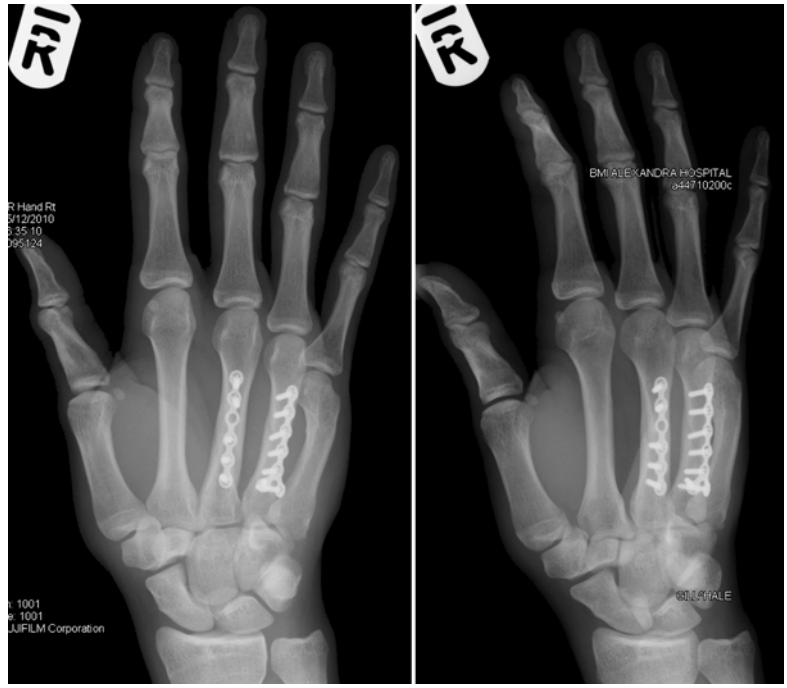


Fig. 10.9 Plating of metacarpal fractures

addition, specialised intramedullary devices [53, 54] have been developed, but their superiority over simple wires are yet to be proven in clinical studies.

Intramedullary fixation is not suitable for spiral fractures, but more suited for transverse or short oblique fractures. The intramedullary devices are particularly useful to fix extra-articular metacarpal head fractures that may be difficult to approach through an open technique. However, one has to appreciate the highly variable geometry of the canal when considering intramedullary fixation of a fracture [2].

Open reduction may be required in cases with fracture comminution, irreducible fractures, multiple fractures or open fractures. The approach is through an inter-metacarpal longitudinal incision. The incision is planned, so as not to be directly over the extensor tendon, which reduces the risk of post operative adhesions. The tendon is retracted to expose the fracture and minimal periosteal stripping is performed to facilitate reduction. It is crucial to fit the plate to the reduced bone, rather than the opposite, as the latter may result in mal-reduction, as the final screw is tightened (Fig. 10.9). This problem may be overcome with the advent of the locking plate, which functions as an internal external-fixator, where perfect bony contact with the plate is not necessary. It also has the theoretical advantage of affording stable, unicortical fixation, which could minimise flexor tendon irritation [55]. However, the extra cost of locking plate over non-locking plate is yet to be justified by the results of a prospective randomised clinical trial. Regardless of the fixation device used, the basic principles of careful tissue handling, anatomical reduction and stable fixation construct thus allowing early mobilisation, would have more influence on ensuring an optimal outcome. Particular attention should be paid to achieve a layered closure over the hardware, to minimise tendon irritation.

Ozer et al. compared intramedullary nail with plate-screw fixation for 52 displaced, extra-articular metacarpal fractures [56]. Both achieved functional and comparable total active motion, without significant difference in union rates or DASH scores. Nailing was quicker to perform, but was associated with higher incidences of loss of reduction, penetration of MCP joint and secondary surgeries for hardware removal.

Base Fractures and Carpometacarpal Fracture-Dislocations

These injuries are usually as a result of axial load on the hand, with the wrist flexed and reflect high energy dissipation. The frequently encountered pattern is a fracture and/or dislocation of the little with/without ring finger CMC joints [57]. When presented with an apparently isolated ring finger metacarpal fracture, one needs to be vigilant for an associated CMC joint injury [58]. Careful assessment of the ulnar nerve function, particularly the deep motor branch, is paramount, due to its intimate relationship to the joint and the potential risk of being damaged [59, 60]. The little finger metacarpal base may be fractured with a radial corner held in place by strong intermetacarpal ligaments. The shaft is then retracted proximally by the flexor and extensor carpi ulnaris muscles [61]. The fracture is reduced with axial distraction and direct dorsal pressure over the subluxed base, while percutaneous Kirscher wires are inserted into the carpus and neighbouring metacarpal. Open reduction may be necessary when there is interposed bony fragment or ligaments [62, 63]. Other rarer patterns of injury have been described, including volar dislocation [60], divergent dislocation [64] and transcarpal CMC dislocations [65] (Fig. 10.10a–d).

Kjaer-Peterson et al. reported the outcomes of 64 intra-articular fractures of the fifth metacarpal base, which were treated by both conservative and surgical means, after a median follow-up of 4.3 years [62]. They concluded that the fractures were difficult to reduce and frequently resulted in intermittent pain, decreased grip strength and osteoarthritis. Minimally displaced fractures were noted to have a high propensity for late displacement, which were not controlled by plaster cast [62]. In contrast, Lundeen and Shin reported satisfactory results with closed reduction and plaster immobilisation of 37 similar fractures [66]. They showed that the outcome was not affected by fracture type, extent of subluxation or articular incongruity, or the presence of arthrosis. However, both studies were retrospective with significant numbers lost to follow-up.

Thumb Metacarpal Fractures

The thumb has unique anatomy and functions deserving special consideration. Due to its multiplanar motion, rotational deformity is more tolerable. Angulation deformity in the sagittal or coronal planes of up to 30° may be compatible with little loss of function because of the compensatory motion in the neighbouring joints. However, the appearance may not be acceptable to the patient. In contrast, intra-articular base fractures are challenging to manage, in order to prevent stiffness and secondary arthritis.

Head Fractures

These intra-articular fractures are managed according to the principle of anatomic reduction. Condylar fractures may be reduced with the help of a pointed clamp, before fixation with percutaneous wires. If closed means fail, open reduction is performed via a dorsal approach, through the interval of extensor pollicis longus (EPL) and extensor pollicis brevis (EPB). Care is taken not to damage the branches of superficial radial nerve. Screw fixation can be achieved if the fragment is large enough.

Shaft Fractures

The key points in managing a shaft fracture, which is often the consequence of direct trauma leading to significant comminution, are restoration of length and proper soft tissue tensioning. An external fixator is ideal to achieve those aims. Open reduction and internal fixation with a fixed angle device can be employed, but at the expense of further soft tissue trauma and devascularisation of the bone.

Extra-articular Base Fractures

The typical deformity subsequent to this injury is apex dorsal angulation. This is due to deforming forces of abductor pollicis brevis, adductor pollicis and flexor pollicis brevis flexing the distal fragment, whilst the abductor pollicis longus (APL) extends the proximal fragment [67]. It is vitally important to exclude an intra-articular involvement by careful exam-

Fig. 10.10 (**a**, **b**) Radiographs showing metacarpal shaft fractures of the middle and ring fingers associated with CMC joint dislocation of the little finger. (**c**, **d**) The injuries were successfully treated with closed reduction and intramedullary wiring for the shaft fractures and percutaneous pinning for the CMC joint dislocation

ination of the radiographs. Nonsurgical treatment is usually adequate. Reduction is achieved with axial traction and direct pressure over the apex of the fracture. A thumb spica is then applied, keeping the thumb in extension and abduction.

Intra-articular Base Fractures and Carpometacarpal Fracture-Dislocations

Intra-articular base fracture, with a volar-ulnar fragment, is known as Bennett's fracture [68]. The fragment is held attached by the volar oblique ligament, whilst the shaft is retracted by the APL and adductor pollicis, resulting in flexion, supination and proximal migration [69]. Closed reduction is performed with axial traction, combined with dorsal pressure at the base of the thumb. The aim is to neutralise the deforming forces and reduce the shaft onto the volar fragment. Once satisfactory reduction is confirmed on image intensifier, percutaneous Kirschner wires are inserted from the base of the metacarpal into the trapezium, as described by Wagner [70]. Another technique is the use of intermetacarpal pinning [71]. The wire is removed after 4 weeks, but a thumb spica is worn for an additional fortnight.

The long term significance of articular incongruity at the CMC joint, hence the need for open reduction, has been questioned. Cannon et al. reviewed 25 patients with Bennett's fractures, which were mainly treated with plaster immobilisation [72]. After a mean follow-up of 9.6 years, 5 had malrotation, 21 had loss of movement, 16 had articular separation of more than 1 mm and 23 had varus angulation. However, no definitive relationship between initial reduction and subsequent development of symptomatic arthritis could be established. In contrast, Kjaer-Peterson et al. retrospectively reviewed 41 Bennett's fractures after a median follow-up of 7.3 years and suggested that residual displacement was associated with a higher incidence of radiographic arthritis and clinical symptoms [73]. Livesley reported long term outcomes of 17 Bennett's fractures, which were treated with closed reduction and casting [74]. After an average follow-up of 26 years, 17 were symptomatic and all had a reduced mobility and grip strength. Radiographic arthritis and joint subluxation were observed in most patients. Operative intervention was thus advocated by the author [74].

Lutz et al. compared the results of closed reduction Kirschner wire fixation, with open reduction internal fixation of 32 Bennett's fractures [75]. After a mean follow-up of 7 years, no significant difference was detected in pain, range of movement, strength or radiographic arthritis. However, the percutaneous group was more likely to develop an adduction deformity of the thumb.

A T- or Y-shaped articular fracture of the base is known as Rolando's fracture [76]. The comminution may be more severe than suggested by the plain radiographs [77]. As such, CT scans may have a role in defining the extent of fragmentation. The fracture is inherently unstable and anatomical reduction is difficult to achieve using closed means. However, open reduction is technically demanding and the fragments may become completely devascularised as a result. Longhoff et al. reviewed 17 Rolando's fractures, with a median follow-up of 5.8 years and failed to establish a correlation between the quality of reduction and the development of late symptoms and arthritis [77]. Yet the authors advocated restoration of the joint surface as the aim of treatment.

Gelberman et al. employed the oblique traction technique and reported satisfactory results [78]. A small skin incision is made just distal to the fracture and just volar and radial to the EPB. A wire is drilled obliquely through the shaft of the metacarpal, distal to the fracture, aiming to emerge through the middle of the web of the thumb. The proximal end of the wire is bent and the wire is advanced until it engages the bone. The distal end allows traction via a banjo outrigger splint. For complex fractures, a combination technique of intermetacarpal external fixation with bone grafting and adjunctive internal fixation may be necessary [79]. If the fragments are of adequate size, an open reduction internal fixation may be performed, with small lag screws incorporated through a T- or L-shaped plate [80]. The joint is approached through a Wagner incision along the subcutaneous border of the metacarpal. The thenar muscles are reflected subperiosteally and the joint capsule is incised.

Pure Trapeziometacarpal Joint Dislocation

Trapeziometacarpal (TMC) joint dislocations are rare and are invariably dorsal [81] (Fig. 10.11). Cadaveric study has shown the dorsoradial ligament

Fig. 10.11 Example of pure TMC joint dislocation

to be the primary restraint to dorsal dislocation of the joint, with the volar oblique ligament permitting dislocation by subperiosteal stripping from the base of the thumb metacarpal [82]. The joint was found to be most stable in pronation and extension after reduction. In clinical setting, dislocation may have spontaneously reduced on presentation. Diagnosis is then based on high index of suspicion and stressed radiographs, compared to the contralateral side. Differentiating a partial from a complete ligament rupture is vital, as a partial injury is amenable to cast immobilisation for 6 weeks.

The optimal treatment remains controversial. Closed reduction, with or without Kirschner wire fixation [81], open reduction internal fixation [83] and early ligament reconstruction [84] have all been described with variable results. In a retrospective review of 12 cases, delay to seeking treatment (of more than a day) was postulated as the cause of failure of closed reduction [81]. A common sequela of the injury is late instability, that may be treated with ligament reconstruction, as described by Eaton and Littler [85].

A retrospective review compared the results of two sequential groups of patients who had undergone either closed reduction and pinning, or early ligament reconstruction, after a traumatic TMC joint dislocation [84]. Of the eight patients in the former group, three developed recurrent instability and one had degenerative arthritis, requiring further operations. On that basis, the authors changed their treatment strategy regarding this injury, to early ligament reconstruction, which resulted in better preservation of range of motion and grip strength [84].

Clinical Pearl

Operative treatment

When exposing the MCP joint, use a longitudinal curvilinear incision in order to prevent a tender scar being placed directly over the joint.

When performing ORIF of metacarpal shaft fractures, use skin and fascia incisions that are offset to each other to facilitate tissue coverage of the hardware during closure.

When performing intramedullary pinning of metacarpal, use a mini-open approach to expose the base. Employ a drill sleeve and drill the entry hole in an oblique direction to facilitate passage of the wire.

For the ring finger, the wire is left exposed to reduce the risk of inadvertent extensor tendon injury. For the little finger, the wire can be buried to prevent it from being caught.

When fixing a Bennett's fracture, it is not necessary to pin the volar-ulnar fragment.

Rehabilitation

A postoperative rehabilitation regime is determined by the extent of bony and soft tissue injuries, stability of fracture, rigidity of fixation and patient's compliance. There should be effective communication between the surgeon and therapist

when prescribing post-operative rehabilitation. Early mobilisation is the key, as it promotes tendon gliding and decreases tissue oedema. However, this needs to be adapted to individual circumstances and deviation is sometimes necessary in patients with doubtful compliance.

Customised splints are useful to rest the joints between exercise sessions, protect the fracture and prevent contracture [3]. When there is associated tendon injury, dynamic splinting can be employed and the rehabilitation is best supervised by a specialist hand therapist.

Complications

The success of managing metacarpal injuries not only depends on careful assessment and instituting the appropriate treatment, but also relies on the subsequent monitoring for potential complications and having a strategy to deal with them, when they occur. Complications could arise as a result of the injury and/or the treatment given.

Infection

In a review of 146 open injuries to the hand, infection occurred in 11 % of the patients and all were in Gustillo type II or III injuries [86]. It reflected the severity of the initial insult and was associated with a poor outcome. Notably, preoperative wound cultures were of no value in predicting the risk of infection or the likely microorganisms. In another series of 200 open hand fractures, there were 9 wound infections and the incidence was associated with wound contamination, delay in treatment greater than 24 h, or systemic illness [87].

The use of Kirschner wires, leading to pin site infections could occur, but are rare if the wires are left in for less than 4 weeks.

Hardware Issues

Satisfactory results have been reported with the use of plates and screws as the internal fixation for metacarpal fractures [44]. However, others have noted significant complications. In a series of 41 metacarpal and 27 phalangeal fractures treated with minicondylar plates, 44 % developed hardware-related complications, 19 % had extensor lag and 12 % had infections [88]. The series, however, included 30 open fractures, 19 with severe soft tissue injury and 30 which required a bone graft. Another series of plate fixations reported a 42 % incidence of complications, including stiffness, malunion, non-union and tendon rupture [89]. In a series of 57 patients, managed with low profile miniplates, hardware removal was required in 8 patients and extensor tenolysis in 3 [90]. In general, metacarpal fractures tend to perform better than phalangeal fractures [88, 89].

Post-traumatic Osteoarthritis

A definitive correlation between articular incongruity and the development of post-traumatic arthritis is still debated. Moreover, the radiographic arthritis does not necessarily translate into clinical symptoms. Patients should be managed according to their symptoms and the impact on their function. In the case of thumb CMC joint, fusion is a viable option, particularly when there is associated instability.

Stiffness

Stiffness is determined by a combination of injury factors (severity of skeletal and soft tissue injury), patient factors (age, genetic composition) and treatment factors (duration and position of immobilisation, surgical dissection and implants used). The severity of soft tissue disruption should indicate the intensity of therapy required subsequently. In addition, one has to remain vigilant for the potential complication of complex regional pain syndrome, even after seemingly innocuous injury to the hand.

Bone Loss

Severe open injuries associated with segmental bone loss can be treated optimally with radical

debridement, immediate autogenous bone grafting, stable fixation and adequate soft tissue coverage [91, 92]. This treatment preserves normal bony length and allows intrinsic muscles to function under normal tension [92]. Additionally, it optimises venous and lymphatic return and eliminates dead space, thus reducing the risk of infection. Furthermore, immediate bony stability provides a platform for soft tissue reconstruction [91].

Fig. 10.12 Malunion of fifth metacarpal. Note the evidence of previous pinning

Malunion

Malunions of a boxer's fracture may be associated with an uncomfortable grip, due to the prominent metacarpal head in the palm, a painful dorsal bump and cosmetic appearance that is unacceptable to individual patients [11] (Fig. 10.12). In addition, pseudoclawing of the finger, as a result of dynamic imbalance of the surrounding soft tissues, is sometimes observed [93] (Fig. 10.13). However, the functional impact of malunion of the little finger metacarpal neck or shaft fractures, has been questioned [94]. Patients should thus be assessed and counselled carefully before embarking on corrective osteotomy.

Angulation deformity can be corrected with a closing wedge, opening wedge or a pivot osteotomy [11]. The effect on the soft tissues as a result of shortening (closing wedge) or lengthening (opening wedge) has to be taken into consideration. There is a general consensus that a rotational deformity is not acceptable and it may be corrected with a step osteotomy [95] or a rotational osteotomy [96].

Fig. 10.13 Appearance of pseudoclawing as a result of a boxer's fracture

Conclusions and Personal Views

Management of metacarpal fractures should focus on restoring the skeletal stability and hand function. The optimal treatment is determined by the extent of injury to the bone and surrounding soft tissues.

Current evidence would support that no specific immobilisation is required for the uncomplicated boxer's fracture (up to 70° angulation), as satisfactory results, without significant functional restriction, have been reported [25, 26, 28, 97]. Treatment strategies should thus be aimed at pain relief and early mobilisation.

For the metacarpal fracture with unacceptable deformity, our preferred technique is closed reduction and internal fixation, using a pre-bent, blunted 1.6 mm Kirschner wire. The wire is blunted to prevent inadvertent cortical penetration as the wire is inserted. A 30° bend is made at the terminal 1 cm of the wire to allow change of passage and engagement of the distal fragment. A gentler bow is created in the wire to effect three-point fixation, once within the canal. A small incision is made at the base of the metacarpal, on either its radial or ulnar corner. Blunt dissection is carried out, taking care not to damage the tendons and cutaneous nerves. A 2 mm drill or sharp awl is used to open the medullary canal. The wire is then mounted onto a Jacob's chuck and is passed in an antegrade fashion. A light mallet may be used to facilitate wire passage. At the fracture site the wire and handle can be rotated to help fracture reduction. Once in place, reduction and stability are assessed on fluoroscopy. Occasionally a second wire is required. The wire is left exposed to reduce the risk of inadvertent extensor tendon injury. However, the wire fixing the little finger metacarpal can be buried to prevent it from being caught. Pin sites should be checked regularly. The wire may be easily removed in the outpatient department, unless a significant bend was placed in the wire, when a general anaesthetic may be preferred.

For the Bennett's fracture, our preferred treatment is manipulation under anaesthesia and percutaneous pinning. Transarticular pinning may be combined with intermetacarpal pinning, so long as to achieve the aim of stable reduction. For the Rolando's fracture with sizeable fragments, open reduction and internal fixation is favoured; otherwise percutaneous pinning is preferred for more complex fractures. For the acute TMC joint dislocation, urgent reduction is indicated. If closed reduction is successful, a careful examination under anaesthesia is performed to assess the stability. If closed means fails, or if there is significant residual instability, open reduction with formal repair of the dorsoradial ligament is favoured. We recommend immobilisation of the thumb in pronation and extension, with a combination of cast and Kirschner wires, for 6 weeks. We reserve ligament reconstruction for the chronic cases, or those who subsequently develop symptomatic late instability.

Future Development

Problems associated with metallic hardware in the treatment of hand fractures have stimulated the development of bioabsorbable implants, including miniplates, rods, pins, tacks and screws [98]. Currently available devices are made of poly L-lactide (PLLA) and copolymers of polylactides (P(L/DL)LA) and polyglycolide (PLGA). As the biomechanical studies of the new materials demonstrate strength and stiffness comparable to traditional metallic implants [99], clinical studies showing satisfactory results with their usage are emerging [100]. Concerns with inflammatory reactions, which may occur with degradation of the early generation device, appear to have been resolved with modern self-reinforced implants [98]. Clinical application of bioabsorbable implants is likely to increase with further advances in bioengineering and related technology.

References

1. Court-Brown CM, Caesar B. Epidemiology of adult fractures: a review. Injury. 2006;37:691–7.
2. Lazar G, Schulter-Ellis FP. Intramedullary structure of human metacarpals. J Hand Surg Am. 1980;5:477–81.

3. Kozin SH, Thoder JJ, Lieberman G. Operative treatment of metacarpal and phalangeal shaft fractures. J Am Acad Orthop Surg. 2000;8:111–21.
4. Strauch RJ, Rosenwasser MP, Lunt JG. Metacarpal shaft fractures: the effect of shortening on the extensor tendon mechanism. J Hand Surg Am. 1998;23:519–23.
5. Al-Qattan MM. Outcome of conservative management of spiral/long oblique fractures of the metacarpal shaft of the fingers using a palmar wrist splint and immediate mobilisation of the fingers. J Hand Surg Eur Vol. 2008;33:723–7.
6. Low CK, Wong HC, Low YP, et al. A cadaver study of the effects of dorsal angulation and shortening of the metacarpal shaft on the extension and flexion force ratios of the index and little fingers. J Hand Surg Br. 1995;20:609–13.
7. Blair WF, Steyers CM. Techniques in hand surgery. Baltimore: Williams & Wilkins; 1996.
8. Birndorf MS, Daley R, Greenwald DP. Metacarpal fracture angulation decreases flexor mechanical efficiency in human hands. Plast Reconstr Surg. 1997;99:1079–83; discussion 84–5.
9. Royle SG. Rotational deformity following metacarpal fracture. J Hand Surg Br. 1990;15:124–5.
10. Lee SG, Jupiter JB. Phalangeal and metacarpal fractures of the hand. Hand Clin. 2000;16:323–32, vii.
11. Thurston AJ. Pivot osteotomy for the correction of malunion of metacarpal neck fractures. J Hand Surg Br. 1992;17:580–2.
12. Brewerton DA. A tangential radiographic projection for demonstrating involvement of metacarpal heads in rheumatoid arthritis. Br J Radiol. 1967;40:233–4.
13. Eyres KS, Allen TR. Skyline view of the metacarpal head in the assessment of human fight-bite injuries. J Hand Surg Br. 1993;18:43–4.
14. Henderson JJ, Arafa MA. Carpometacarpal dislocation. An easily missed diagnosis. J Bone Joint Surg Br. 1987;69:212–4.
15. Hodgson PD, Shewring DJ. The 'metacarpal cascade lines'; use in the diagnosis of dislocations of the carpometacarpal joints. J Hand Surg Eur Vol. 2007;32:277–81.
16. Mehara AK, Bhan S. Rotatory dislocation of the second carpometacarpal joint: case report. J Trauma. 1993;34:464–6.
17. Page SM, Stern PJ. Complications and range of motion following plate fixation of metacarpal and phalangeal fractures. J Hand Surg Am. 1998;23: 827–32.
18. McElfresh EC, Dobyns JH. Intra-articular metacarpal head fractures. J Hand Surg Am. 1983;8: 383–93.
19. Shewring DJ, Thomas RH. Collateral ligament avulsion fractures from the heads of the metacarpals of the fingers. J Hand Surg Br. 2006;31:537–41.
20. Anakwe RE, Aitken SA, Cowie JG, et al. The epidemiology of fractures of the hand and the influence of social deprivation. J Hand Surg Eur Vol. 2011;36:62–5.
21. Kilbourne BC. Management of complicated hand fractures. Surg Clin North Am. 1968;48:201–13.
22. Bloem JJ. The treatment and prognosis of uncomplicated dislocated fractures of the metacarpals and phalanges. Arch Chir Neerl. 1971;23:55–65.
23. Eichenholtz SN, Rizzo 3rd PC. Fracture of the neck of the fifth metacarpal bone – is over-treatment justified? JAMA. 1961;178:425–6.
24. Hunter JM, Cowen NJ. Fifth metacarpal fractures in a compensation clinic population. A report on one hundred and thirty-three cases. J Bone Joint Surg Am. 1970;52:1159–65.
25. Ford DJ, Ali MS, Steel WM. Fractures of the fifth metacarpal neck: is reduction or immobilisation necessary? J Hand Surg Br. 1989;14:165–7.
26. Statius Muller MG, Poolman RW, van Hoogstraten MJ, et al. Immediate mobilization gives good results in boxer's fractures with volar angulation up to 70 degrees: a prospective randomized trial comparing immediate mobilization with cast immobilization. Arch Orthop Trauma Surg. 2003;123:534–7.
27. Leung YL, Beredjiklian PK, Monaghan BA, et al. Radiographic assessment of small finger metacarpal neck fractures. J Hand Surg Am. 2002;27:443–8.
28. Breddam M, Hansen TB. Subcapital fractures of the fourth and fifth metacarpals treated without splinting and reposition. Scand J Plast Reconstr Surg Hand Surg. 1995;29:269–70.
29. Bansal R, Craigen MA. Fifth metacarpal neck fractures: is follow-up required? J Hand Surg Eur Vol. 2007;32:69–73.
30. Konradsen L, Nielsen PT, Albrecht-Beste E. Functional treatment of metacarpal fractures 100 randomized cases with or without fixation. Acta Orthop Scand. 1990;61:531–4.
31. Braakman M, Oderwald EE, Haentjens MH. Functional taping of fractures of the 5th metacarpal results in a quicker recovery. Injury. 1998;29:5–9.
32. Harding IJ, Parry D, Barrington RL. The use of a moulded metacarpal brace versus neighbour strapping for fractures of the little finger metacarpal neck. J Hand Surg Br. 2001;26:261–3.
33. Lowdon IM. Fractures of the metacarpal neck of the little finger. Injury. 1986;17:189–92.
34. Lamb DW, Abernethy PA, Raine PA. Unstable fractures of the metacarpals. A method of treatment by transverse wire fixation to intact metacarpals. Hand. 1973;5:43–8.
35. Galanakis I, Aligizakis A, Katonis P, et al. Treatment of closed unstable metacarpal fractures using percutaneous transverse fixation with Kirschner wires. J Trauma. 2003;55:509–13.
36. Foucher G. "Bouquet" osteosynthesis in metacarpal neck fractures: a series of 66 patients. J Hand Surg Am. 1995;20:S86–90.
37. Wong TC, Ip FK, Yeung SH. Comparison between percutaneous transverse fixation and intramedullary K-wires in treating closed fractures of the metacarpal neck of the little finger. J Hand Surg Eur Vol. 2006;31:61–5.

38. Winter M, Balaguer T, Bessiere C, et al. Surgical treatment of the boxer's fracture: transverse pinning versus intramedullary pinning. J Hand Surg Eur Vol. 2007;32:709–13.
39. Facca S, Ramdhian R, Pelissier A, et al. Fifth metacarpal neck fracture fixation: locking plate versus K-wire? Orthop Traumatol Surg Res. 2010;96:506–12.
40. Strub B, Schindele S, Sonderegger J, et al. Intramedullary splinting or conservative treatment for displaced fractures of the little finger metacarpal neck? A prospective study. J Hand Surg Eur Vol. 2010;35:725–9.
41. Viegas SF, Tencer A, Woodard P, et al. Functional bracing of fractures of the second through fifth metacarpals. J Hand Surg Am. 1987;12:139–43.
42. Geiger KR, Karpman RR. Necrosis of the skin over the metacarpal as a result of functional fracture-bracing. A report of three cases. J Bone Joint Surg Am. 1989;71:1199–202.
43. McMahon PJ, Woods DA, Burge PD. Initial treatment of closed metacarpal fractures. A controlled comparison of compression glove and splintage. J Hand Surg Br. 1994;19:597–600.
44. Bosscha K, Snellen JP. Internal fixation of metacarpal and phalangeal fractures with AO minifragment screws and plates: a prospective study. Injury. 1993;24:166–8.
45. Grundberg AB. Intramedullary fixation for fractures of the hand. J Hand Surg Am. 1981;6:568–73.
46. Al-Qattan MM. Metacarpal shaft fractures of the fingers: treatment with interosseous loop wire fixation and immediate postoperative finger mobilisation in a wrist splint. J Hand Surg Br. 2006;31:377–82.
47. Al-Qattan MM, Al-Lazzam A. Long oblique/spiral mid-shaft metacarpal fractures of the fingers: treatment with cerclage wire fixation and immediate post-operative finger mobilisation in a wrist splint. J Hand Surg Eur Vol. 2007;32:637–40.
48. Al-Qattan MM, Al-Zahrani K, Al-Arfaj N, et al. A modified technique of dental wire fixation for spiral/oblique metacarpal and phalangeal fractures of the fingers. J Hand Surg Eur Vol. 2010;35:325–6.
49. Manueddu CA, Della Santa D. Fasciculated intramedullary pinning of metacarpal fractures. J Hand Surg Br. 1996;21:230–6.
50. Faraj AA, Davis TR. Percutaneous intramedullary fixation of metacarpal shaft fractures. J Hand Surg Br. 1999;24:76–9.
51. Dickson RA. Rigid fixation of unstable metacarpal fractures using transverse K-wires bonded with acrylic resin. Hand. 1975;7:284–6.
52. Shehadi SI. External fixation of metacarpal and phalangeal fractures. J Hand Surg Am. 1991;16:544–50.
53. Gonzalez MH, Hall Jr RF. Intramedullary fixation of metacarpal and proximal phalangeal fractures of the hand. Clin Orthop Relat Res. 1996;327:47–54.
54. Nordyke MD, Lewis Jr RC, Janssen HF, et al. Biomechanical and clinical evaluation of the expandable intramedullary fixation device. J Hand Surg Am. 1988;13:128–34.
55. Ochman S, Doht S, Paletta J, et al. Comparison between locking and non-locking plates for fixation of metacarpal fractures in an animal model. J Hand Surg Am. 2010;35:597–603.
56. Ozer K, Gillani S, Williams A, et al. Comparison of intramedullary nailing versus plate-screw fixation of extra-articular metacarpal fractures. J Hand Surg Am. 2008;33:1724–31.
57. Liaw Y, Kalnins G, Kirsh G, et al. Combined fourth and fifth metacarpal fracture and fifth carpometacarpal joint dislocation. J Hand Surg Br. 1995;20:249–52.
58. Chong AK, Chew WY. An isolated ring finger metacarpal shaft fracture? – beware an associated little finger carpometacarpal joint dislocation. J Hand Surg Br. 2004;29:629–31.
59. Peterson P, Sacks S. Fracture-dislocation of the base of the fifth metacarpal associated with injury to the deep motor branch of the ulnar nerve: a case report. J Hand Surg Am. 1986;11:525–8.
60. O'Rourke PJ, Quinlan W. Fracture dislocation of the fifth metacarpal resulting in compression of the deep branch of the ulnar nerve. J Hand Surg Br. 1993;18:190–1.
61. Petrie PW, Lamb DW. Fracture-subluxation of base of fifth metacarpal. Hand. 1974;6:82–6.
62. Kjaer-Petersen K, Jurik AG, Petersen LK. Intra-articular fractures at the base of the fifth metacarpal. A clinical and radiographical study of 64 cases. J Hand Surg Br. 1992;17:144–7.
63. Kumar R, Malhotra R. Divergent fracture-dislocation of the second carpometacarpal joint and the three ulnar carpometacarpal joints. J Hand Surg Am. 2001;26:123–9.
64. Agarwal A, Agarwal R. An unusual farm injury: divergent carpometacarpal joint dislocations. J Hand Surg Br. 2005;30:633–4.
65. Garcia-Elias M, Bishop AT, Dobyns JH, et al. Transcarpal carpometacarpal dislocations, excluding the thumb. J Hand Surg Am. 1990;15:531–40.
66. Lundeen JM, Shin AY. Clinical results of intraarticular fractures of the base of the fifth metacarpal treated by closed reduction and cast immobilization. J Hand Surg Br. 2000;25:258–61.
67. Day CS, Stern PJ. Fractures of the metacarpals and phalanges. In: Wolfe SC, Hotchkiss RN, Pederson WC, Kozin SH, editors. Green's operative hand surgery, vol. 1. 6th ed. Philadelphia: Churchill Livingstone; 2011.
68. Bennett EI. Fractures of the metacarpal bones. Dublin J Med Sci. 1882;73:72–5.
69. Henry MH. Fractures and dislocations of the hand. In: Bucholz RW, Heckman JD, Court-Brown CM, editors. Rockwood and Green's fractures in adults, vol. 1. 6th ed. Philadelphia: Lippincott Williams & Wilkins; 2006. p. 823–55.
70. Wagner CJ. Method of treatment of Bennett's fracture dislocation. Am J Surg. 1950;80:230–1.
71. Johnson EC. Fracture of the base of the thumb. A new method of fixation. JAMA. 1944;126:27–8.

72. Cannon SR, Dowd GS, Williams DH, et al. A long-term study following Bennett's fracture. J Hand Surg Br. 1986;11:426–31.
73. Kjaer-Petersen K, Langhoff O, Andersen K. Bennett's fracture. J Hand Surg Br. 1990;15:58–61.
74. Livesley PJ. The conservative management of Bennett's fracture-dislocation: a 26-year follow-up. J Hand Surg Br. 1990;15:291–4.
75. Lutz M, Sailer R, Zimmermann R, et al. Closed reduction transarticular Kirschner wire fixation versus open reduction internal fixation in the treatment of Bennett's fracture dislocation. J Hand Surg Br. 2003;28:142–7.
76. Rolando S. Fracture de la base du premier metacarpien et principa ement sur une variété non encore décrite. Presse Med. 1910;18:303.
77. Langhoff O, Andersen K, Kjaer-Petersen K. Rolando's fracture. J Hand Surg Br. 1991;16:454–9.
78. Gelberman RH, Vance RM, Zakaib GS. Fractures at the base of the thumb: treatment with oblique traction. J Bone Joint Surg Am. 1979;61:260–2.
79. Buchler U, McCollam SM, Oppikofer C. Comminuted fractures of the basilar joint of the thumb: combined treatment by external fixation, limited internal fixation, and bone grafting. J Hand Surg Am. 1991;16:556–60.
80. Foster RJ, Hastings 2nd H. Treatment of Bennett, Rolando, and vertical intraarticular trapezial fractures. Clin Orthop Relat Res. 1987;214:121–9.
81. Watt N, Hooper G. Dislocation of the trapeziometacarpal joint. J Hand Surg Br. 1987;12:242–5.
82. Strauch RJ, Behrman MJ, Rosenwasser MP. Acute dislocation of the carpometacarpal joint of the thumb: an anatomic and cadaver study. J Hand Surg Am. 1994;19:93–8.
83. Shah J, Patel M. Dislocation of the carpometacarpal joint of the thumb. A report of four cases. Clin Orthop Relat Res. 1983;175:166–9.
84. Simonian PT, Trumble TE. Traumatic dislocation of the thumb carpometacarpal joint: early ligamentous reconstruction versus closed reduction and pinning. J Hand Surg Am. 1996;21:802–6.
85. Eaton RG, Littler JW. Ligament reconstruction for the painful thumb carpometacarpal joint. J Bone Joint Surg Am. 1973;55:1655–66.
86. McLain RF, Steyers C, Stoddard M. Infections in open fractures of the hand. J Hand Surg Am. 1991;16:108–12.
87. Swanson TV, Szabo RM, Anderson DD. Open hand fractures: prognosis and classification. J Hand Surg Am. 1991;16:101–7.
88. Ouellette EA, Freeland AE. Use of the minicondylar plate in metacarpal and phalangeal fractures. Clin Orthop Relat Res. 1996;327:38–46.
89. Stern PJ, Wieser MJ, Reilly DG. Complications of plate fixation in the hand skeleton. Clin Orthop Relat Res. 1987;214:59–65.
90. O'Sullivan ST, Limantzakis G, Kay SP. The role of low-profile titanium miniplates in emergency and elective hand surgery. J Hand Surg Br. 1999;24:347–9.
91. Saint-Cyr M, Miranda D, Gonzalez R, et al. Immediate corticocancellous bone autografting in segmental bone defects of the hand. J Hand Surg Br. 2006;31:168–77.
92. Stahl S, Lerner A, Kaufman T. Immediate autografting of bone in open fractures with bone loss of the hand: a preliminary report. Case reports. Scand J Plast Reconstr Surg Hand Surg. 1999;33:117–22.
93. Flatt AE. Closed and open fractures of the hand. Fundamentals of management. Postgrad Med. 1966;39:17–26.
94. Westbrook AP, Davis TR, Armstrong D, et al. The clinical significance of malunion of fractures of the neck and shaft of the little finger metacarpal. J Hand Surg Eur Vol. 2008;33:732–9.
95. Manktelow RT, Mahoney JL. Step osteotomy: a precise rotation osteotomy to correct scissoring deformities of the fingers. Plast Reconstr Surg. 1981;68:571–6.
96. Gross MS, Gelberman RH. Metacarpal rotational osteotomy. J Hand Surg Am. 1985;10:105–8.
97. Kuokkanen HO, Mulari-Keranen SK, Niskanen RO, et al. Treatment of subcapital fractures of the fifth metacarpal bone: a prospective randomised comparison between functional treatment and reposition and splinting. Scand J Plast Reconstr Surg Hand Surg. 1999;33:315–7.
98. Waris E, Ashammakhi N, Kaarela O, et al. Use of bioabsorbable osteofixation devices in the hand. J Hand Surg Br. 2004;29:590–8.
99. Bozic KJ, Perez LE, Wilson DR, et al. Mechanical testing of bioresorbable implants for use in metacarpal fracture fixation. J Hand Surg Am. 2001;26:755–61.
100. Jensen CH, Jensen CM. Biodegradable pins versus Kirschner wires in hand surgery. J Hand Surg Br. 1996;21:507–10.

Fractures of Carpal Bones Other than the Scaphoid

11

Matthew Nixon and Ian A. Trail

Keywords
Fracture • Avascular necrosis • Greater arc • Lunate • Triquetrum • Pisiform • Capitate • Hamate • Trapexium • Trapezoid

Introduction

Isolated fractures of non-scaphoid carpal fractures are rare, with an estimated incidence of around 1.1 % of all fractures and 18 % of hand fractures [1]. However, as they are frequently missed on routine plain radiographs, their true incidence may be higher than traditionally reported. Fractures are more common in the triquetrum, trapezium and hamate than in the capitate, trapezoid and pisiform. The incidence of lunate fractures is hard to estimate, due to the similar nature of Kienböck's, which frequently presents as osteonecrosis and a fracture.

The largest series of carpal fractures was described by Garcia-Elias, who noted that, other than the scaphoid, the triquetrum and trapezium were the most commonly injured carpus [2]. He also noted a number of characteristics of carpal fractures, including the tendency to occur in young, functionally demanding patients. Because of the small nature of the bones they are often difficult to reduce closed, are often intra-articular and easily missed on initial presentation. They are often a result of high energy trauma and so are frequently associated with ligamentous injuries and wrist instability. Their proximity to adjacent neurovascular structures means these structures are also at risk of injury.

Mechanism

These fractures usually occur as a result of a fall onto an outstretched hand, which initially transmits forces onto the distal carpal row and then the proximal carpal row, with increased force the wrist is pushed into hyperextension. Hyperextension is limited by the volar ligaments and these can become disrupted, particularly around the lunate, resulting in a fracture dislocation injury which may either be a mainly osseous injury (greater arc peri-lunate dislocation) or purely ligamentous injury (lesser arc peri-lunate

M. Nixon, MD, FRCS (Orth) (✉)
Department of Orthopaedics,
Countess of Chester Hospital,
Liverpool Road, Chester CH2 1UY, UK
e-mail: Matthew.Nixon@nhs.net

I.A. Trail, MBCHB, MD, FRCS (Edin), FRCS (Lon), ECFMG
Department of Orthopaedics,
Wrightington Hospital, Hall Lane, Appley Bridge,
Wigan, Lancashire WN6 9EP, UK
e-mail: upperlimb@wrightington.org.uk

I.A. Trail, A.N.M. Fleming (eds.), *Disorders of the Hand: Volume 1: Hand Injuries*,
DOI 10.1007/978-1-4471-6554-5_11, © Springer-Verlag London 2015

dislocation). With a dorsiflexion mechanism, fractures can occur either as a peri-lunate greater arc (described below), as an avulsion of the ligaments from the volar surface, or as a dorsal impaction of the carpus on the radius. Avulsion fractures can occur elsewhere, such as at the hook of the hamate, the pisiform and the trapezial ridge. Finally, axial loading forces (such as punching a wall with a clenched fist) can lead to longitudinal fractures with disruption of the carpus on either side of the capitate.

Clinical Pearls: Epidemiology

1.1 % of all fractures
True incidence probably higher due to injuries being easily missed
High energy injuries in young patients
High association with ligamentous injuries
Lunate, capitate, trapezoid and hamate are at particular risk of avascular necrosis

Anatomy

The intrinsic blood supply to the wrist is important in understanding healing potential of carpal bone fractures. On the volar surface there are four anastomotic arterial arches between the radial and ulnar arteries. These are the radio-carpal, inter-carpal, superficial palmar and deep palmar arches. These are further connected to three dorsal arches via the dorsal radio-carpal, dorsal inter-carpal and basal metacarpal arches.

Of these vessels, the palmar radio-carpal and palmar inter-carpal vessels are important in the supply to the lunate and triquetrum. The recurrent radial and ulnar branches of the deep palmar arch supply the distal carpal row. The capitate and 20 % of lunates have only a single nutrient vessel and so are at particular risk of post-traumatic osteonecrosis [3]. The trapezoid and hamate lack comprehensive interosseous anastomotic networks and so are also at risk of osteonecrosis. In contrast the trapezium, triquetrum and pisiform have better interosseous networks and so are less likely to develop osteonecrosis [4].

Greater-Arc Injuries

This is a variation of peri-lunate dislocation that involves the peri-lunate osseous structures – classically involving fractures to the scaphoid, capitate, hamate and triquetrum (as opposed to lesser arc injuries that involve injury to the peri-lunate ligamentous structures). Treatment involves open reduction and internal fixation of each bone, usually with good results [5].

Lunate Fractures

Mechanism

Because it is well encapsulated in the radial lunate fossa, the lunate is rarely injured in isolation. Lunate fractures may occur after a fall onto an outstretched hand, or be the result of damage from a lunate dislocation. With a fall, the capitate head is forced into the lunate fossa, resulting in either a longitudinal split of the lunate (Fig. 11.1), or the volar lip of the lunate being sheared off as the head of the capitate dislocates. In hyperextension, the dorsal lip of the lunate can be crushed between the capitate and dorsal rim of the radius. Occasionally, the lunate may fracture as part of a peri-lunate dislocation [6, 7].

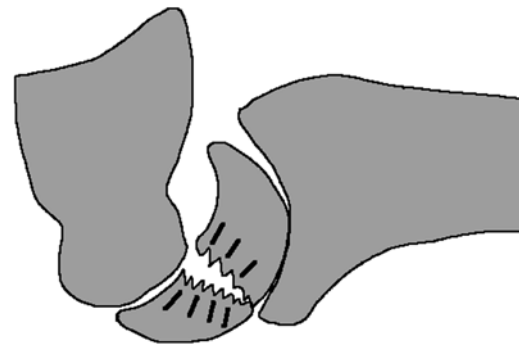


Fig. 11.1 Forced hyperextension may result in impaction of the capitate on the volar lip of the lunate, resulting in a shear fracture of the lunate body

Diagnosis

Patients complain of pain and swelling over the lunate, just distal to Lister's tubercle. Routine radiographs or CT scanning are used to confirm the diagnosis. Diagnostic difficulty arises in the presence of a fracture in a bone with established osteonecrosis – as this constitutes Kienböcks disease (discussed in detail elsewhere). Radiographic factors suggestive of an injury being due to Keinböck's rather than acute trauma include the involvement of the sub-chondral region of the proximal articular surface and the presence of a crescent sign.

Treatment

Because of the precarious blood supply, left untreated, lunate fractures have a high incidence of non-union and development of osteonecrosis (secondary Kienböck's disease). Undisplaced injuries can be managed with a short period of immobilisation. Flexion of the metacarpophalangeal joints within the cast may reduce the compression of the capitate on the lunate and so prevent fragmentation.

Displaced fractures need open reduction and either k-wire or screw fixation. This is usually done through a dorsal approach between the third and fourth extensor compartments. This allows good access to lunate body and dorsal rim fractures. Avulsion of the scapho-lunate ligament from the dorsal surface can also be repaired through this approach, although may also be accessed arthroscopically and repaired with a small screw or suture anchors to prevent carpal instability [8].

Volar ridge rim fractures can be difficult to approach and as the fragment is often small, and difficult to fix. In these cases, stabilisation of the capito-lunate joint with a K-wire, to prevent volar subluxation, may be appropriate. If required, volar fragments can be exposed via an extended carpal tunnel approach, along the ulnar side of Palmaris Longus to protect the median nerve.

Clinical Pearls: Lunate Injuries

May precede or result from Keinbocks disease

High risk of avascular necrosis

Body fractures best accessed from the dorsum of the wrist

Volar rim fractures at risk of capito-lunate instability

Triquetrum Fractures

Incidence

The triquetrum is around the third most commonly fractured carpal bone and a number of distinct fracture patterns are encountered, as a result of different mechanisms.

The most common fracture type is a dorsal cortical avulsion fracture, which may be due to a variety of mechanisms [9]. They usually occur due to forced palmar-flexion of the wrist, which results in avulsion of the dorsal radiotriquetral and triquetroscaphoid ligaments from the dorsum of the triquetrum. Impingement of the distal ulna upon the dorsum of the carpus (through forced ulnar deviation) can lead to a similar type of injury and it has been shown that ulnar length is higher in patients with such fractures, in keeping with the theory that this is a shear fracture of the ulnar on the triquetrum [10]. Dorsal avulsion fractures may also occur with extremes of hyper-extension and with this mechanism, it is thought to be hamate impinging on the triquetrum that causes the fracture.

Triquetral body fractures are usually high energy injuries, occurring in extreme dorsiflexion of the wrist, or as part of a greater arc peri-lunate injury. Other causes of body fractures are extreme axial loading, which can lead to impaction of the ulna on the triquetrum and direct blows to the wrist. These fractures may be in a number of planes.

Fig. 11.2 CT reconstruction image demonstrating SL avulsion injury with dorsal avulsion of DRC ligament from triquetrum (Images provided from Adam Watts, with permission)

Avulsion fractures of the volar ligamentous structures (in particular the ulno-capitate, ulno-triquetral or luno-triquetral ligaments) are usually more serious than dorsal avulsion fractures and tend to occur with wrist hyper-extension in radial deviation (Fig. 11.2).

Diagnosis

There is usually swelling and tenderness in the ulnar side of the wrist, localised around the triquetrum, which can be more easily assessed in radial deviation of the wrist. As triquetral fractures can occur as part of greater arc injuries, the wrist should be examined for associated instability.

Fractures of the triquetral body can normally be visualised on the AP projection of standard wrist views, with dorsal chip fractures being seen on the lateral projection. Volar avulsion injuries may be better seen in radial deviation views as this unmasks the triquetrum. CT scan may be required to confirm the diagnosis or to properly visualise volar avulsion injuries.

Treatment

Dorsal cortical avulsion fractures require only a short (2–3 weeks) period of immobilisation until swelling and discomfort subside. Patients can expect a good functional outcome, and if non- or fibrous union develop, this can be successfully treated with excision of the fragment [11].

Fractures of the body are usually due to a high energy injury with associated soft tissue injury. Isolated and undisplaced body fractures rarely progress to non-union and just need plaster immobilisation for 4–6 weeks. Displaced body fractures need open reduction, repair of the damaged soft tissues and fixation with either K-wires or screws.

Volar avulsion injuries represent a significant injury to the luno-triquetral ligamentous complex. The priority in such injuries is to achieve wrist stability through ligamentous repair or reconstruction or luno-triquetral arthrodesis, depending on the timing of presentation.

Complications

Post operative complications include continued pain, non/mal-union and post-traumatic osteoarthritis. Symptomatic non-union of peripheral chip fractures is rare, and can be treated with surgical excision, and larger fragments by bone graft fixation [12, 13].

Clinical Pearls: Triquetrum Fractures

Dorsal cortical avulsions may be due to
- Dorsal ligament avulsion, ulnar or hamate impingement
- These usually heal well with a short period of immobilisation

Avulsion of the volar ligaments are more serious injuries leading to instability

Body fractures are high energy and often part of greater arc injuries requiring fixation

Pisiform Fractures

Anatomy

The pisiform lies on the ulnar border of Guyon's canal, which carries the ulnar artery and nerve. It articulates with the concave facet of the triquetrum and has numerous soft tissue attachments. These include the FCU tendon (in which it is ensheathed), the origin of the abductor digiti minimi muscle, the piso-metacarpal and piso-hamate ligaments and the flexor and extensor retinacula.

Mechanism

Fractures usually occur due to a fall onto an outstretched hand, resulting in either a direct blow to the pisiform or an avulsion of one of its soft tissue attachments.

A number of fracture patterns are encountered. Transverse body fractures are effectively a rupture of the FCU tendon through the pisiform and are a consequence of sudden contracture of the muscle, whilst the pisiform is locked into the triquetrum – as may occur during a fall onto an outstretched hand. A similar mechanism may lead to a parasagittal fracture, in which the ulnar rim of the pisiform is damaged but the FCU tendon remains in continuity. Comminuted fractures usually are the result of a direct blow to the hypothenar region.

Diagnosis

Patients typically present with tenderness over the pisiform, with pain on resisted wrist flexion. The integrity of the ulnar nerve should be assessed, due to its proximity to the pisiform [14]. Standard wrist views are usually sufficient to visualise these injuries. The piso-triquetral joint may be better seen with 30° of wrist flexion or utilising the carpal tunnel view. CT scans best visualise these injuries.

Treatment

These injuries are nearly always treated conservatively with cast immobilisation for 4–6 weeks, with most healing by bony or fibrous union.

A number of complications may occur following pisiform fractures. Acutely these injuries may be associated with ulnar neuropathies. These are normally neuropraxias and resolve spontaneously, however exploration is indicated if symptoms persist for more than 3 months.

Causes of chronic pain include malunion, (which can lead to post-traumatic osteoarthritis in the piso-triquetral joint), calcific tendonitis of the FCU tendon and fracture non-union (particularly in comminuted or widely separated transverse fractures).

Treatment of chronic pisiform related pain is by pisiformectomy (in which the bone is shelled out of the FCU tendon) which usually provides complete pain relief with minimal loss of movement or strength [15, 16] This is achieved via a Z incision, with the transverse limb over the proximal palmar crease.

Clinical Pearls: Pisiform Fractures

Forms the attachment of several muscles and ligaments

Therefore at risk of non-union

Pisiform excision is usually successful at relieving chronic non-union pain

Hamate Fractures

Anatomy

The hook of the hamate projects from the base of the bone into the hypothenar eminence and is the site of origin of numerous intrinsic muscles and ligaments. These features leave it at particular risk of injury. Beneath the hook lie the flexor tendons to the ring and little fingers and these may also be affected by such fractures. It is located distally and radially to the pisiform, forming the lateral border of Guyon's canal. The usual blood supply to the hamate is via dorsal and palmar vessels creating an anastomosis in the hamate body and a separate nutrient vessel supplying the hook which enters at the level of Guyon's canal [17]. Up to a third of patients lack the hook nutrient vessel and so are at increased risk of non-union and osteonecrosis.

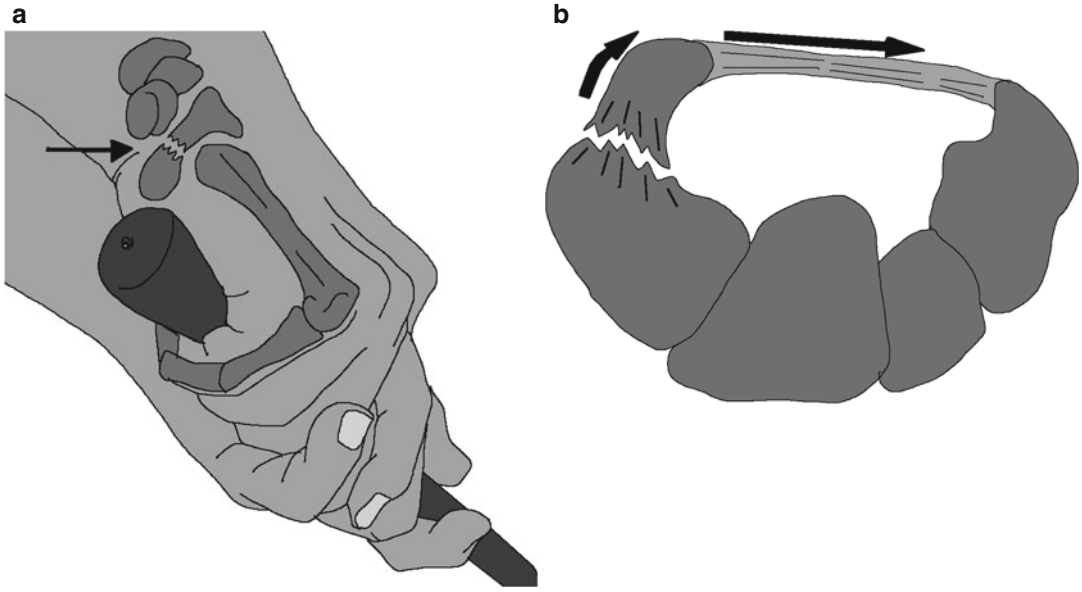


Fig. 11.3 Demonstrating mechanism of hook of hamate avulsion fracture. (**a**) Mechanism of action for hook of hamate fracture with forceful flexor tendon contracture whilst tightly griping an object. The *arrow* highlights the location of the fracture. (**b**) Cross section diagram of the forces acting upon the hook of the hamate with *arrow* highlighting the direction of tensile forces

Fractures should be distinguished from bipartite hamate, which will have a smooth cortical surface.

Mechanism

Fractures of the hook of the hamate are the most common fracture of this bone and usually the result of indirect trauma (for example the mis-hitting of a golf club into the ground, thus rapidly transferring the energy from the golf club onto the transverse carpal ligament and subsequently onto the hook of the hamate) [18]. Fractures may be caused by an acute injury or chronic over-use [19]. An alternative mechanism is a direct blow to the hypothenar region. Such injuries may frequently present as chronic ulnar sided wrist pain [20]. Fractures of the hook of the hamate have been classified into tip avulsion, base and waist fractures (Figs. 11.3, 11.4, and 11.5) [21].

Fractures of the hamate body are rarer. Axial loading of the fourth or fifth carpo-metacarpal joint may result in a CMC dislocation associated with a dorsal shear fracture of the hamate body, which is often intra-articular. This joint normally has a large degree of motion and is important for gripping. Other causes of body fractures include perilunate fracture dislocations (causing proximal pole fractures), direct blows to the ulnar side (causing fractures of the medial tuberosity) and high energy trauma (which can cause a number of fracture patterns, including sagittal oblique fractures).

Diagnosis

Pain is usually worse on the ulnar side of the palm, accentuated by gripping and axial loading of the ring and little fingers, with point tenderness of the hook 1–2 cm distal to the pisiform. As they are often missed acutely, there is frequently a long history of pain. Due to the proximity to the median and ulnar nerves, parasthesia in these nerves or motor weakness of the intrinsics may also be present. Grip strength may be reduced due to pain or weakness of the intrinsics secondary to neurological injury. An Allen test should assess the patency of the radial and ulnar arteries. The flexor tendons to the little and ring fingers run under the hook and if irritated there is pain on resisted finger flexion, worse with ulnar deviation.

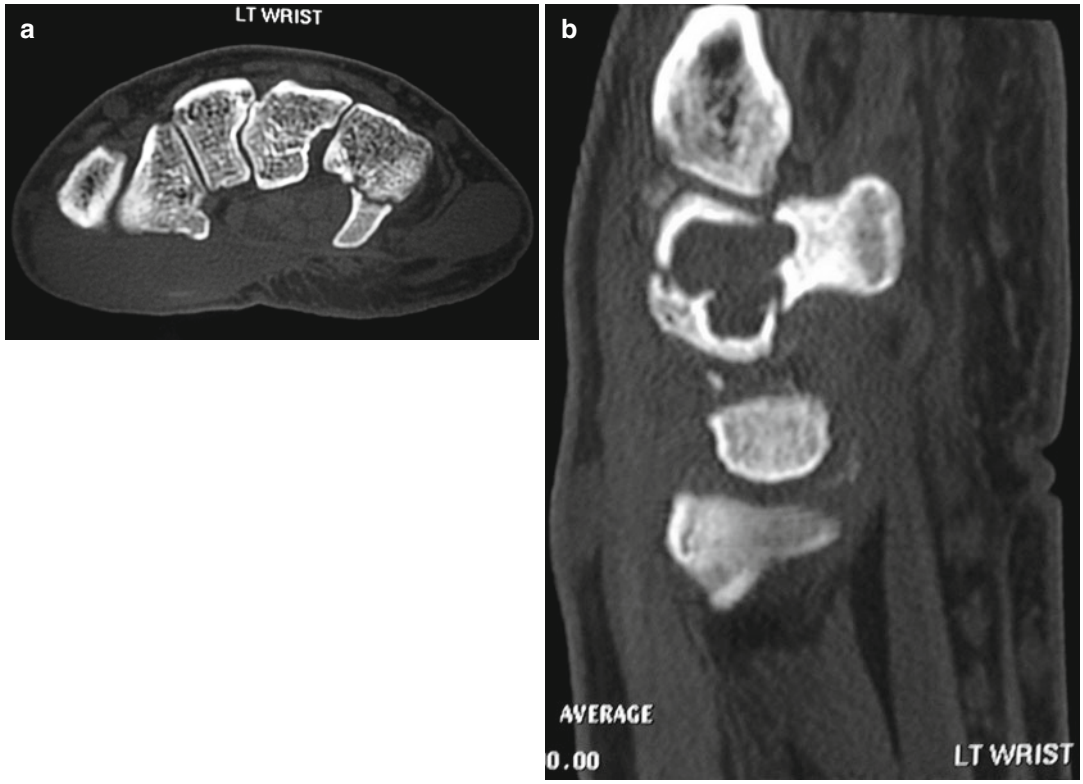


Fig. 11.4 Demonstrating CT images of a hook of hamate fracture with associated cyst (Images provided from Adam Watts, with permission)

If this is chronic, it may present as a tendon rupture to these fingers [22].

Fractures of the hamate are difficult to detect on routine wrist views and are frequently missed initially. A 45° pronation view is useful to assess dorsal hamatometacarpal fracture dislocations. 30° tilted lateral or carpal tunnel views are useful to assess the hook of the hamate [5]. CT scanning is useful for further evaluation and for intra-articular injuries.

Treatment

The difficulty in acute management of hook of the hamate injuries is detection. However, if appropriately identified and immobilised, most heal well with conservative treatment. Because of delayed presentation, however, non-union is relatively common and is usually the result of too early mobilisation. Because the hook of the hamate is a point of attachment for the hypothenar muscles, the action of flexor digiti minimi brevis and opponens digiti minimi cause tension at the fracture site, causing non-union.

The management of displaced fractures or non-unions is either excision or fixation of the fragment. Excision is effective in preventing tenosynovitis and allows most patients to return to sporting activities [23–25]. This is performed by identifying the ulnar neurovascular structures and tracing them as they enter Guyon's canal. Particular care should be taken to protect the dorsal motor branch, which lies close to the base of the hook at the fracture site. Damage to this is the most common complication of this surgery [24, 26]. After identification, the fragment can be excised and the base smoothed down to prevent irritation.

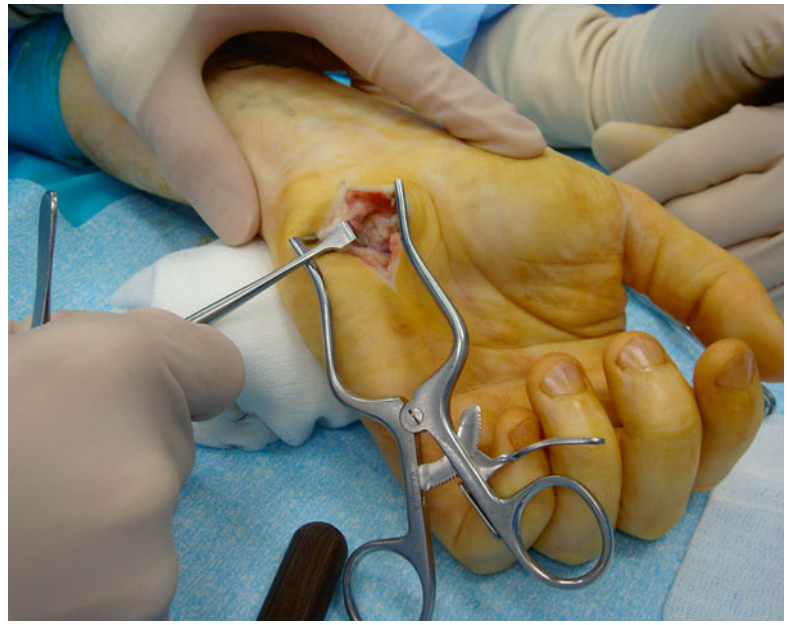


Fig. 11.5 Surgical exposure to the hook of the hamate (Images provided from Adam Watts, with permission)

An alternative treatment is fixation of the fragment, which theoretically maintains the tension in the flexor tendons and improves function when compared to excision [27, 28]. However, this is technically demanding and carries the risk of future tendon problems. The hook is usually approached from the volar wrist, but may alternatively be lagged using a dorsal percuataneous technique [25, 27 and 29].

Fractures to the hamate body can be approached dorsally between the fourth and fifth extensor tendons. Associated dislocation of the CMC joint is stabilised with k-wires and the hamate fracture can then be fixed using small plates and screws. Awareness of the proximity of the dorsal motor branch of the ulnar nerve on the underside of the hamate is important when fixing these fractures.

Avascular necrosis may occur following fractures, but with its dual blood supply, these are rare if fractures are adequately immobilised. As the hook acts as a fulcrum for the flexor tendons of the little finger, malunion and surface irregularities can lead to flexor tenosynovitis in up to 25 % of patients, which can subsequently lead to tendon attrition or rupture [30]. It may also lead to ulnar neuritis or ulnar artery occlusion. These complications can also be treated by decompression through excision of the fragment and smoothing down of the irregular surface.

Clinical Pearls: Hamate Fractures

The hook of the hamate
- provides the attachment of numerous ligaments and tendons
- has a poor blood supply with a high risk of developing AVN
- may be injured via indirect traction via the transverse carpal ligament
- if missed, injuries may be excised or repaired
- may be associated with flexor tendon or neurovascular injury

Hamate body fractures
- are rarer and usually due to high energy injuries

Capitate Fractures

Incidence

Injuries are rare because of the protected position of the capitate and they may either be isolated or, more usually, associated with scaphoid or other carpal injuries. The head of the capitate is almost completely covered in cartilage, highly mobile and supplied by a retrograde end-arteriole vessel. It therefore has many similarities to the scaphoid and so has similar risks of nonunion and osteonecrosis.

Mechanism

Scaphocapitate syndrome is a scaphoid fracture together with a transverse proximal pole injury of the capitate that rotates 180°, thought to be part of an incomplete greater arc injury [31]. Because of the high association with other injuries, the wrist needs careful evaluation for instability. The usual mechanism is a fall on a dorsiflexed wrist with radial deviation [32]. This initially exerts a force on the scaphoid, which usually fractures first, but with increasing dorsiflexion the head of the capitate strikes the dorsal rim of the radius, fractures and may slip back into the lunate fossa rotated 180° with the articular surface of the lunate fossa facing upwards into the fracture site (Fig. 11.6) [33].

The fractures may be classified into being in the proximal, middle or distal third of the bone. Osteochondral injuries to the capitate may be encountered during perilunate dislocations.

Imaging

Scaphocapitate injuries are frequently detected on standard plain films. However, a high index of suspicion is needed, as in the presence of an obvious scaphoid fracture, the capitate fracture can be overlooked. Isolated injuries often require CT scanning to establish the diagnosis.

Treatment

Capitate fractures that are stable, isolated and undisplaced can be treated with cast immobilisation. Isolated displaced fractures need reduction and wire or headless screw fixation, which may be arthroscopically assisted. This may be complicated if the proximal fragment has rotated 180°.

Scaphocapitate injuries are usually very unstable and require both injuries to be addressed simultaneously. The capitate is best accessed through a dorsal incision, which can also be used for the scaphoid, or this can be addressed through a separate volar approach. Following open reduction, wrist stability can usually be restored with either K-wire or screw internal fixation.

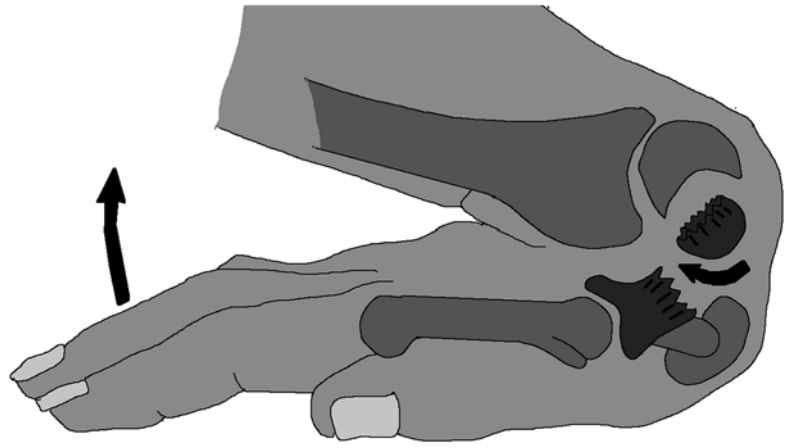


Fig. 11.6 Demonstrating the 180° rotation of the proximal capitate fragment, *arrow* highlights direction of force acting upon the hand and wrist

AVN is a relatively common complication of capitate fractures, and can affect the proximal pole and/or distal body [34]. The vascularity of chronic pseudarthrosis can be assessed using MRI and corticocancellous bone graft used to promote union and restore the height of the capitate to restore wrist biomechanics [35].

Clinical Pearls: Capitate Fractures

Are often associated with scaphoid fractures

Capitate head fractures may rotate 180° and require open reduction

These injuries are at risk of AVN and chronic wrist instability

Trapezium Fractures

Incidence

Fractures of the trapezium are usually associated with fractures of either the first metacarpal or the radius. They are rare, constituting between 1 and 5 % of all carpal fractures and the largest series being 34 cases [36].

Mechanism

The protected position of the trapezium under the thumb protects it from direct blows, and most injuries are indirect as a consequence of axial loads upon the thumb. This can lead to a shear fracture through the radial side of the body of the trapezium, which can subsequently displace proximally with the attached metacarpal, in a similar fashion to a Bennett's fracture [37]. Trapezial ridge fractures may occur as a consequence of direct trauma to the base of the ridge (type 1) or avulsion of the transverse carpal ligament from the tip of the ridge (type 2) [38].

Diagnosis

These injuries usually present with swelling and tenderness at the first carpometacarpal (CMC) joint, with associated painful/weakened pinch strength. Often these injuries can be detected with standard wrist AP, true lateral (Roberts) or Betts view (with the beam directed towards the CMC joint with the wrist pronated). Carpal tunnel views may help assess trapezial ridge fractures. CT scanning is frequently used.

Treatment

Most fractures are the result of relatively high energy and so even non displaced body fractures need close monitoring. Immobilisation is with a thumb spica for 4 weeks. Articular displacement or subluxation of the CMC joint are indications for fixation of trapezial body fractures (achieved with either K-wires or compression screws). The fracture can be accessed via a volar approach, with elevation of the thenar muscles and protection of the radial artery. Percutaneous fixation may be augmented by arthroscopic manipulation of the fracture [39]. Bony fragments may alternatively be excised, which can be combined with FCR beak ligament reconstruction if the CMC joint is subsequently unstable.

Trapezial ridge fractures occurring at the base require either immobilisation or stabilisation, depending on the degree of displacement. Ridge tip fractures may be treated by excision if symptomatic.

Complications

Continued pain frequently due to post traumatic osteoarthritis may require treatment as for first CMC arthritis. Damage to the radial artery and sensory branches of the radial nerve may occur after fixation. Finally, the FCR tendon is prone to developing tendonitis, as it rubs against

mal-united fractures, in a fashion similar to that described for the flexor tendons under the hook of the hamate. In chronic cases, this can be addressed by excision of the fracture fragment.

Trapezoid Fractures

The trapezoid is a wedge shaped bone with strong ligamentous attachments and as such fractures are extremely rare, with an incidence of less than 1 % of carpal fractures. Isolated fractures are even more unusual, with less than 20 cases reported [2].

The usual mechanism of injury is an axial load on the index metacarpal, resulting in a sagittal shear fracture or dorsal avulsion of the trapezoid, frequently associated with dislocation of index CMC joint [39].

As well as regional pain and swelling, tenderness may be elicited on movement of the index metacarpal. Routine plain films usually detect these injuries, although CT is frequently required to fully evaluate them [40].

Undisplaced fractures can be treated conservatively in a cast for around 4 weeks. Displaced injuries require open reduction and K-wire fixation. Painful mal- or non-unions are best treated with carpo-metacarpal arthrodesis, as excision may lead to proximal migration of the index finger [41].

Clinical Pearls: Trapezium and Trapezoid Fractures

The trapezium may fracture at the ridge or the body

May lead to CMC arthritis or chronic FCR tendonitis

Trapezoid fractures are very rare and are usually the consequence of index CMCJ dislocation

Summary

Although rare, fractures of the carpal bones are probably under diagnosed. They are often caused by high energy trauma and so are frequently associated with ligamentous or other bony injuries. They may also present late as a cause of persistent wrist pain following trauma or repetitive use. Management involves having a high index of suspicion and appropriate imaging to make the appropriate diagnosis, with definitive treatment requiring a combination of surgical and non-surgical modalities.

References

1. Larsen CF, Brøndum V, Skov O. Epidemiology of scaphoid fractures in Odense, Denmark. Acta Orthop Scand. 1992;63(2):216–8.
2. Garcia-Elias M. Carpal bone fractures (excluding the scaphoid). In: Watson HK, Weinberg J, editors. The wrist. Philadelphia: Lippincott Williams & Wilkins; 2001. p. 174–81.
3. Cooney WP, Linscheid RL, Dobyns JH. Fractures and dislocations of the wrist. In: Rockwood CA, Green DP, Bucholz RW, Heckman JD, editors. Rockwood and Green's fractures in adults, vol. 1. 4th ed. Philadelphia: Lippincott-Raven; 1996. p. 745–867.
4. Gelberman RH, Panagis JS, Taleisnik J, Baumgaertner M. The arterial anatomy of the human carpus. Part I: the extraosseous vascularity. J Hand Surg Am. 1983;8(4):367–75.
5. Akahane M, Ono H, Sada M, Saitoh M. Fracture of hamate hook – diagnosis by the hamate hook lateral view. Hand Surg. 2000;5(2):131–7.
6. Moneim MS. Management of greater arc carpal fractures. Hand Clin. 1988;4(3):457–67. Review.
7. Briseño MR, Yao J. Lunate fractures in the face of a perilunate injury: an uncommon and easily missed injury pattern. J Hand Surg Am. 2012;37(1):63–7. Epub 2011 Nov 3.
8. Dana C, Doursounian L, Nourissat G. Arthroscopic treatment of a fresh lunate bone fracture detaching the scapholunate ligament. Chir Main. 2010;29(2):114–7. Epub 2010 Feb 23.
9. Levy M, Fischel RE, Stern GM, Goldberg I. Chip fractures of the os triquetrum: the mechanism of injury. J Bone Joint Surg Br. 1979;61-B(3):355–7.
10. Garcia-Elias M. Dorsal fractures of the triquetrum-avulsion or compression fractures? J Hand Surg Am. 1987;12(2):266–8.

11. Höcker K, Menschik A. Chip fractures of the triquetrum. Mechanism, classification and results. J Hand Surg Br. 1994;19(5):584–8.
12. Sin CH, Leung YF, Ip SP, Wai YL, Ip WY. Non-union of the triquetrum with pseudoarthrosis: a case report. J Orthop Surg (Hong Kong). 2012;20(1):105–7.
13. Al Rashid M, Rasoli S, Khan WS. Non-union of isolated displaced triquetral body fracture – a case report. Ortop Traumatol Rehabil. 2012;14(1):71–4.
14. Matsunaga D, Uchiyama S, Nakagawa H, Toriumi H, Kamimura M, Miyasaka T. Lower ulnar nerve palsy related to fracture of the pisiform bone in patients with multiple injuries. J Trauma. 2002;53(2):364–8.
15. Carroll RE, Coyle Jr MP. Dysfunction of the pisotriquetral joint: treatment by excision of the pisiform. J Hand Surg Am. 1985;10(5):703–7.
16. Gómez CL, Renart IP, Pujals JI, Palou EC, Busquets RC. Dysfunction of the pisotriquetral joint: degenerative arthritis treated by excision of the pisiform. Orthopedics. 2005;28(4):405–8.
17. Failla JM. Hook of hamate vascularity: vulnerability to osteonecrosis and nonunion. J Hand Surg Am. 1993;18(6):1075–9.
18. Bryan RS, Dobyns JH. Fractures of the carpal bones other than lunate and navicular. Clin Orthop Relat Res. 1980;149:107–11.
19. Guha AR, Marynissen H. Stress fracture of the hook of the hamate. Br J Sports Med. 2002;36(3):224–5.
20. O'Grady W, Hazle C. Persistent wrist pain in a mature golfer. Int J Sports Phys Ther. 2012;7(4):425–32.
21. Milch H. Fractures of the hamate bone. J Bone Joint Surg Am. 1934;16:459–62.
22. Pajares-López M, Hernández-Cortés P, Robles-Molina MJ. Rupture of small finger flexor tendons secondary to asymptomatic nonunion of the hamate hook. Orthopedics. 2011;34(2):142. doi:10.3928/01477447-20101221-35.
23. Aldridge 3rd JM, Mallon WJ. Hook of the hamate fractures in competitive golfers: results of treatment by excision of the fractured hook of the hamate. Orthopedics. 2003;26(7):717–9.
24. Smith P, Wright TW, Wallace PF, Dell PC. Excision of the hook of the hamate: a retrospective survey and review of the literature. J Hand Surg Am. 1988;13(4):612–5.
25. Scheufler O, Radmer S, Andresen R. Dorsal percutaneous cannulated mini-screw fixation for fractures of the hamate hook. Hand Surg. 2012;17(2):287–93.
26. Fredericson M, Kim BJ, Date ES, McAdams TR. Injury to the deep motor branch of the ulnar nerve during hook of hamate excision. Orthopedics. 2006;29(5):456–8.
27. Scheufler O, Radmer S, Erdmann D, Germann G, Pierer G, Andresen R. Therapeutic alternatives in nonunion of hamate hook fractures: personal experience in 8 patients and review of literature. Ann Plast Surg. 2005;55(2):149–54.
28. Demirkan F, Calandruccio JH, Diangelo D. Biomechanical evaluation of flexor tendon function after hamate hook excision. J Hand Surg Am. 2003;28(1):138–43.
29. Watson HK, Rogers WD. Nonunion of the hook of the hamate: an argument for bone grafting the nonunion. J Hand Surg Am. 1989;14(3):486–90.
30. Bishop AT, Beckenbaugh RD. Fracture of the hamate hook. J Hand Surg Am. 1988;13(1):135–9.
31. Vance RM, Gelberman RH, Evans EF. Scaphocapitate fractures. Patterns of dislocation, mechanisms of injury, and preliminary results of treatment. J Bone Joint Surg Am. 1980;62(2):271–6.
32. Rand JA, Linscheid RL, Dobyns JH. Capitate fractures: a long-term follow-up. Clin Orthop Relat Res. 1982;165:209–16.
33. Stein F, Siegel MW. Naviculocapitate fracture syndrome. A case report: new thoughts on the mechanism of injury. J Bone Joint Surg Am. 1969;51(2):391–5.
34. Milliez PY, Kinh Kha H, Allieu Y, Thomine JM. Idiopathic aseptic osteonecrosis of the capitate bone. Literature review apropos of 3 new cases. Int Orthop. 1991;15(2):85–94.
35. Morisawa Y, Ikegami H, Takayama S, Toyama Y. A case of pseudarthrosis of the capitate. Hand Surg. 2003;8(1):137–40.
36. Pointu J, Schwenck JP, Destree G, Séjourné P. Fractures of the trapezium. Mechanisms. Anatomo-pathology and therapeutic indications. Rev Chir Orthop Reparatrice Appar Mot. 1988;74(5):454–65.
37. Garneti N, Tuson CE. Sagittally split fracture of trapezium associated with subluxated carpo-metacarpal joint of thumb. Injury. 2004;35(11):1172–5.
38. Palmer AK. Trapezial ridge fractures. J Hand Surg Am. 1981;6(6):561–4.
39. Wiesler ER, Chloros GD, Kuzma GR. Arthroscopy in the treatment of fracture of the trapezium. Arthroscopy. 2007;23(11):1248.e1–4. Epub 2007 Jan 5.
40. Kain N, Heras-Palou C. Trapezoid fractures: report of 11 cases. J Hand Surg Am. 2012;37(6):1159–62. Epub 2012 Apr 21.
41. Kam ML, Sreedharan S, Teoh LC, Chew WY. Severe isolated trapezoid fracture: a case report. Hand Surg. 2011;16(2):185–7.

Acute Scaphoid Fractures

12

Nick R. Howells, Rouin Amirfeyz, and Tim R.C. Davis

Keywords
Acute scaphoid fracture • Wrist fracture • Scaphoid fracture • Imaging of scaphoid • Classification • Surgery for scaphoid fracture

Introduction

The scaphoid is the most commonly fractured carpal bone, accounting for 11 % of all fractures of the hand and 60 % of fractures of the carpus [1]. Detection and treatment of scaphoid injuries can be challenging as the clinical symptoms and signs can be subtle and non-union may occur. If a scaphoid non-union goes untreated, radiological osteoarthritis is almost inevitable and pain probable. As they occur most commonly in young men, the resultant socio-economic consequences can be considerable.

Scaphoid Fractures
Most common carpal bone fracture
Diagnosis often missed
M:F ratio – 5:1
Fracture of young adults – Peak in third Decade
Tendency to mal/non-union
If untreated non union leads to arthritis

Mechanism of Injury

Scaphoid fractures most commonly occur as a result of a fall onto an outstretched hand, with forced extension of the wrist. Common mechanisms include a simple low energy fall, a fall during sporting activities, or wrist hyperextension in a road traffic accident. Fractures can also occur with forced palmar flexion of the wrist and axial loading of the flexed wrist, as with punching. Fractures sustained on "test your strength" punch bag machines are not uncommon [2–4]. Rarer mechanisms include the result of a forced kickback when using a starter handle of an old motor vehicle or a cement mixer. Both

N.R. Howells, MSc, FRCS (Trauma & Orth)
R. Amirfeyz, MD, MSc, FRCS (Trauma and Orth)
Department of Orthopaedics, Bristol Royal Infirmary, Bristol BS2 8HW, UK
e-mail: nicholas.howells@nbt.nhs.uk; rouin.amirfeyz@uhbristol.nhs.uk

T.R.C. Davis, FRCS (✉)
Department of Orthopaedics, Nottingham University Hospitals, Queen's Medical Centre, Nottingham NG7 2UH, UK
e-mail: tim.davis@nuh.nhs.uk

I.A. Trail, A.N.M. Fleming (eds.), *Disorders of the Hand: Volume 1: Hand Injuries*,
DOI 10.1007/978-1-4471-6554-5_12, © Springer-Verlag London 2015

mechanisms result in a forced and sudden dorsiflexion at the wrist, often with associated radial deviation. Cadaveric study has revealed that once the wrist is extended beyond 95° the proximal pole of the scaphoid becomes compressed between the radius and the capitate and causes it to fail. Secondary to this compression, the degree of wrist extension at impact is thought to influence the location of the scaphoid fracture. The greater the degree of wrist extension, the more distal the fracture.

Anatomy

The scaphoid is situated radially in the carpus and bridges the proximal and distal carpal rows. Scaphoid is derived from the Greek word *skaphe,* meaning boat. It is concave in both ulnar and palmar directions and is covered with articular cartilage along its entire proximal, distal and medial surfaces and half of its lateral surface. Its blood supply is from the radial artery. Dorsal branches supply 70 % of the bone, including the proximal pole. The remaining 30 % is from the palmar vessels. The vessels enter the distal third of the scaphoid and run within the bone to the proximal pole. There is a variable, small additional blood supply to the proximal pole via the ligament of Testut.

The alignment of the scaphoid is complex and somewhat variable. It lies with 45° (30–60) palmar tilt on a lateral radiograph and 45° radial tilt on a PA radiograph relative to the long axis of the radius. The average scapholunate angle is 45° (35–60) measured on lateral radiograph between the long axis of the scaphoid and long axis of the lunate.

The scaphoid articulates with the trapezium and trapezoid distally and is attached to both by joint capsule. It also articulates with the convex surface of the capitate and proximally with the scaphoid fossa of the distal radius. Its most complex articulation is with the lunate.

As the stabiliser between the proximal and distal carpal rows, the scaphoid is potentially unstable and its stability relies on three intrinsic and two extrinsic ligaments. The intrinsic ligaments allow for synchronous scaphoid and lunate movement, with the scaphoid and lunate flexing with wrist radial deviation and extending with ulnar deviation. The extrinsic ligaments are the radioscaphoid and the radioscaphocapitate ligaments.

When a scaphoid fractures, the proximal fragment tends to extend with the attached lunate and the distal fragment remains flexed, creating a "humpback" deformity.

Classification of Scaphoid Fractures

The most widely used and clinically useful classification of scaphoid fractures is by simple anatomical location.

- Proximal Pole
- Waist
- Distal Pole

The Russe and Herbert classification systems are the most commonly described, but both are poor predictors of fracture union and demonstrate only fair inter and intraobserver reliability [5].

The Russe classification describes three types of waist fractures in terms of fracture line orientation in relation to the long axis of the scaphoid:

- vertical oblique
- horizontal oblique
- transverse

Vertical oblique fractures are the rarest (5 % of scaphoid fractures) and are potentially unstable, as they have the highest shear forces [6].

The Herbert Classification is more comprehensive and differentiates between stable and unstable fracture patterns, which may be helpful in guiding treatment options [7].

- Type A: Incomplete waist or tuberosity fractures defined as stable
- Type B: Displaced waist or proximal pole fractures defined as unstable
- Type C: Delayed union
- Type D: Established nonunion

Diagnosis

The diagnosis of the scaphoid fracture should start with a careful history and examination.

History

Attention to the details of the mechanism of injury is too often overlooked. Time of onset of wrist pain can aid differentiation of a scaphoid fracture, which is usually painful immediately, from a soft tissue injury where pain may only develop after several hours. However, an adrenaline fuelled sportsman may not notice the pain from his/ her fracture until the end of the game. Prior history of wrist pain may often indicate a recurrent soft tissue problem or a long-standing non-union rather than an acute fracture.

Clinical Examination

Inspection

Swelling in the anatomical snuffbox may be present and is best seen by abducting the thumb to produce a concavity and comparison with the contralateral side. This can take time to develop and is an indication of a wrist joint effusion or haemarthrosis. There may be no swelling, or more generalised swelling, around the wrist.

Palpation

Palpation should be performed with a single fingertip over all aspects of the carpus and comparison should be made to the contralateral side. Tenderness may be expected over the scaphoid tubercle, in the anatomical snuff-box or dorsally over the proximal pole of the scaphoid just distal to Lister's tubercle, depending on the location of the fracture. In isolation, tenderness in these sites is sensitive, but not specific. The specificity of the clinical signs used in evaluation of suspected scaphoid injuries is improved when they are considered in combination [8].

Movement

The wrist can retain a reasonable arc of pain free movement following a scaphoid fracture, but there is usually pain at the extremes of passive movement.

Special Tests

Several special tests have been described, which include thumb axial compression, resisted pronation, ulnar deviation of a pronated wrist and pain on tapping the tip of an abducted thumb. None is specific or widely adopted [9, 10].

Imaging

If history and examination raise the slightest suspicion of a scaphoid fracture, then scaphoid series plain radiographs are indicated. The most common views performed as part of a scaphoid series are, postero-anterior, lateral, 45° ulnar oblique and 45° radial oblique. In addition a Ziter's fist clenched view taken PA with the wrist ulnar deviated and the x-ray beam at 20° to the longitudinal axis of the wrist is often useful for identifying waist fractures [11, 12]. Undisplaced scaphoid fractures may not be visible on scaphoid series radiographs. For this reason a common practice is to repeat the x-rays at 2 weeks, by which time bone resorption at the fracture site may have made the fracture visible [13].

Some studies report that between 30 and 40 % of scaphoid fractures are not identified following initial clinical assessment and scaphoid series radiographs [12]. Furthermore the inter and intra observer reliability for diagnosis of suspected fracture from scaphoid series x-rays is poor [14, 15]. Reasons suggested for the difficulty in interpretation of scaphoid views include; a line which is mistaken as a fracture may be formed by the dorsal lip of the radius overlapping the scaphoid, a white line running across the scaphoid waist may be formed by the proximal end of the scaphoid tuberosity and the dorsal ridge of the scaphoid can appear bent on the radial oblique view [10]. All three may result in the over-diagnosis of scaphoid fractures, but careful evaluation of good quality scaphoid series x-rays demonstrates the majority of acute fractures and repeat radiographs of suspected fractures are usually negative [10].

Alternative imaging modalities are increasingly being used, both to identify occult fractures and also to assess the configuration and displacement of the fracture.

Bone scintigraphy has been used for patients with suspected fractures which are not visible on repeat x-rays at 2 weeks and it continues to be used in certain centres. Sensitivity has been

reported up to 100 %, but in most studies specificity remains poor in comparison to CT and MRI. Focal increased uptake suggests acute fracture; diffuse increased uptake may indicate synovitis and reduced uptake may indicate early ischaemia or necrosis. However, they provide no information regarding fracture displacement or associated soft tissue injuries [14, 16].

Computed Tomography (CT) scans have 93–94 % sensitivity and 96–99 % specificity for the diagnosis of suspected scaphoid fractures [17, 18]. Interestingly, despite good inter- and intra-observer reliability, studies have shown a false positive rate. This is thought to be due to vascular foramen within the scaphoid mimicking unicortical fractures [19].

Magnetic Resonance Imaging (MRI) is generally accepted as the gold standard investigation for the diagnosis of a suspected fracture of the scaphoid. Its sensitivity is quoted at 95–100 % and its specificity is approaching 100 % [13, 17, 18]. One study reported the use of MRI at 72 h post injury in patients with clinically suspicious, but not radiologically evident, scaphoid fractures. It showed 100 % specificity and sensitivity and quoted potential direct cost savings of $7,200 per 100,000 population, when compared to standard follow up with repeat x-rays and clinical examination [20]. If indirect costs (time off work and economic implications) are taken into consideration, then the argument for the use of MRI to identify occult fractures in the clinically suspicious patient becomes even stronger. Recent studies have shown CT, MRI and scintigraphy have comparable diagnostic performance characteristics for detecting a true scaphoid fracture from suspected fractures [18, 21]. As a result, the Royal College of Radiologists have concluded that, on current evidence, they are comparable for triaging patients with a suspected fracture [22].

Diagnosis of Scaphoid Fractures

Careful History – Focus on mechanism of injury and site of pain

Combination of positive clinical signs increases likelihood of fracture

Initial examination and radiographs will miss a proportion of injuries

MRI, CT and Bone Scintigraphy are sensitive and specific for the diagnosis of the suspected scaphoid fracture

Management

Acute scaphoid fractures require treatment with either a period of cast immobilisation or operative fixation. The surgeon should be aware of the relevant anatomy, potential risks and likely outcomes in order to provide appropriate management options for specific patients.

Non-operative Management

Fractures of the scaphoid tuberosity are appropriate for conservative management. They can be treated safely and reliably in cast for 4–6 weeks. Non-union is extremely rare [23]. Some surgeons treat these injuries with a removable splint, which is worn for pain relief [10].

Undisplaced scaphoid waist fractures unite with non-operative treatment in below-elbow plaster for 4–8 weeks. However, the development of cannulated headless compression screws and percutaneous techniques for their insertion has prompted some surgeons to treat patients with these injuries surgically. Their aim is to reduce the period of immobilisation, the rate of non-union and the time to union [24, 25]. A recent systematic review found no benefit of fixation over cast immobilisation for undisplaced waist fractures, in terms of non union rate, grip strength, range of movement or patient satisfaction [26]. Studies have reported slightly reduced rates of time to union of around 4–5 weeks and slightly earlier return to work with percutaneous operative fixation, but assessment of time to union is very imprecise and also depends on the timing of assessments. Furthermore, any benefit of operative fixation has to be balanced against the associated risks of adverse events, such as screw misplacement and distraction of the fracture, especially in centres which do not routinely perform operative fixation [27].

Typical periods of cast immobilisation and likely success of non-operative management in a plaster cast

Location	Period of immobilisation (weeks)	Union rate. Non-operative (%)
Tuberosity	4–6	100
Undisplaced Waist	4–8	Up to 95
Displaced Waist	8–12	50
Proximal Pole	12	Up to 40

The majority of scaphoid waist fractures are presently treated in cast. The type of cast and position of the wrist in the cast have been the subject of several studies and most surgeons use either a below elbow "Colles type" cast, or a below elbow scaphoid cast, which includes the thumb up to the inter-phalangeal joint. A prospective randomised study and a more recent meta-analysis found no difference in rates of union associated with the use of these two casts [27, 28]. Wrist position in cast was assessed in a prospective randomised controlled trial, comparing cast immobilisation in flexion and extension. There was no difference in rates of union, but immobilisation in flexion led to a slight restriction in wrist extension, which persisted at 6 month follow up. It is therefore advised to immobilise the wrist in slight extension, which is also the most functional position [29].

Non-operative Management of Scaphoid Fractures

Scaphoid Tuberosity and isolated waist fractures, unless severely displaced

Union rates of >90 % in the above injuries

Thumb immobilisation does not impact on union rates

Cast with wrist in slight extension for improved function

Assessment of Fracture Union

How long should patients remain in cast? The intuitive answer is until there is clinical and radiological evidence of union, but Watson Jones followed this principle in the 1950s and immobilised patients for up to 10 months, which is clearly not appropriate [10]. What this question is essentially asking is how does one determine when a scaphoid fracture has united?

Clinical indications of union are the absence of pain and improving function. A common protocol for a scaphoid waist fracture is to assess the patient for signs of persisting tenderness, following 8 weeks cast immobilisation. If there is persisting tenderness then some surgeons will immobilise further to a maximum of 12 weeks [10]. Assessment of union on plain x-rays is often difficult between 8 and 12 weeks and has poor inter and intraobserver reliability. The difficulty in assessing union has been attributed to the absence of a periosteal reaction, due to the intra-articular nature of the scaphoid, the fracture lying obliquely to the x-ray beam so that parallax produces the impression of bridging callus and union of only a portion of the cross-section of the fracture, such that the fracture line is still clearly visible, though the union has occurred [30].

A novel approach has been to perform a CT scan of patients with waist fractures at 4 weeks. If these show the fracture is undisplaced and appears to have united across 50 % of its cross section, then the fracture has almost certainly united sufficiently to discard the cast and mobilise the wrist [31].

A pragmatic approach to the assessment of union is:

- If the wrist is painless and the x-rays suggest union, then union is very likely.
- If the wrist is painful and x-rays suggest non-union then there is a high risk of non-union.
- If the wrist is painless but x-rays suggest non-union, or the wrist is painful but x-rays suggest union then further investigation is needed, i.e. a CT scan.

Factors Which May Influence the Union of Scaphoid Fractures Treated Non-operatively

Failure to diagnose and treat the fracture such that the wrist and fracture are not immobilised

Patient non-compliance (removes his/her cast)
Fracture displacement
Proximal pole fragment avascularity
Fracture comminution
Smoking
Associated carpal instability

Operative Management

There is varied opinion as to when it is appropriate to undertake operative intervention for scaphoid fractures. Some quoted indications are:

Indications for Operative Management of Scaphoid Fractures

Definite
- Displacement – >2 mm
- Delay – delay in diagnosis of >4 weeks makes union in plaster less likely
- Dislocation – as part of a more extensive injury e.g. trans-scaphoid perilunate

Probable
- Proximal pole fractures

Other
- Comminution
- Displacement >1 mm
- Intrascaphoid angle >35°
- Height to length ratio >0.65

Approximately 30 % of fractures of the scaphoid waist are displaced and these are more likely to develop mal-union or non-union [10]. Assessment of displacement on plain radiographs is inaccurate [5, 32], hence CT (Fig. 12.1) or MRI scanning is required for a clearer assessment [32]. These injuries may be best managed with reduction and fixation, but many will be comminuted and some will be technically difficult to accurately reduce, compress and stabilise, even with modern cannulated screw fixation techniques. Union rates with operative fixation of up to 93 % are quoted, as well as lower rates of mal-union in comparison to non-operative management [33]. What is less clear is how displaced is displaced? Some surgeons quote a fracture gap

Fig. 12.1 Sagital CT scan of scaphoid showing displacement of the fracture site

of 1 mm, an intrascaphoid angle greater than 35° or a scapholunate angle greater than 60°, all measured on plain scaphoid series X-ray [34]. Others point out that patients meeting these criteria have been included in large series of patients treated non-operatively and their functional outcome was no worse than for those deemed undisplaced [10]. A recent study has looked at a series of patients with displaced scaphoid waist fractures treated non-operatively in a plaster cast for up to 12 weeks. Displacement was assessed with CT at 4 weeks. All of the patients with fracture gap less than 2 mm united, but non-union was associated with a fracture gap of greater than 2 mm. Non-union was also associated with large areas (>50 % of articular surface) of bone resorption on the 4 week CT scan [35].

Proximal Pole Fractures

These occur in the proximal 20 % of the scaphoid length and must be differentiated from scaphoid waist fractures. This is as they have a higher rate

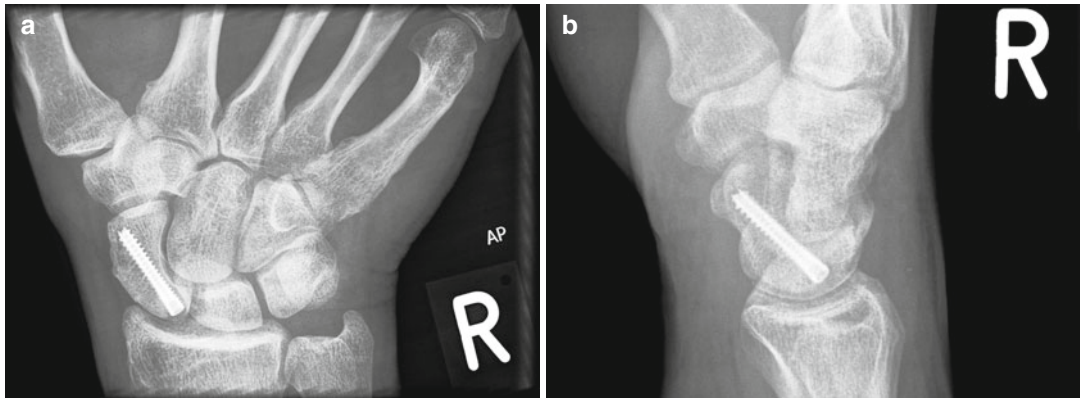


Fig. 12.2 Proximal pole screw fixation proximal to distal. (**a**) Posteroanterior (**b**) Lateral views

of non-union in comparison to waist and distal fractures and also because the results of surgery for established non-union at this site are poor. Both are probably due to the poor blood supply and the inherent instability of proximal fractures. The available evidence on operative vs. conservative management is scarce. Most surgeons have a low threshold for surgical intervention, certainly with evidence of displacement or deformity. Fixation is best performed antegrade (proximal to distal), using a headless screw (Fig. 12.2), a small cortical screw or a Kirschner wire [10, 36].

Surgical Techniques

Open – Palmar Approach

This is the classic Russe approach, which provides excellent exposure of the entire palmar surface of the scaphoid and avoids damage to the predominantly dorsal blood supply to the scaphoid. It is the standard approach for open reduction and fixation of displaced fractures of the waist.

A longitudinal incision is made radial to the flexor carpi radialis tendon, which is retracted in an ulnar direction. The incision is continued distally as a hockey stick over the scaphoid tubercle. The deep fascia is divided in line with the skin and the radial artery is identified and protected on the radial side of the wound. The palmar wrist capsule is divided longitudinally. A horizontal capsular incision to expose the scaphotrapezial joint may or may not be required [6].

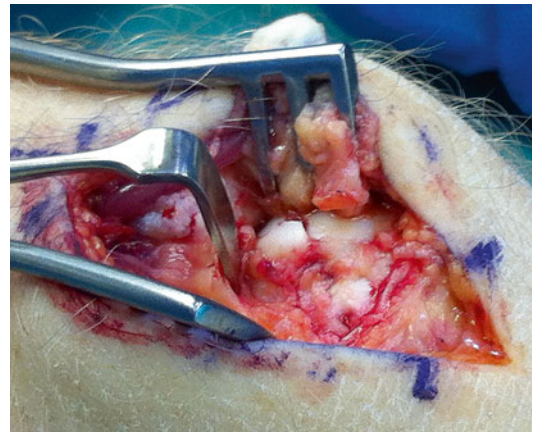


Fig. 12.3 Dorsal approach to scaphoid. Displaced proximal pole fractures are best reduced and stabilized through this technique

Open – Dorsal Approach (Fig. 12.3)

The dorsal approach is an alternative that provides excellent exposure to the proximal portion of the scaphoid. It is the approach of choice for proximal pole fractures, as it allows antegrade screw insertion into the central portion of the small proximal pole fragment, which is much simpler than retrograde insertion through the palmar approach. The approach is an oblique incision over and distal to the Lister's tubercle. The extensor retinaculum is divided longitudinally, taking care to identify and protect branches of the superficial radial nerve. The tendons of the second and third extensor compartments (ECRL, ECRB and EPL) are retracted and the wrist

capsule is incised longitudinally, taking care not to damage either the main dorsal ridge blood supply or the scapholunate ligament.

Percutaneous

Percutaneous techniques have grown in popularity following the development of cannulated compression screws. Their use should minimise the risks of capsular scarring and damage to the scaphoid blood supply. They are probably only appropriate for undisplaced or minimally displaced fractures, as closed reduction of displaced fractures can be extremely difficult and, without arthroscopy, there is no way of confirming an accurate fracture reduction. A retrograde technique with a distal, palmar entry point is used for distal and waist fractures. An antegrade technique with a dorsal, proximal entry point is used for proximal pole fractures.

Whichever approach is used, it is important that the guidewire is passed along the central longitudinal axis of the scaphoid and image intensifier screening is important to assess this. For the palmar approach, the guidewire should be inserted with the wrist supinated and extended. For the dorsal approach, it should be flexed and pronated.

Some surgeons combine percutaneous fixation with wrist arthroscopy, which allows assessment of the fracture reduction and any associated cartilage damage or ligament injury [37].

Surgical intervention for scaphoid fracture ideally requires a fixation device that can provide interfragmentary compression and allow early mobilisation of the wrist out of plaster. Thus, if the fracture allows, a cannulated headless screw is preferred to Kirschner wires. The original Herbert screw provided interfragmentary compression, by having different screw pitches proximally and distally, but was not cannulated. Strong titanium alloys have allowed the development of smaller cannulated screw systems, which retain sufficient strength to provide compression and stability. Some of these allow adjustment of the degree of interfragmentary compression.

Outcome of Acute Scaphoid Fractures

Outcome of Acute Scaphoid Fractures

90 % Union at 2 years with non-operative and operative treatment

If union is achieved long term functional outcome is good

11 % of patients report mild persistent symptoms

Malunion – does not affect short term functional outcome

Malunion – may result in increased incidence of arthritis

Mild radiological arthritis reported in 5 % of patients with united fractures at 7 years 85 % or more of acute scaphoid fractures will unite if diagnosed and treated by cast immobilisation [5, 27, 28]. Those that unite generally function well and restoration of a normal range of movement and grip strength is usual [38, 39]. Long-term studies have shown that even those patients who experience complications following operative fixation go on to function well if union is achieved [40]. A proportion of patients, whether treated operatively or non-operatively, do experience persistent symptoms despite union, most commonly pain, though sometimes loss of motion. This has been attributed to articular cartilage damage or malunion. A study of 229 consecutive patients, with united scaphoid fracture, revealed a 5 % rate of radiographic osteoarthritis at 7 years [38].

Malunion most commonly occurs as a result of flexion of the scaphoid fracture, resulting in a humpback deformity. Assessment of mal-union is unreliable on both plain radiographs and CT scans [41]. It is often defined as either a lateral intra-scaphoid angle of greater than 35° or a height to length (H/L) ratio of ≥0.6 [39]. A cadaveric study of simulated malunion demonstrated marked restrictions in radiocarpal extension and midcarpal motion and some, but not all, clinical studies have suggested such mal-union may result in persistent pain and stiffness and increase the long term risk of post traumatic osteoarthritis [28, 38, 42, 43].

Conclusions

Scaphoid waist fractures continue to cause anxiety for several reasons, including:

1. The difficulties encountered distinguishing soft tissue injuries (suspected scaphoid fractures) from scaphoid fractures, which are not visible on scaphoid series X-rays.

   However, increased availability of CT or MRI a.llows the detection, or exclusion, of fractures in patients with continuing symptoms and signs of a fracture though "normal" scaphoid series X-rays.
2. Concern that the scaphoid fracture may not heal with non-operative treatment, either due to poor vascularity or displacement.

   Research suggests that proximal fragment vascularity is not a major determinant of the outcome of non-operative treatment of scaphoid waist fractures in plaster, whereas displacement does significantly affect the union rate [44].

   Displacement is not reliably assessed on scaphoid series radiographs, but is readily assessed with CT. The vast majority of scaphoid waist fractures with 2 mm or less displacement (gapping) assessed with CT unite with non-operative treatment, though the union rate diminishes with greater amounts of displacement. As the majority of scaphoid waist fractures seen in a general Orthopaedic practice are undisplaced, or have less than 2 mm displacement, non-operative treatment in a below elbow plaster cast remains an acceptable treatment for the majority of scaphoid fractures. Reduction and fixation of waist fractures, with greater amounts of displacement may increase the union rate in this sub-group of fractures, though these fractures will not be as easy to treat operatively, as they are unstable and there may be fracture comminution.
3. Concern that the scaphoid fracture will unite with malunion if treated non-operatively.

   Although some surgeons are concerned about the effect of malunion on the outcome of scaphoid fractures, its adverse effect on the clinical outcome is undoubtedly much less than that of non-union, and it is probable that mild amounts of malunion do not affect the clinical outcome.
4. Difficulty determining when a scaphoid fracture has united sufficiently to allow free mobilisation of the wrist.

   When it is uncertain whether a scaphoid fracture has, or has not united, a CT scan of the scaphoid will usually clarify the situation. If 50 % or more of the cross section of the fracture appears united, then it is probably safe to mobilise the wrist in anticipation of consolidation of the fracture with the passage of time.

Proximal pole fractures are much less common than waist fractures and their management is difficult. They have a higher rate of non-union than waist fractures and, if non-union occurs, it is more difficult to treat than one of a waist fracture. For these reasons acute operative fixation appears attractive and is indicated for displaced fractures, but this does not guarantee union.

References

1. Hove LM. Fractures of the hand. Distribution and relative incidence. Scand J Plast Reconstr Surg Hand Surg. 1993;27:317–9.
2. Weber ER, Chao EY. An experimental approach to the mechanism of scaphoid waist fractures. J Hand Surg Am. 1978;3:142–8.
3. Sutton PA, Clifford O, Davies TRC. A new mechanism of injury for scaphoid fractures: "test your strength" punch-bag machines. J Hand Surg Eur. 2010;35E(5):419–20.
4. Mayfield JK. The mechanism of carpal injuries. Clin Orthop Relat Res. 1980;149:45–54.
5. Desai VV, Davis TRC, Barton NJ. The prognostic value and reproducibility of radiological features of the fractured scaphoid. J Hand Surg (Eur). 1999;24B(5):586–90.
6. Russe O. Fracture of the carpal navicular. Diagnosis, non-operative treatment and operative treatment. J Bone Joint Surg Am. 1960;42:759–68.
7. Herbert TJ. Fractured scaphoid. St Louis: Quality Medical; 1990.
8. Parvizi J, Wayman J, Kelly P, Moran CG. Combining the clinical signs improves diagnosis of scaphoid fractures. J Hand Surg (Eur). 1998;23B(3):324–7.
9. Waizenegger M, Barton NJ, Davis TRC, Wastie ML. Clinical signs in scaphoid fractures. J Hand Surg. 1994;19B:743–7.

10. Gunal I, Barton NJ, Calli I. Current management of scaphoid fractures: twenty questions answered. London: Royal Society of Medicine Press; 2002.
11. Ziter FMH. A modified view of the carpal navicular. Radiology. 1973;108:706–7.
12. Duckworth AD, Ring D, McQueen MM. Assessment of the suspected fracture of the scaphoid. J Bone Joint Surg [Br]. 2011;93-B:713–9.
13. Adams JE, Steinmann SP. acute scaphoid fractures. Hand Clin. 2010;26:97–103.
14. Tiel-van Buul MM, van Beek EJ, Broekhuizen AH, et al. Radiography and scintigraphy of suspected scaphoid fracture: a long-term study in 160 patients. J Bone Joint Surg [Br]. 1993;75-B:61–5.
15. Dias JJ, Thompson J, Barton NJ, Gregg PJ. Suspected scaphoid fractures. The value of radiographs. J Bone Joint Surg. 1990;72-B:98–101.
16. Brismar J. Skeletal scintigraphy of the wrist in suggested scaphoid fracture. Acta Radiol. 1988;29:101–7.
17. Ring D, Lozano-Calderon S. Imaging for suspected scaphoid fracture. J Hand Surg Am. 2008;33:954–7.
18. Yin ZG, Zhang JB, Kan SL, Wang XG. Diagnosing suspected scaphoid fractures: a systematic review and meta-analysis. Clin Orthop. 2010;468:723–34.
19. Adey L, Souer JS, Lozano-Calderon S, et al. Computed tomography of suspected scaphoid fractures. J Hand Surg Am. 2007;32:61–6.
20. Gaebler C, Kukla C, Breitenseher M, et al. Magnetic resonance imaging of occult scaphoid fractures. J Trauma. 1996;41:73–6.
21. Malee W, Doornberg JM, Ring D, et al. Comparison of CT and MRI for diagnosis of suspected scaphoid fractures. J Bone Joint Surg Am. 2011;93-A:20–8.
22. Royal College of Radiologists. Maing the best use of clinical radiology services guidelines for doctors. 5th ed. London: Royal College of Radiologists; 2003.
23. Mody BS, Belliappa PP, Dias JJ, Barton NJ. Nonunion of fractures of the scaphoid tuberosity. J Bone Joint Surg Br. 1993;75:423–5.
24. Dias JJ, Wildin CJ, Bhowal B, Thompson SR. Should acute scaphoid fractures be fixed? A randomised controlled trial. J Bone Joint Surg Am. 2005;87:2160–8.
25. Mcqueen MM, Gelbke MK, Wakefield A, Will EM, Gaebler C. Percutaneous screw fixation versus conservative treatment for fractures of the waist of the scaphoid. A prospective randomised study. J Bone Joint Surg Br. 2008;90:66–71.
26. Modi CS, Nancoo T, Powers D, Ho K, Boer R, Turner SM. Operative versus non-operative treatment of acute undisplaced and minimally displaced scaphoid waist fractures- a systematic review. Injury. 2009;40:142–51.
27. Yin Z, Zhang J, Kan S, Wang P. Treatment of acute scaphoid fractures. Systematic review and meta-analysis. Clin Orthop Relat Res. 2007;460:142–51.
28. Clay NR, Dias JJ, Costigan PS, Gregg PJ, Barton NJ. Need the thumb be immobilised in scaphoid fractures? A randomised prospective trial. J Bone Joint Surg [Br]. 1991;73-B:828–32.
29. Hambidge JE, Desai VV, Schranz PJ, Compson JP, Davis TRC, Barton NJ. Acute fractures of the scaphoid. Treatment by cast immobilization with the wrist in flexion and extension? J Bone Joint Surg [Br]. 1999;81-B:91–2.
30. Dias JJ, Taylor M, Thompson J, Brenkel IJ, Gregg PJ. Radiographic Signs of union of scaphoid fractures. An analysis of interobserver agreement and reproducibility. J Bone Joint Surg [Br]. 1988;70-B:299–301.
31. Geoghegan JM, Woodruff MJ, Bhatia R, Dawson JS, Kerslake RW, Downing ND, Oni JA, Davis TRC. Undisplaced scaphoid waist fractures: is 4 weeks' immobilisation in a below-elbow cast sufficient if a week 4 CT scan suggests fracture union? J Hand Surg Eur Vol. 2009;34:631.
32. Bhat M, McCarthy M, Davis TRC, Oni JA, Dawson S. MRI and plain radiography in the assessment of displaced fractures of the waist of the carpal scaphoid. J Bone Joint Surg [Br]. 2004;86-B:705–13.
33. Rettig ME, Kozin SH, Cooney WP. Open reduction and internal fixation of acute displaced scaphoid waist fractures. J Hand Surg. 2001;26A:271–6.
34. Cooney WP, Dobyns JH, Linscheid RL. Fractures of the scaphoid: a rational approach to management. Clin Orthop Relat Res. 2007;460:142–51.
35. Amirfeyz R, Bebbington A, Downing ND, Oni JA, Davis TRC. Displaced scaphoid waist fractures: the use of a week 4 CT scan to predict the likelihood of union with nonoperative treatment. J Hand Surg (Eur). 2011;36(6):498–502.
36. Haisman JM, Rohde RS, Weiland AJ. Acute fractures of the scaphoid. J Bone Joint Surg Am. 2006;88:2750–8.
37. Slade 3rd JF, Gutow AP, Geissler WB. Percutaneous internal fixation of scaphoid fractures via an arthroscopically assisted dorsal approach. J Bone Joint Surg Am. 2002;84-A Suppl 2:21–36.
38. Lindstrom G, Nystrom A. Incidence of post-traumatic arthrosis after primary healing of scaphoid fractures: a clinical and radiological study. J Hand Surg Eur. 1990;15:11–3.
39. Forward DP, Singh HP, Dawson S, Davis TRC. The clinical outcome of scaphoid fracture malunion at 1 year. J Hand Surg Eur Vol. 2009;34:40.
40. Dias JJ, Dhukaram V, Abhinav A, Bhowal B, Wildin CJ. Clinical and radiological outcome of cast immobilisation versus surgical treatment of acute scaphoid fractures at a mean follow-up of 93 months. J Bone Joint Surg [Br]. 2008;90-B:899–905.
41. Ring D, Patterson JD, Levitz S, Wang C, Jupiter JB. Both scanning plane and observer affect measurements of scaphoid deformity. J Hand Surg Am. 2005;30:696–701.
42. Burgess RC. The effect of a simulated scaphoid malunion on wrist motion. J Hand Surg Am. 1987;12:774–6.
43. Nakamura P, Imaeda T, Miura T. Scaphoid malunion. J Bone Joint Surg Br. 1991;73:134–7.
44. Dawson JS, Martel AL, Davis TRC. Post-gadolidium enhancement patterns in acute scaphoid fractures and the relationship to fracture healing. J Bone Joint Surg Br. 2001;83B:809–14.

Fractures of the Distal Radius and Distal Radioulnar Joint

13

Douglas A. Campbell and Louise A. Crawford

Keywords

Fracture • Radius • Wrist • Ulna • Articular • Insufficiency • Associated injuries • Classification • Imaging • Distal radioulnar joint • Columnar theory • Angular stability • Variable angle

Introduction

Fractures of the distal radius are amongst the most common fractures seen in everyday orthopaedic practice. They were originally thought to be simple dislocations in the days prior to radiographic examination. The French physician, JL Petit, challenged this view in 1705, when he proposed that these post-traumatic deformities were actually caused by fractures of the bones and not dislocations of the joints. Some years later, the ideas of Claude Pouteau (published posthumously in 1783) supported this theory;

> These fractures are most often taken for contusions, luxations incomplete, or for separation of the radius from the ulna at their junction near the wrist.

D.A. Campbell, ChM, FRCS.Ed, FRCS(Orth), FFSEM(UK) (✉)
Department of Trauma & Orthopaedic Surgery, Leeds Teaching Hospitals NHS Trust, Great George Street, Leeds LS1 3EX, UK
e-mail: handandwristclinic@live.co.uk

L.A. Crawford, FRCS(Tr & Orth)
Department of Orthopaedic Surgery, Tameside General Hospital, Fountain Street, Ashton Under Lyne, Lancashire OL6 9RW, UK
e-mail: lcrawford@doctors.org.uk

For many (even contemporary) surgeons, the name of Abraham Colles is associated most closely with fractures of the distal radius, in an eponymous description that continues to be widely employed. Yet Colles published his work in 1814 – 80 years before the discovery of x-rays, so it remains unclear exactly what he was describing in the injury that now, all too frequently, bears his name. He did, however, crystallize the reasons behind the confusion that had existed in diagnosing a dislocation rather than a fracture when he wrote;

> …the absence of crepitus and of the other usual symptoms of fracture rendered the diagnosis extremely difficult..

Indeed, it is this finding that still characterizes many distal radius fractures – namely the inherent ‘stability’ of the displaced and impacted fragments that can continue to allow good function in the early stages after injury. These are the typical findings of the extra articular ‘bending’ fracture of the metaphysis, the common pattern found after a simple low energy injury in insufficient bone. Those fragility fractures that are deemed ‘unacceptably displaced’ have traditionally been

I.A. Trail, A.N.M. Fleming (eds.), *Disorders of the Hand: Volume 1: Hand Injuries*,
DOI 10.1007/978-1-4471-6554-5_13, © Springer-Verlag London 2015

managed by attempts at manipulation, often without secure stabilization, but then simply allowed to heal in malunion when inevitable redisplacement occurs. The reasons that the management of these simple injuries has been so different from fractures in other anatomical areas are that they represent such a significant workload, in terms of number of cases per annum, in all hospitals treating them, and (most importantly) that final functional outcome cannot be reliably predicted on the fracture pattern alone. The changing demographics in our society and the improvements in the economics of healthcare provision have seen the burgeoning epidemic of these injuries treated in very different ways over the past two decades. Yet no consensus exists as to the 'best' method of treatment and proponents of different treatments continue to try to prove the unprovable.

Add to this the relatively recent (60 years) recognition that articular fractures occur in a different type of injury and victim and in many cases have a very different outcome to the simple extra-articular bending fracture then it becomes apparent that the term 'distal radius fracture' is widely heterogenous.

The most recent understanding of these injuries now recognizes the importance of the distal ulna in certain fracture patterns, producing injuries of the distal radioulnar joint (DRUJ) that threaten forearm rotation, stability and positioning of the hand. It can now be understood why fractures in this area should be referred to as 'wrist fractures' in view of the involvement of all aspects of that articulation, not simply fractures of the radius.

This chapter aims to describe these areas of evolving knowledge, yet also provide a balanced and reasoned set of principles to produce a framework for managing the 'wrist fracture'.

The Injury

It is critical to first understand the nature of the injury before making decisions about treatment.

Fractures of the distal radius have been subject to analysis, grouping and classification for decades, in an attempt to recognize common fracture patterns, with the aim of providing guidance to management and an ability to more reliably predict outcome. No single classification system has been successful in this, but an understanding of the aims of the authors of such systems provides us with several general and repeatable principles.

Frykman's classification considered both bones of the wrist in the injury together, and is important in acknowledging that this should have been the beginning of more detailed work on 'wrist' fractures [1]. However, subsequent classifications moved away from this 'joint injury' approach and isolated each bone individually, to look at fracture patterns. This resulted in assessment of individual components of the injury (radial fracture, ulnar fracture etc) rather than the injury to the articulation as a whole. Not surprisingly, these classifications were relatively reliable at describing individual fracture patterns, but completely unreliable at predicting outcome. For years, the literature struggled with comparison reports of outcome using different treatment methods for what were regarded as 'similar' injuries, yet the failure to consider the wrist as a single unit (albeit with separate components) produced contradictory reports of success and failure, using the same treatment techniques because only one component of the injury was considered.

Our understanding of the wrist as a whole joint was significantly clarified by the work of Rikli and Regazzoni who described the '3 column concept' (see Fig. 13.1) [2]. This work focused on the mechanical function of the intact wrist in transferring load. Three distinct structural 'columns' were defined – two in the radius (the 'radial' and 'intermediate') and one in the ulna (the 'ulnar' column). Not only does this highlight the importance of the wrist as a multicomponent single unit, it also provides a framework for understanding how the wrist sustains skeletal injury and how best to reconstruct that in a functional manner. The 3 column concept has been further studied to examine the individual roles of each of the 3 columns, revealing the importance of the intermediate column as the main load bearing area in the skeleton of the wrist. The radial column

functions mainly as a stable buttress to support the carpus, whilst the ulnar column's function is as a pivot around which forearm rotation is produced – reinforcing the need for stability in this structure.

The work of Pechlaner concluded that almost all distal radial fractures (with the exception of dorsal rim avulsion fractures) could be reproduced by a hyperextension force [3]. Further biomechanical studies have shown that, in the neutral position, the load is transferred across the wrist to the central or volar portions of the scaphoid and lunate fossae (see Fig. 13.2a). When the wrist is in extension (the common position when this injury occurs) the load transfers to the dorsal rim of the scaphoid fossa and the central part of the lunate fossa (see Fig. 13.2b). This explains the 'dye-punch' injury, seen when the lunate splits the lunate fossa centrally and forces fragments of bone to be impacted.

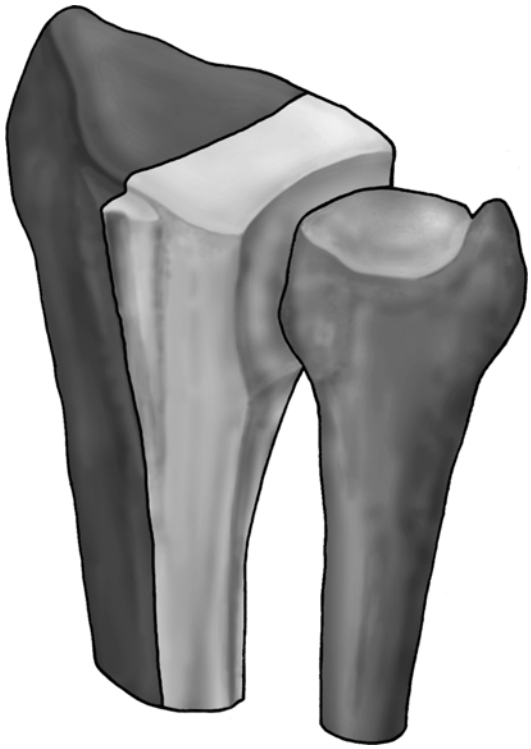


Fig. 13.1 The 3-column concept

Add to this the recognition that the intermediate column not only contains the keystone load bearing lunate fossa, but also the radial component of the distal radioulnar joint (DRUJ) – the sigmoid notch, which contributes to the arguably more important function of rotation of the forearm and it can be immediately understood that this area represents the key to fracture reconstruction, stabilization and function. More recent CT analysis of almost 100 articular fractures of the distal radius has confirmed that dorsally displaced fractures tend to involve the lunate fossa and sigmoid notch, whilst palmar flexed fractures involve mainly the scaphoid fossa [4].

It has also become apparent that outcome after wrist fractures is influenced by more than just the skeletal injury. Significant injury to other structures in the wrist will affect functional outcome. These injuries are often difficult to identify in the early stages after fracture and arthroscopic evaluation has revealed that the majority of wrist fractures have an 'associated' ligamentous or TFCC injury. The challenge is identifying which of these will be 'significant' and therefore which demand early treatment. It is clear that not all associated injuries need early treatment, but it is often unclear which ones demand an early interventional approach. The traditional approach has been to identify and repair each and every aspect of injury, but it is now becoming clear that a more discriminating approach is appropriate.

Injuries to the articular surface are regarded as 'more severe' injuries because of the poten-

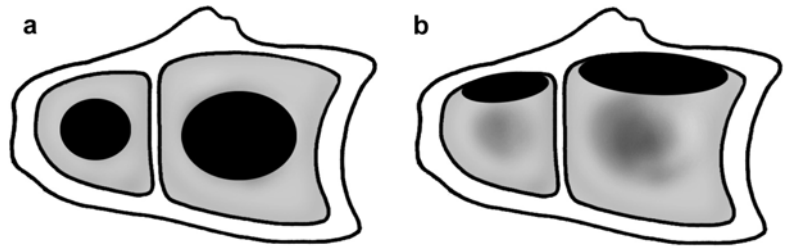


Fig. 13.2 Load pattern across the radiocarpal joint (**a**) the wrist in neutral at impact (**b**) the wrist in extension at impact

tial impact on ultimate function. The classification of Melone described fragmentation patterns in articular involvement and recognized the importance of these [5]. Earlier work by Knirk and Jupiter had concluded that an articular step of greater than 2 mm produced an unacceptably high risk of secondary degenerative arthritis that demanded reduction and stabilization [6]. That work was of paramount importance in identifying the need for accurate assessment of articular injuries, but it must be interpreted in the light of the imaging technology available in the mid-1980s and it has since become clear that the conclusions were flawed [7]. As such, it has to be accepted that the true influence of displacement of articular fractures remains unclear at this point in time.

Articular fragmentation patterns have now been more extensively studied and a number of key principles defined. It has only recently been recognized that the attachments of the extrinsic wrist ligaments around the rim of the distal radius seem to play a key role in determining the location of fracture lines [8]. Articular fractures have been found to occur more frequently between the known extrinsic ligament attachments, rather than at a point on the radial rim where the ligament attaches. As described above, when the wrist is injured in a position of extension (the common situation), the load transfer and impact occur at the centre of the lunate fossa. The volar extrinsic ligaments become taut in the position of wrist extension and their significant tensile strength either protects the area of bone on the radial rim at the site of ligament attachment from fracture, or (if the tensile load is significant enough) results in avulsion of this area of bone. Bone is stronger in compression than tension, so smaller fragments are avulsed around the rim of the radius, whilst larger fragments are created on the articular surface, due to the large compression force on the surface of hard bone.

These properties of bone and ligament change with age, especially in females [9]. The bone mass, density and microtrabecular structure of the distal radius show changes of osteoporosis, whilst the structure and mechanical properties of ligaments also change. This means that avulsion fractures are seen more commonly in younger patients, whilst bone failure (in compression) and mid substance ligament tears are more likely to occur in the older population.

This theory naturally leads to an understanding of why two broad categories of injury exist in the distal radius. These distinct sub-types are found almost exclusively in two different demographic groups – the elderly, mainly female, population and the young, active adult. Wrist fractures are also seen with some frequency in the skeletally immature, where they almost exclusively involve the distal radius alone.

The low energy classic ‘bending’ fracture occurs in an area of insufficient bone, after a minor stumble or fall in an elderly or osteoporotic individual. These injuries involve a progressive compression of the weakened metaphyseal area, meaning that articular fractures are seen less frequently in the elderly. The usual mode of injury is loaded hyperextension, when the dorsal surface (the ‘compression’ side of the fracture) is seen to fragment with this compressive force and effective bone loss often ensues.

In contrast, wrist fractures in the young active adult typically occur after significant violence, such as a fall at speed or from height, or as a component of multiple injuries in high energy trauma. In these circumstances, the bone quality is generally good and the fracture patterns reflect this, with shattering of the ‘hard’ bone (subchondral and articular), fragmentation of the ‘compression’ zone, but no progressive deformation with increasing force, as would be typical in an osteoporotic ‘bending’ fracture. In the young, healthy adult, bone failure is sudden and dramatic, not slow and progressive. Articular fragmentation is common and, although a normal thickness of articular cartilage is usually present at the time of injury, chondrocyte injury can occur due to the instantaneous compressive load on these cells at the moment of injury. Furthermore, the force pattern results in the carpus being ‘driven into’ the distal radius surface, resulting in fragmentation and impaction of fragments deep to the joint surface (the ‘dye-punch’ injury).

Fractures that enter the DRUJ will have a significant impact on function [10]. Loss of the forearm rotation arc, particularly supination, can produce a profound disability, with common secondary problems in the shoulder and neck. The DRUJ has two component skeletal structures; the sigmoid notch of the distal radius (mentioned above) and the distal ulna. The widespread acceptance of the 3-column concept has brought rightful attention to the distal ulna. Injuries that disturb the skeletal anatomy or stability of this bone will interfere with forearm rotation. Rotation occurs throughout the length of the forearm, not simply at either end and it should be regarded as a bicondylar 'joint' in its own right. There is no doubt that injury to the DRUJ affects forearm rotation, but abnormalities along the entire axis of the forearm must be sought when managing an individual with loss of forearm rotation. This chapter concentrates solely on forearm issues involving the DRUJ. Fractures of the distal ulna can involve the head, neck and/or styloid process and will be covered in more detail later in this chapter.

Injuries to other significant structures are also frequently seen in high energy fractures. The 'shearing' fracture causes an instantaneous dislocation of the radiocarpal joint and the initial radiographs may only reveal minimal subluxation of the joint. The presence of avulsed fragments of the attachments of the extrinsic ligaments bear witness to the energy required to create these potentially unstable fracture patterns.

Clinical Pearl

The 3 column concept is an invaluable aid in understanding the injury and planning treatment

The intermediate column is the key to successful reduction and stabilization of distal radius fractures

Injuries to structures other than the distal radius will have an impact on outcome

Injury 'types' are related to bone quality

Forearm rotation is critical to useful, comfortable upper limb function

Classification

Many classification systems have been described for fractures of the distal radius. In addition to the Frykman and Melone systems mentioned above, there are other classifications which merit more detailed descriptions.

The AO Comprehensive Classification described three categories of fracture sub-types for injuries throughout the skeleton. Type A – extra articular; Type B – partial articular; and Type C – complete articular [11]. These three sub-types also correspond to the deforming forces that cause them, namely bending, shearing and axial compression respectively. This system is highly detailed and an excellent tool for accurate classification of the distal radial injury, but is a difficult system to use in everyday clinical life. It is, however, recognized to be one of the most reproducible and reliable systems available for articular fractures of the distal radius [12].

Jupiter and Fernandez published their simple, yet comprehensive, classification system (the 'Universal System') in 1997 (see Fig. 13.3) [13]. This aimed to describe injuries based on their deforming force and also to provide guidance on the broad categories of appropriate treatment for each type. The five described fracture types included the same three sub-types of the AO Comprehensive Classification system (bending, shear and compression), together with carpal avulsions and high-energy 'combined' patterns of injury. Whilst this system does not allow an accurate sub-classification of many different fracture patterns, it provides an understanding of injury mechanism and how that produced the observed injury. This is an extremely useful system in everyday practice, although even it has been shown to have a low inter and intra observer reliability [14].

Clinical Pearl

No single classification system can predict outcome

The Universal System is useful in understanding the injury and planning the treatment

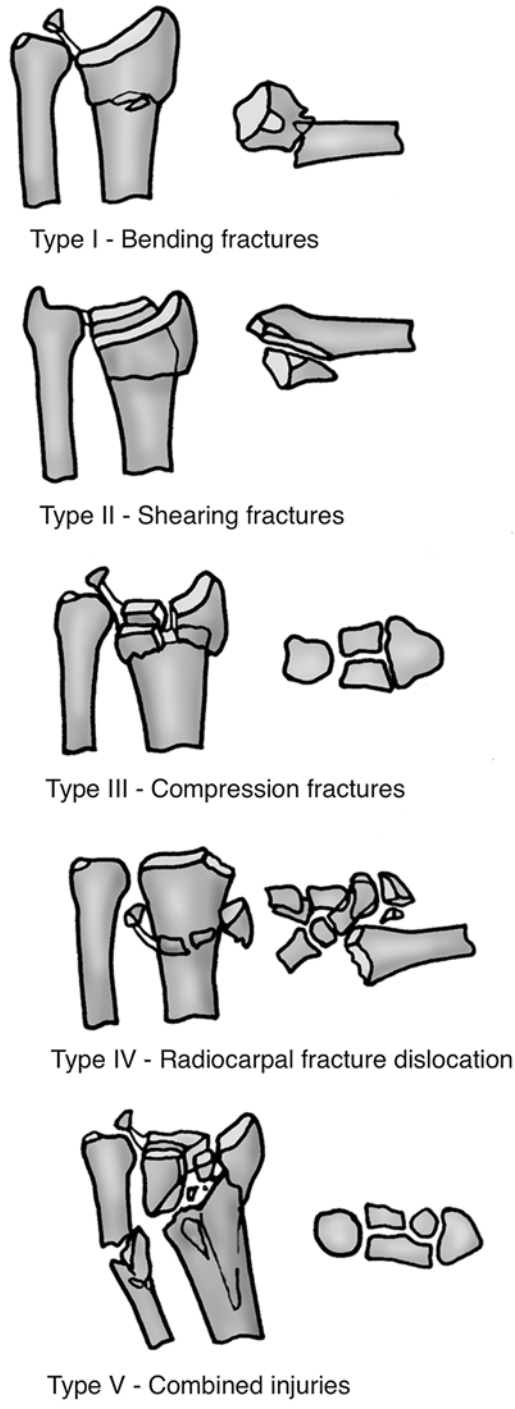


Fig. 13.3 Universal system of classification of distal radius fractures

What Influences Outcome?

Radiographic parameters have traditionally been used to predict instability (and therefore risk of further displacement), when the final position of fracture union was considered the most important single prognostic factor. Lafontaine and colleagues described radiographic risk factors for instability in distal radius fractures as;

- Dorsal tilt >20°
- Comminution
- Intra articular involvement
- Associated fracture of the ulna
- Patients older than 60 years [15].

The presence of three, or more, of these were said to lead to an increased risk of fracture instability, but contemporary clinical experience tells us that some of these factors are accompanied by a risk of instability when present in isolation. These radiographic factors were described at a time when it was believed that radiographs provided a reliable and repeatable measurement. We now know this is not the case and these 'measurable' factors must be considered in this light.

Radial shortening has been suggested as a single reliable indicator of late instability, but not poor outcome (at least in those patients over 55 years of age), whilst age alone has also been proposed as the only reliable predictor [16–18].

Articular displacement has been shown in many studies to produce a significant increase in the risk of development of osteoarthritis. However, there is no relationship between the *development* of degenerative change and the *symptoms* that would indicate further treatment [19].

These pieces of evidence relate to *radiographic* outcome, but what about *functional* outcome?

It would seem that an important radiological predictor of functional outcome is the presence of significant carpal malalignment [20]. This can be caused by loss of normal palmar tilt (in either a palmar or dorsal direction), which can result in a compensatory shift of the intercarpal axes (see Fig. 13.4). Associated intrinsic ligament injuries

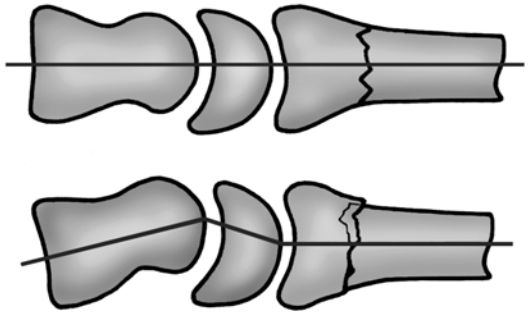


Fig. 13.4 Dorsal tilt results in compensatory carpal malalignment

can also result in carpal malalignment. Persistent dorsal tilt, of greater than 10°, has been shown to be associated with a poorer functional outcome in young adults, probably by virtue of the adaptive carpal instability it creates [21, 22]. More recent studies are in agreement with this and go further, by recommending that articular step-offs and gaps should be less than 2 mm, radial length should be restored to within 2 mm and carpal alignment should be normalized if acceptable function is to be expected [23]. This would suggest that it is, indeed, the adaptive carpal instability that is the functional predictor, rather than the angle of dorsal tilt. However, there are many circumstances where the true nature of a 'poor' outcome can be difficult to identify. Ulnar sided wrist pain (due to radial shortening, or previously quiescent ulnar abutment), loss of grip strength (due to reduced wrist extension or post-fracture pain), reduced supination (due to DRUJ malalignment or ligament injury), or the less identifiable features such as those complex issues involved in work-related injuries can all produce a 'poor' result. The presence of any of these factors will produce a different 'functional' outcome in different individuals (with different needs and demands). 'Outcome' is undoubtedly multifactorial and can be not only difficult to objectively assess, but also to predict, in an individual case, although this should not deter the recording of measurable outcome parameters.

The use of 'scoring systems' first became accepted in the early 1950s after the publication of the Demerit Scoring System of Gartland and Werley [24]. The Disabilities of Arm, Shoulder & Hand (DASH) and Patient-Rated Wrist Evaluation (PRWE) scores are also widely used in attempting to measure outcome. Even though these are all validated systems, they remain subjective and will vary with different patients and their individual needs. It is, however, vital to include subjective measurements in any reliable scoring system [25].

The question *"what is an acceptable reduction?"* can now be understood to be difficult to answer in objective and repeatable terms. Whilst it remains unclear as to exactly what we should be measuring in assessing functional outcome (partly because 'function' means different things to different people), absolute measurements of range of motion are a poor predictor of functional disturbance, whilst grip strength, hand dominance and residual wrist pain seem to be more reliably associated [26–28]. Age also plays a role when considering accuracy of reduction and subsequent union, with patients over 65 years tolerating malunion better than those under 65 years of age [29].

The effect of changes in bone mineral density can be difficult to separate from the effects of ageing and diminished functional demands. It would seem that osteoporosis is a significant risk factor for malunion, but that the functional effects of that malunion are often well tolerated. A randomized study of 85 such 'bending' fractures, treated by external fixation or plaster cast immobilization, resulted in a significant (50 %) rate of malunion, yet range of motion, ability to perform daily activities, and overall 'function' were not limited [19].

Clinical Pearl

Radiographic parameters do not predict outcome, particularly in low energy injuries

Certain fracture patterns may increase the risk of degenerative disease, but will not predict later symptoms

Carpal malalignment seems to be the most reliable predictor of functional outcome in the high energy injury

Imaging

Plain radiographs remain the mainstay for diagnosis of fractures of the distal radius. Significant information can be gleaned from plain radiographs, if certain views are obtained and certain principles understood.

The measurement of distances and angles on plain radiographs is notoriously difficult to reproduce, although there is evidence to suggest that computer assisted measurement on digital images is up to 31 % more accurate than traditional methods [30]. Plain radiographs must be standardized. Even a small degree of rotation of the wrist into pronation or supination will affect many of the indices that are commonly measured on plain radiographs.

In a standard lateral view, (with the x-ray beam perpendicular to the long axis of the radial shaft) a 'true' lateral view is obtained when the palmar cortex of the pisiform is seen to lie in the middle third of the interval between the palmar surfaces of the capitate and distal pole of the scaphoid (see Fig. 13.5) [31]. If the lateral view is malrotated, the indices for measurement show wide variation with palmar tilt varying between −4° and 15°, depending on the position of rotation [32]. This has obvious implications for the assessment of adequacy of reduction in a clinical case and for the accuracy of previously published data in studies measuring radiographic angles.

A number of standard angles and distances are routinely assessed on plain radiographs of the wrist (see Fig. 13.6). On the posteroanterior (PA) view, radial inclination, radial height, radial translation and ulnar variance are common and well-recognized landmark measurements. The concept of ulnar variance is interesting because it is actually radial variance that occurs, with the ulna remaining fixed throughout forearm rotation and the radius moving around it. Both the volar and dorsal rims of the distal radius will be seen on a PA view, with the dorsal rim normally more distal (see Fig. 13.7a, b). These landmarks are reversed in a dorsally displaced fracture, when the dorsal rim is the more proximal of the two (see Fig. 13.7c, d). The gap between the radius and the scaphoid can also provide information. If this gap is reduced, then it is likely that (in the absence of degenerative disease) the carpus has impacted into the distal radius, as the articular surface fractures and the fragments are depressed. A radioscaphoid gap of greater than 3 mm, after application of a bridging external fixator, represents over distraction [33]. Radial translation is important to recognize, because of the effects on DRUJ and the stability of forearm rotation (see later).

Fig. 13.5 The palmar cortex of the pisiform lies in the middle third of the area between the palmar surfaces of capitate and distal scaphoid pole in a 'true' lateral x-ray. The solid line represents the distance between the palmar surfaces of the capitate and distal scaphoid pole. The dotted lines represent this distance divided into thirds.

Normally, the only measurement commonly made on the lateral view is palmar tilt. As noted above, this measurement is affected by the rotational alignment of the wrist. One critical osseous landmark to recognize on a plain lateral radiograph is the 'teardrop' (see Fig. 13.8). This represents the volar rim of the lunate facet – a key area in fracture stability, due to the origin of the palmar extrinsic ligaments. This area is critical in restoring stable anatomy to the radius (and therefore the wrist).

Compression injuries of the articular surface can result in fragment depression (a 'dye-punch' injury) or fragment separation. The distance from the dorsal to palmar rims of the distal radius on

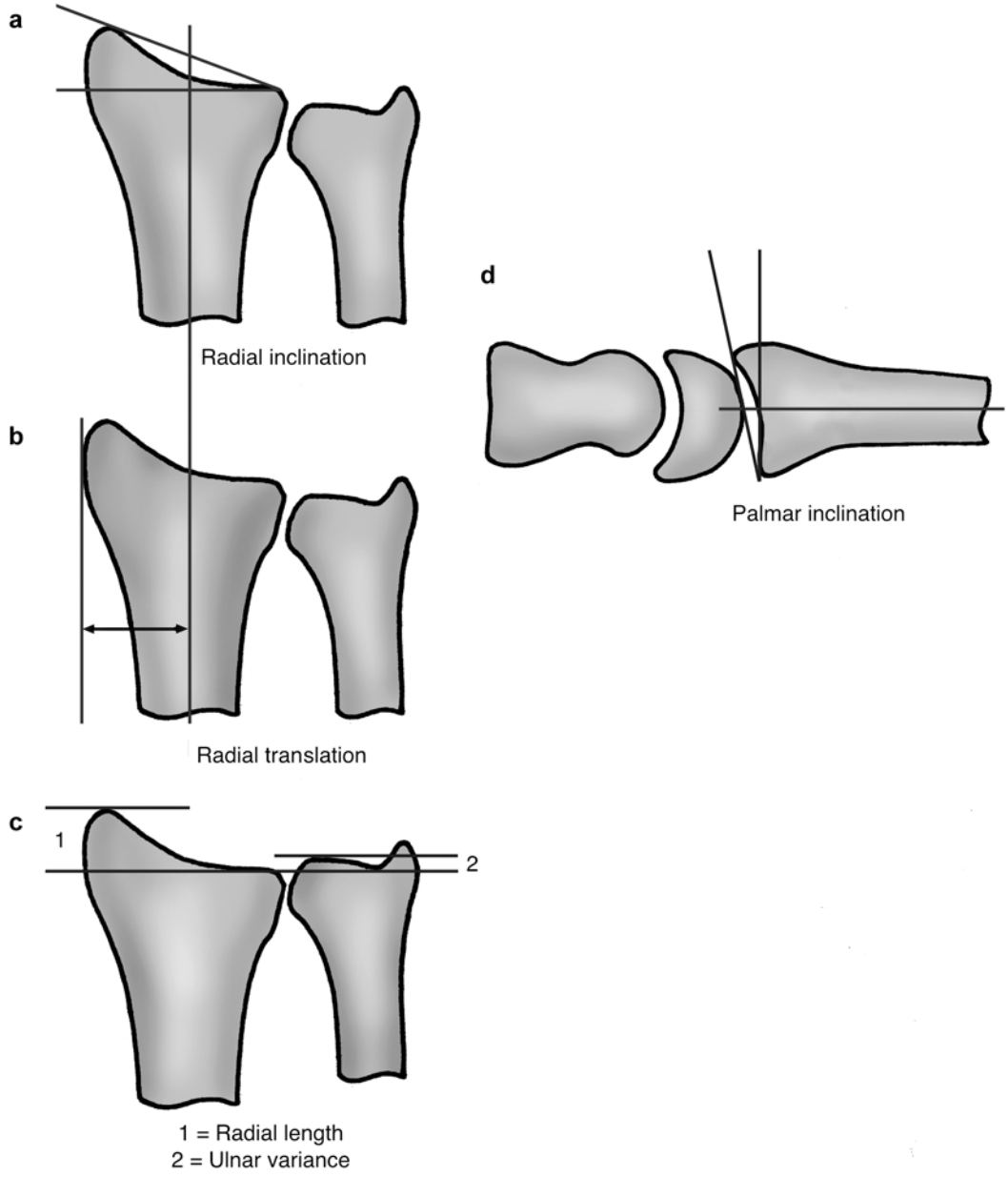


Fig. 13.6 Standard radiographic angles to measure on wrist x-rays. (**a**) Radial inclination (**b**) Radial translation (**c**) Radial height (RH) and ulnar variance (UV) (**d**) Palmar inclination

the lateral view is defined as the 'AP distance'. This parameter is often widened in an axial load injury, although the actual fragment displacement may be difficult to identify (see Fig. 13.9a, b). An assessment of AP distance will draw attention to, and help identify, this possibility.

Fractures involving the lunate fossa also involve the sigmoid notch (and therefore the DRUJ). A true lateral radiograph will show the lunate fossa, but not the scaphoid fossa (since it lies at 10–15° to the x-ray beam). A 20° angled lateral view is required to show the scaphoid fossa (which constitutes 50 % of the distal radius articular surface) (see Fig. 13.10a, b).

The acceptance of the angled lateral view can be extended to perform a similar 10° angled pos-

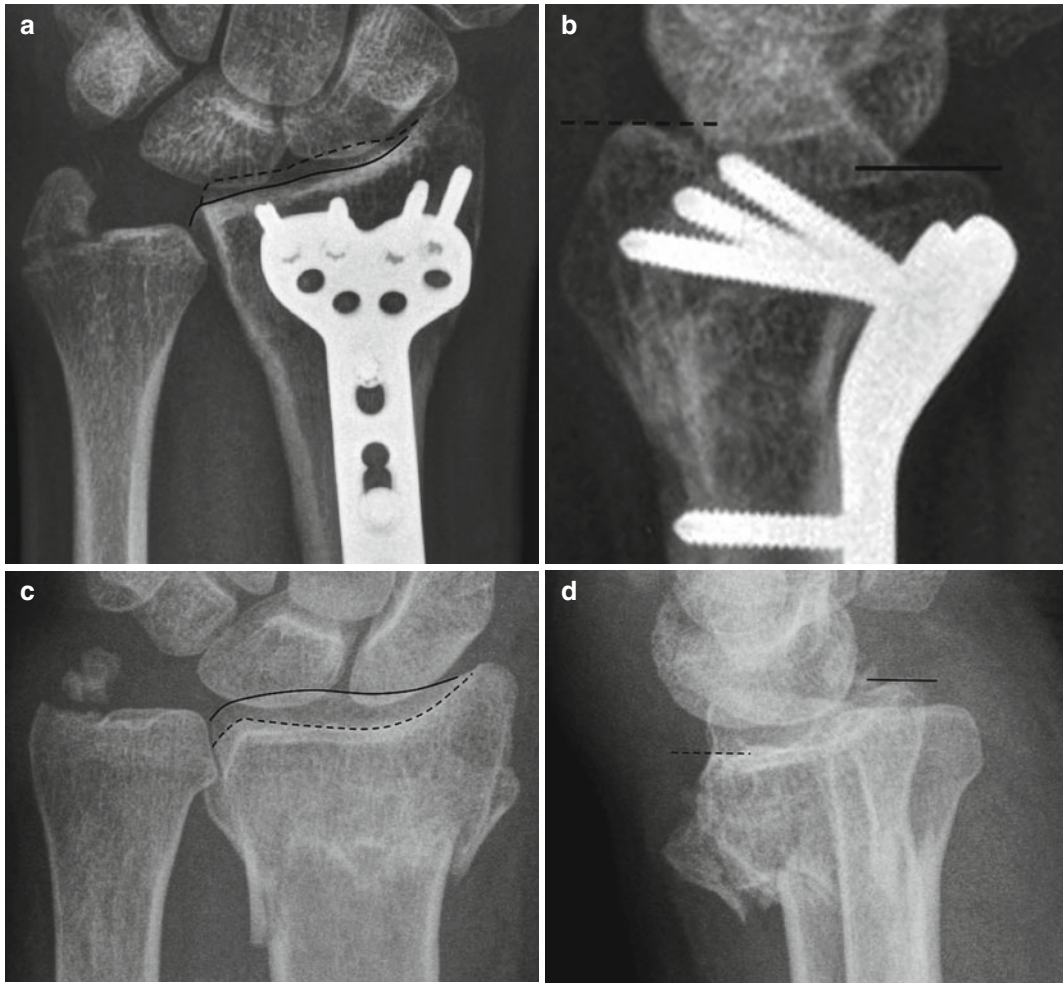


Fig. 13.7 Dorsal (*dotted line*) and volar (*complete line*) rims of the distal radius (**a**) Normal PA view (**b**) Normal lateral view (**c**) PA view of dorsally displaced fracture (**d**) Lateral view of dorsally displaced fracture

Fig. 13.8 The 'teardrop' outlined on a lateral radiograph indicates the volar rim of the lunate facet

teroanterior (PA) view, to both accurately show the presence of an articular step-off and/or to confirm the position of subchondral implants, such as screws and K wires [34].

CT scans are used with increasing regularity to accurately outline the nature of the fracture pattern and there is no doubt that they provide more accurate assessments of displacement than plain radiographs [35, 36]. Modern 3D reconstruction images allow the surgeon a true insight into the exact fracture pattern and fragment displacement. Whilst it is prudent to employ contemporary and sophisticated imaging to accurately understand fracture patterns, there is a risk that this will lead

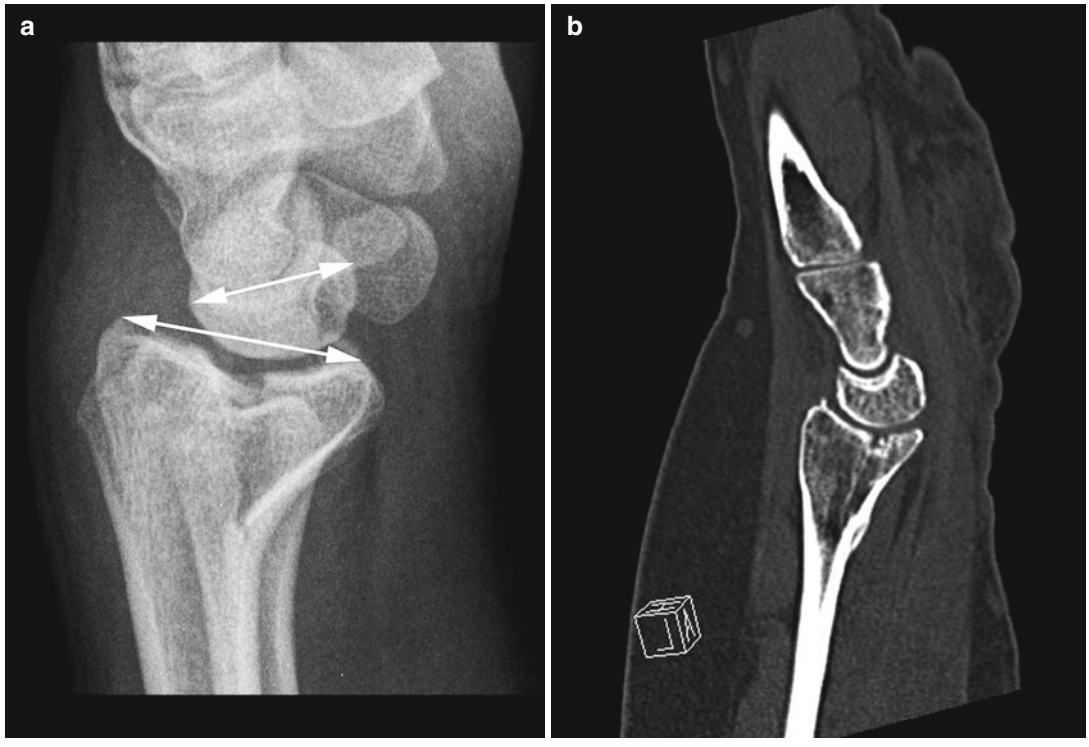


Fig. 13.9 Widening of the AP distance of the distal radius (**a**) Lateral radiograph (**b**) CT scan. The *short arrow* represents the AP diameter of the normal lunate. The *long arrow* represents the abnormally widened diameter of the lunate fossa

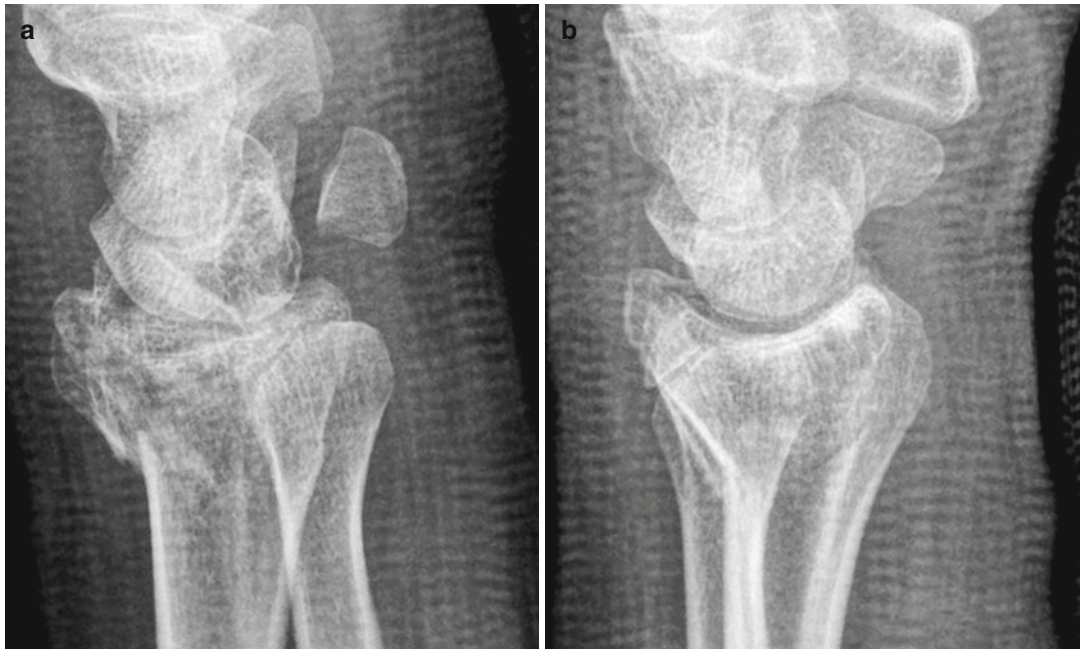


Fig. 13.10 Standard and angled radiographs (**a**) Standard lateral (**b**) Angled lateral

to a situation where the wealth of information available on a plain radiograph (which is usually available at an early stage) may be overlooked. It is essential that plain radiographs are revisited and reassessed after a CT scan has been performed, so that they can be fully understood and better interpreted in future cases.

Other imaging modalities such as ultrasound, arthrography and MRI have no place in identification and assessment of the acute skeletal injury, but contribute enormously in the subsequent identification of associated injuries.

Clinical Pearl

Plain radiographs often contain enough information with which to assess the injury pattern

However, the 3 dimensional nature of the distal radius and wrist must be considered when assessing 2 dimensional plain radiographs

CT scans are indicated to clarify certain fracture patterns in order to make a decision about surgical management

Extra Articular Fractures

An extra articular fracture is one in which the fracture line does not involve either the radiocarpal or distal radio-ulnar joints. These injuries are common, almost exclusively closed and usually involve the radial metaphysis. Higher energy injuries can extend into the diaphysis, resulting in highly unstable fragmentation patterns. Extra-articular fractures are often regarded as simple fractures, in which an acceptable functional outcome will almost always result. This is not necessarily the case and the effects of these fracture patterns on the wrist as a whole, not just the radius, must be considered.

Shortening of the radius will alter the relative proportion of load borne across the wrist through the radius and distal ulna. Loss of only 2–3 mm of radial length will double the load through the distal ulna and this will result in significantly increased pressure through the TFCC, as it 'stretches' over the ulnar head, threatening its integrity, longevity and ultimately, stability. Ulnar sided wrist pain is more apparent in pronation (when the ulna is functionally 'longer'). This wrist position is the position of function for many activities, particularly with the dominant hand such as writing, cutting, using cutlery or typing on a keyboard.

Radial shortening will also result in incongruity of the DRUJ (as the sigmoid notch alters its position relative to the ulnar head). The DRUJ ligaments alter their tension, the contact pattern across the joint changes and forearm rotation can either be restricted (most notably in supination) or painful [37]. Loss of palmar tilt will alter the pattern of load transferred from the carpus to the distal radius [38]. The more the palmar tilt is lost, the more load is concentrated on the dorsal rim of the distal radius, producing more rapid degeneration. The effects of loss of palmar tilt are, if anything, more apparent on the carpus than on the load pattern of the distal radius. Increasing dorsal tilt of the distal radius will alter the capitolunate and radiolunate axes, resulting in an adaptive carpal instability (see Fig. 13.4). The existence of this carpal deformity is thought to have some considerable significance in outcome and is often used as a criterion for reduction of a fracture, or for treatment of established malunion. Tilting of the distal radial fragment also changes the relationship of the component parts of the DRUJ, which can adversely affect the joint's performance [39].

Radial translation of the distal fragment will have an effect on the stability of the forearm rotation mechanism. Radial translation results in a slackening of the interosseous ligament (IOL) with the result that DRUJ stability can be affected (see Fig. 13.11a–c). Reducing the translated distal fragment re-tightens the IOL and restores the correct tension in the forearm ligaments.

Most extra-articular fractures can be managed in a cast, once a stable reduction has been achieved. There is no proven advantage in restricting forearm rotation in a sugar-tong splint when compared to a standard short-arm cast [40]. Similarly, there is no convincing data to support immobilization in a particular extreme of either pronation or

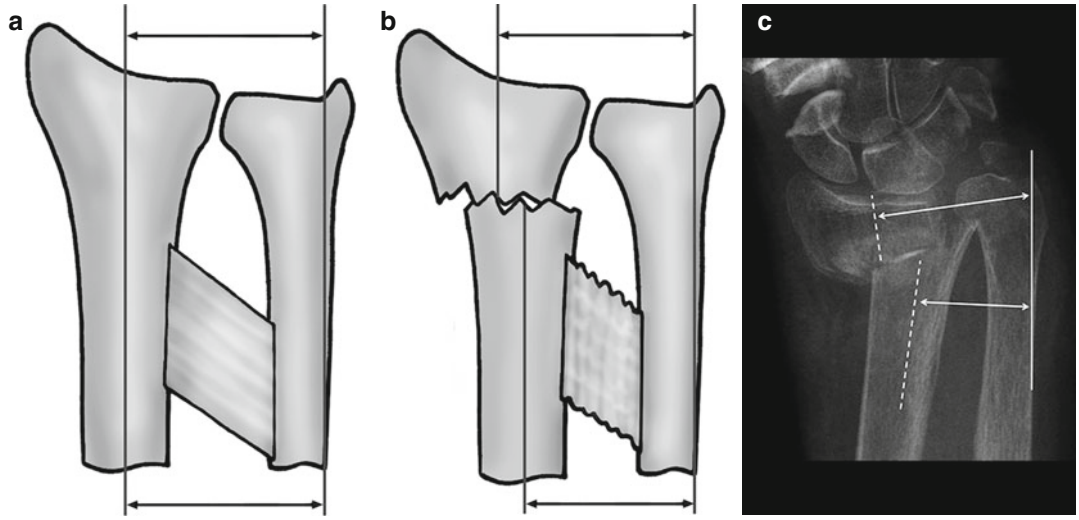


Fig. 13.11 The effect of radial translation on DRUJ stability (**a**) Normal anatomy and tension in the distal oblique band (DOB) (**b**) Radial translation slackens the DOB and produces instability (**c**) Radially translated fracture with slackening of DOB. The solid line represents the first reference point for measuring radial translation – the medial cortex of the ulna. The dotted line represents the second reference point for measuring radial translation – the centre of the radius

supination [41, 42]. Extreme wrist flexion should be avoided, to reduce median nerve problems. Non-operative treatment demands rigorous, disciplined and regular vigilance of radiographs, cast condition and clinical features. It would be regarded as mandatory to review each of these injuries at a time when limb oedema was reducing and the cast may have lost its close-fitting support of the reduction. This is usually at a time between 10 and 14 days after injury but unstable fracture patterns are often less predictable and can displace at any time before union. These fractures should be monitored more closely and more frequently.

There are some undisplaced and stable injuries which can be treated by cast removal at an earlier stage (4 weeks), although a removable splint should be worn for a minimum of 2 more weeks in these cases.

Assistance with stability, for instance using percutaneous K-wires, may be required, if the fracture pattern predicts instability or the reduction can be achieved but not maintained. Open reduction and internal fixation using an angularly stable volar plate is an increasingly popular method of treatment of these injuries, especially if a period in cast is deemed unsuitable or inconvenient for the particular patient. Early open reduction and internal fixation, with an angularly stable volar plate, has been shown in an elegant and well-designed study to provide better outcomes that outweigh the surgical risks, than cast immobilization in those unstable fractures which are initially well reduced [43]. However, there is no noticeable difference in long-term outcome between the two methods of internal stabilization of percutaneous K- wires or internal fixation, so the choice remains a personal, but informed, one [44]. External fixation (with or without additional percutaneous K-wire fixation) is becoming a less popular method of treatment for displaced extra articular distal radius fractures, although there is weak evidence to support the assertion that the outcomes are better than with simple casts alone [45].

Whilst adjunctive non-surgical treatments remain unproven, there has been some encouraging evidence to support the use of low-intensity ultrasound to accelerate healing in a randomized series of 61 distal radius fractures treated in cast [46]. Pulsed low-intensity ultrasound was applied for 20 min each day for the period spent in cast. Loss of reduction was less frequent in the treatment group, with stability achieved at 12 days rather than 25 days in the non-treatment group. In spite of this well designed and conclusive study, this method has not become widely practiced.

Clinical Pearl

Extra articular fractures do not always have an acceptable outcome

There are long term pathomechanical effects associated with permanent radial shortening and dorsal tilt

Radial translation affects tension in the interosseous membrane and DRUJ stability

Most extra articular fractures can be managed in cast

Intra Articular Fractures

Reliable evidence exists to suggest that articular fractures that heal with an incongruent step-off on the joint surface are more likely to cause persistent pain and to develop radiographic changes of secondary osteoarthritis some years later [47–49]. However, no reliable data yet exists to tell us how large such a step-off needs to be before it becomes significant. It would seem that a step-off greater than 2 mm would be regarded as significant in most studies, although a smaller step-off (of greater than just 1 mm) has also been shown to be significant for the development of degenerative disease [50]. One study reported radiographic deterioration of one grade in 27 % of patients treated by internal fixation at 1 year [51]. Only 7 % of this study group had an articular step-off of greater than 2 mm, so the inference is either that smaller step-offs can still result in articular degeneration, or that the energy of impact is itself significant enough to cause these changes. Chondral injuries of the distal radius have been identified in 32 % of fractures in young adults in arthroscopic studies [52].

It is also unclear as to when radiographic changes of degeneration are likely to have an impact on function. At an average of 7.1 years after injury, osteoarthritis (seen on radiographs and CT scans) was present in 76 % of cases in Catalano et al.'s study [49]. Whilst there was a strong relationship between these changes and persisting articular incongruity, functional outcome did not correlate with the magnitude of these articular step-offs and all patients had a good or excellent functional outcome.

There is longstanding evidence to support a program of early movement after articular fractures in an attempt to reduce osteopenia, stiffness, and to stimulate cartilage repair [53, 54]. Early movement can only be allowed if the fracture fragments are stabilized internally at an early stage and studies have been published which emphasise the importance of anatomical reduction, stable internal fixation and early active and passive movement [55]. However, as noted earlier, an anatomical fixation does not always lead to a good functional outcome and vice versa.

Well-designed, large, prospective, randomized studies have been undertaken, but it remains difficult to reach a specific conclusion.

A randomized trial of 144 articular fractures, treated by either external fixation with percutaneous pins or internal fixation with either volar or dorsal plates, concluded that results for the plating group were superior at 24 months [56].

Randomization in a trial of 62 AO type C fractures treated by mini open reduction, percutaneous pins and external fixation or internal fixation with a dorsal plate, was abandoned because of the unacceptably high complication rates seen in the plating group.

No significant differences were found in a randomized trial of 179 articular fractures treated by either external fixation with percutaneous pins or internal fixation at an average 2 year follow-up [57].

These studies all had similar designs, yet completely different conclusions and recommendations, (which supports the significant influence of the spectrum of substrate), although none of them included the types of implant now regularly used in contemporary practice [58]. More recent studies employing angularly stable implants have reported excellent radiographic and functional results. A prospective series of 87 articular fractures in patients with an average age of 49 years (51 % of which were AO type C) reported functional grip strength and range of motion of 85 % of the uninjured side at 12 months [59]. Similar results were reported in a series of 114 fractures using a different volar locking plate [60].

It may seem difficult to reach any evidence-based conclusions, but several key points can be summarized from the current literature:

- It is reasonable to strive to reduce articular fragments to produce a joint surface with no step-off greater than 2 mm.
- Most articular fractures are unstable and will require some form of stabilization with percutaneous or internal fixation, or be allowed to heal with displacement.
- Selection of a particular form of stabilization, or type of implant is less important than working to biomechanical principles of surgical reconstruction.
- Comminuted metaphysis will not provide stable support for articular fragments and requires stable bridging fixation.
- Plain radiographs may not be as reliable as CT scans in assessing articular incongruity.
- Complications occur at times with all surgical techniques.

When the decision has been made to treat a displaced articular fracture by internal fixation, thought must be given to the fracture pattern, the best surgical approach, the most appropriate implant and the details of the surgical plan. Consideration of the 3-column concept is helpful in formulating a surgical plan.

The intermediate column is critical in restoring anatomy and stability to the main load bearing areas of both the radiocarpal and distal radioulnar joints. Compression fractures of the radial articular surface disrupt this column and surgical reconstruction should begin in this area.

The choice of surgical approach will depend upon which specific implant is selected. The choice of implant is made after consideration of the fracture pattern and method of fragment reduction. In the past, a dorsal approach was favoured because the application of a dorsal implant addressed the area of compression and effective bone loss in the fracture. More fractures are dorsally displaced and an approach through the floor of the III extensor compartment allowed direct visualization and reduction of the fracture fragments, as well as inspection of the restored articular surface via an arthrotomy [61]. However, dorsal implants were associated with significant and frequent complications, relating to the proximity of the implant to the extensor tendons and although the surface finish was manipulated and re-designed, this was insufficient to eradicate this problem [55, 62]. This approach also often required additional cancellous bone grafting to fill the void and further stabilize the reduction.

The advent of angularly stable implants for application on the palmar surface of the distal radius ("locking" plates), which were anatomic in their design and featured multiple potential combinations of screw arrangements, radically changed the management of a large proportion of displaced distal radius fractures. These implants are not without potential complications. Placement of the implant too distally on the volar surface of the distal radius, beyond the 'watershed line' (the origin of the volar extrinsic ligaments) puts the flexor tendons at risk of attrition rupture. The use of screws that are too long can cause irritation and rupture of the extensor tendons from the tips of the screws. The identification of crepitant active movement of flexor or extensor tendons which lie adjacent to a metal implant or screw tip should act as a warning of impending attrition and should be considered as an indication for early implant removal [63]. The curved dorsal surface of the distal radius must be taken into account when assessing appropriate screw length on a lateral radiograph. Twenty-five percent of screws placed in a series of 46 volar plates were reported to penetrate the dorsal cortex by up to 6 mm in one study [64]. Direct measurement of the desired screw length with a depth gauge is a much safer method of selecting screw length, although use of the 'skyline' view during surgery is an accurate way to help avoid this problem [65]. Careful and correct positioning of the implant, in combination with accurate selection of appropriate screw lengths, will reduce these risks dramatically and allow the implant to perform as it was designed. These implants perform best when the distal row of screws are positioned to support the subchondral bone of the distal radius [66]. This area of bone is hard, even in osteoporotic patients and a row of angularly stable, locked screws will act as a stable 'shelf' for the articular surface to rest on. The anatomic design of many implants

allows the placement of locked screws in such a position that normal anatomy is restored and the orientation of a stable row of locking screws just underneath the subchondral zone will continue to support the reduced articular surface whilst healing occurs. Bone grafting in the dorsal defect is rarely indicated.

Much attention has been paid to articular fractures with large, identifiable and reducible fragments. Fractures involving the articular rim, especially those involving the volar rim, demand equal vigilance in identification, reduction and stabilization. These injuries can easily be missed, with disastrous late consequences of redisplacement and carpal subluxation [67]. Such volar shearing fractures require the same stable internal fixation as fractures with larger fragments, but this can be technically difficult because of fragment size and location around the 'watershed line'.

There remain some unusual fractures that are difficult to reconstruct anatomically because of their comminution or poor bone quality. Combined involvement of articular surface, metaphysis and diaphysis also presents difficulties to anatomical reconstruction. These represent a particular therapeutic challenge where limited goal setting is an option. One approach that has proved relatively successful in those injuries involving combined fragmentation of articular, metaphyseal and diaphyseal bone, is the use of an internal distraction spanning plate applied through three small dorsal incisions to the radial shaft and middle metacarpal [68]. These injuries involve significant energy transfer and the vascularity and healing of soft tissues will be threatened by a wide surgical approach and fragment dissection. The use of small surgical wounds and judicious placement of K-wires, screws and/or bone graft to support selected fragments is stabilized by the internal distraction provided by the long bridging plate. The plate is removed when healing has occurred (usually around 4 months) and therapy begun. In the described series of 22 patients, a functional range of motion was restored with an average flexion/extension arc of 112° and forearm rotation arc of 153° [68].

Articular fractures of the distal radius represent a challenging injury to manage effectively. They usually involve the harder area of subchondral bone and, as such, are often seen in younger patients with good bone quality, high-energy injuries and high expectations of outcome. Their high-energy nature is associated with additional injury to other structures in the wrist and this can have a significant impact on outcome.

Clinical Pearl

Articular fractures are likely to be unstable

Persistent articular incongruity is a risk factor for persistent pain and degenerative disease

CT scanning is more accurate than plain radiographs in assessing fragment displacement and joint incongruity

There is no reliable evidence to determine how much articular incongruity is 'acceptable'

Fractures of the Distal Ulna

The distal ulna forms one of the 3 columns of the wrist. Its importance cannot be underestimated as both part of the DRUJ and as the essential weight bearing fulcrum for rotational stability of the forearm (see Fig. 13.12).

Fractures of the distal ulna are frequently encountered with a distal radius fracture, but are often simple injuries to the tip of the ulnar styloid. In a study of 130 distal radius fractures, 71 (55 %) were noted to have a coexistent ulnar styloid fracture, of which 28 (21 %) involved the base of the styloid [69]. These injuries are usually ignored, but it is essential to understand, recognize and treat those distal ulnar fractures of significance. DRUJ instability, consequent to an unstable fracture of the distal ulna, has been reported in up to 37 % of distal radius fractures, yet fewer than 10 % of distal radius fractures require surgery on the distal ulna [70, 71].

Distal ulnar fractures were recognized in the classification of Frykman but not independently classified until 1995, when four separate

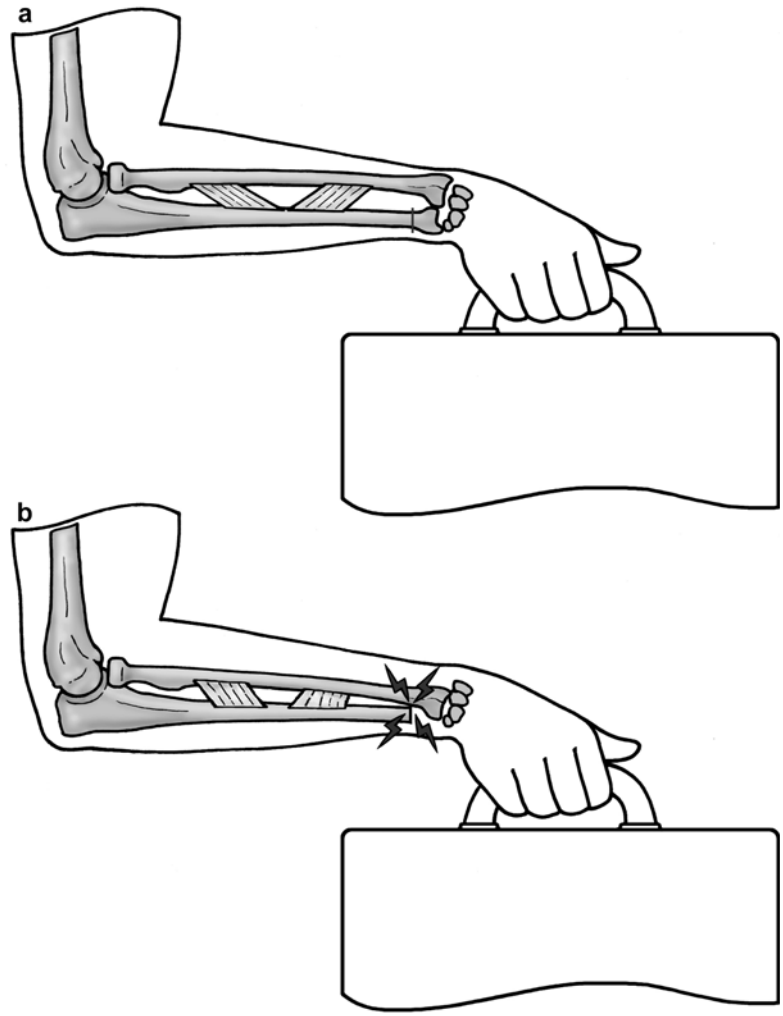


Fig. 13.12 The weight bearing function of the distal ulna (**a**) The normal situation (**b**) Following excision of the lower ulna

fracture types were described which involved the neck, head, styloid and multiple parts (see Fig. 13.13) [72].

Normal pain free joint function will be affected by any articular fracture involving the ulnar head, whilst DRUJ stability will be threatened by those fractures which either result in articular displacement or avulsion of those stabilizing structures which attach to the distal ulna (most usually the distal radioulnar ligaments and foveal insertion of the TFC). The superficial portion of the TFC attaches to the ulnar styloid tip and body and plays little part in stability. However, the deep portion inserts directly into the fovea of the distal ulna (see Fig. 13.14a). Displaced or unstable fractures of the base of the ulnar styloid (especially those oblique fracture patterns involving the metaphysis) are more likely to result in TFC detachment and DRUJ instability. This is not an exclusive

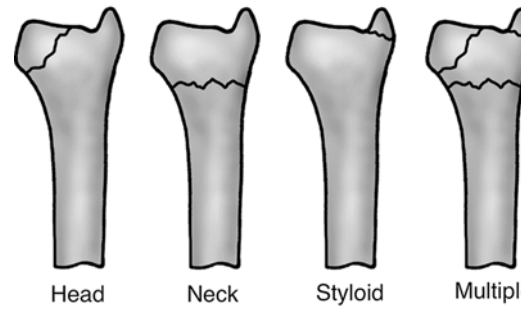


Fig. 13.13 Classification of distal ulnar fractures; Head, Neck, Styloid and Multiple parts

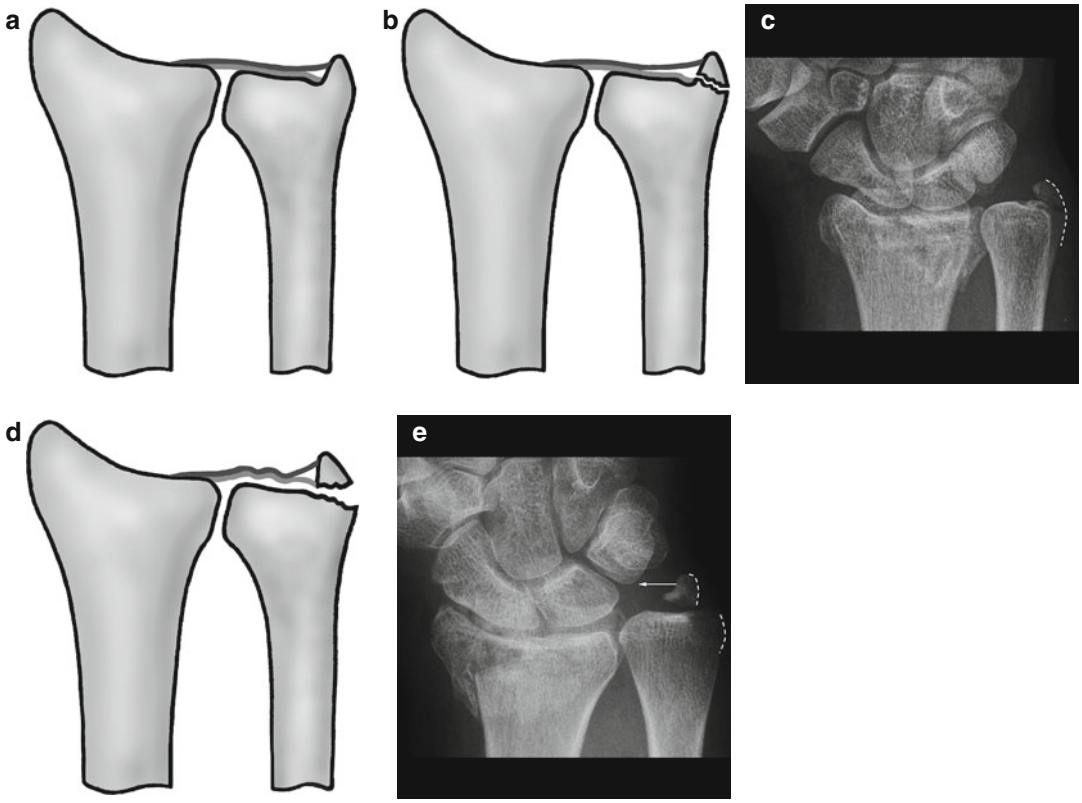


Fig. 13.14 Pathoanatomy of the attachments of the TFCC. (**a**) The attachment of deep and superficial portions of the TFCC. (**b**) Distal styloid fracture with intact deep fibres of the TFCC. (**c**) Radiograph of distal styloid fracture. The dotted line represents the medial cortex of the ulnar styloid. (**d**) Proximal styloid fracture with disruption of the deep fibres of the TFCC. (**e**) Radiograph of proximal styloid fracture showing radial displacement of styloid fragment. The dotted lines represent the medial cortex of the ulnar styloid fragments. The arrow represents the direction and amount of displacement of the distal fragment of ulnar styloid

relationship, merely a clinical suspicion, but these fractures should raise the question about DRUJ stability whenever they are identified. The distal oblique band of the interosseous ligament is an important, yet 'invisible' stabilizing structure of the radioulnar axis. When all distal radioulnar structures around the joint are disrupted, integrity of the distal oblique band will still prevent clinical DRUJ instability [73]. Displacement of the avulsed styloid fragment to the radial side is usually an indication that it is attached to the (torn) TFC and that the interosseous ligament has disrupted sufficiently to allow the fragment to displace radially (see Fig. 13.14b–e). Similarly, widening of the DRUJ on plain x-ray, in combination with an ulnar styloid avulsion is highly suspicious of traumatic DRUJ instability with distal oblique band insufficiency. TFC injuries can occur without fracture to the ulna, or in combination with an 'innocent' ulnar styloid fracture pattern, but these instances are unusual.

Distal ulnar fractures should be treated when they have an identifiable impact on DRUJ congruity or stability. Excellent results have been reported for different distal ulnar fracture patterns using locking plates and specific anatomically contoured locking implants are now available for stabilization of the entire spectrum of distal ulnar fracture patterns [74, 75].

Combined fractures of the distal radius and ulna (other than styloid fractures) represent a special group of injuries. These fractures are associated with a high incidence of radial non-union, if the ulna is not also stabilized [76]. Both bone fractures should be considered as forearm fractures that have occurred near the wrist. As such, both

bones should be stabilized so that forearm rotation can be restored and bony union rates improved.

Non union of the ulnar styloid is seen frequently and is classified as Type 1, when the DRUJ is stable and Type 2, when the DRUJ is unstable [77]. Symptomatic Type 1 non-unions respond to excision of the distal fragment, whilst symptomatic Type 2 non-unions require surgery, involving bone grafting and internal fixation of the non-union, to restore stable functional forearm rotation.

Distal ulnar fractures and symptomatic non-unions can influence functional outcome. Only a minority require active intervention, but those that do can be successfully treated by an understanding and application of the specific principles outlined above.

Clinical Pearl

Fractures of the ulnar styloid are frequent

A minority of ulnar styloid fractures require active treatment

Fractures of the head of the distal ulna are articular fractures

Stability of the DRUJ must be considered in these fracture patterns and assessed after radial stabilization

Fractures of both distal radius and distal ulna should be considered as 'distal forearm fractures'

Associated Injuries

Associated injuries occur frequently in wrist fractures. An arthroscopic study of injuries associated with intra articular distal radius fractures identified TFCC tears (53 %), scapholunate ligament tears (21 %) and lunotriquetral ligament tears (6.7 %) [78].

Certain radiological signs and injury patterns will suggest, but not confirm, possible associated soft tissue injury (Table 13.1). More sophisticated investigations, such as plain arthrography, MRI arthrography and arthroscopy are extremely useful in securing a diagnosis in the uncertain case.

Table 13.1 Radiographic clues of associated injuries

Scapholunate ligament injury
Ring sign of flexed scaphoid
Scapholunate gap >3 mm
Step in Gilula's arcs I and II
Scapholunate angle >80°
Displaced radial styloid fragment
Ulnar positive variance >2 mm
Lunotriquetral ligament injury
Lunotriquetral gap >3 mm
Step in Gilula's arcs I and II
TFCC injury
Widening of DRUJ
Radial displacement of ulnar styloid fragment
Subluxation of distal ulna

Once an associated injury has been identified, a decision must be made whether or not to actively treat that part of the injury complex. In the early acute situation, it can be difficult to know if that particular injury will be symptomatic once the distal radius fracture has healed and rehabilitated. For that reason, some authors recommend early and aggressive surgical repair of each and every identified injury, whilst other advocate a more discriminating approach.

An assessment of radioulnar laxity is not specific enough to identify DRUJ instability that will be symptomatic, even after radial stabilization [79]. The stabilizing effect of the intact distal oblique band of the interosseous ligament will not be appreciated in this passive test. Compression of the ulna against the radius, followed by passive rotation of the forearm through a complete arc of pronation and supination, will produce a 'clunk' if the TFCC and interosseous ligament are disrupted, indicating a likelihood of clinical DRUJ instability. In these cases, stabilization of the injured component in the DRUJ is indicated [80].

Acute scapholunate (SL) ligament tears can be difficult to diagnose, especially when surgical fracture stabilization is performed through a volar approach. In these cases, arthrotomy is not permitted and there is no opportunity for direct ligament inspection. A recent study examining the early (1 year) effects of outcome of SL ligament tears in association with 51 distal radius fractures in young adults revealed that complete

ligament tears progressed to static radiographic changes and greater pain within 1 year, although range of motion and grip strengths were similar to those patients with a partial SL ligament injury [81]. Furthermore, the presence of ulnar positive variance of >2 mm on the initial x-ray was strongly predictive for a complete SL ligament tear. A further study of 40 intra-articular distal radius fractures employed arthroscopy in half the cases to aid articular reduction and identify associated soft tissue injuries [82]. Nine cases (45 %) of SL ligament tears were diagnosed and all were repaired. The outcome in those patients who had additional arthroscopic evaluation was universally improved, suggesting that acute SL ligament tears, when identified, should be repaired early in young adults with distal radius fractures.

Median nerve dysfunction after wrist fracture is not uncommon, but an important distinction must be made between a direct injury to the median nerve itself, or an 'acute' carpal tunnel syndrome [83]. Symptoms that do not begin until the hand has become swollen are most likely to be due to an acute carpal tunnel syndrome. All others are likely to be due to direct nerve trauma at the site of fracture. Any exploration of the nerve must occur at the appropriate location.

Clinical Pearl

Associated injuries are frequent

A high index of suspicion is required to identify them

There is little evidence available to accurately define the likely long term effects of these injuries

Median nerve dysfunction occurs both in the carpal tunnel and at the level of the wrist fracture

Insufficiency Fractures

The majority of insufficiency fractures are of the bending type. They represent the commonest fracture of the upper extremity in those over 65 years. Women over 60 are six times more likely than men of the same age to experience this injury [84].

Distal radius fractures in patients with osteoporosis or low bone mineral density (BMD) are likely to redisplace after closed reduction and splinting [85]. This becomes more likely with advancing age. Redisplacement usually occurs in the first 2 weeks, such that remanipulation and casting is unlikely to be successful at any stage, without supplementary stabilization [22]. Indeed, the redisplacement rate is so universal that it has been stated that reduction of these fractures is futile in the frail, very old, dependent or demented patient [86].

Traditionally, supplementary fixation has been provided by means of percutaneous K-wires because of the low invasiveness, surgical trauma, cost and also the simplicity of the procedure. However, there is good evidence to suggest that K-wires are ineffective in maintaining radial length in insufficiency fractures and that if there is loss of radial length on the initial pre-treatment x-rays (as measured by ulnar variance), then percutaneous pinning is unlikely to improve that [87]. This leads to a logical recommendation that, if any stabilization is deemed necessary, internal fixation with a palmar locking plate should be considered. Excellent clinical results have been reported using palmar locking plates in patients over 60 years of age with redisplacement and as a successful primary treatment in insufficiency fractures, with similar outcomes and complication rates to younger patients [88–91]. Angular stability is essential, since non-locking plates have been shown to produce poorer radiological outcomes [92].

Insufficiency fractures are very different to similar fracture patterns in young adults. Functional outcome has been closely linked to the position of radiographic union in the young adult, yet no such relationship exists in the older population until extreme deformity of radial shortening >6 mm or dorsal tilt >20° occurs [23, 27, 93–100].

It is likely that increasing numbers of insufficiency fractures of the distal radius will be treated by open reduction and internal fixation due to changing demographics, improved general

health and increasing independence of people over the age of 60 years. However, it must be remembered that minor degrees of malunion do seem to be tolerated in this group when assessing functional outcome.

Clinical Pearl

Insufficiency fractures of the distal radius are very common and are a feature of bone type

Insufficiency fractures of the distal radius are unstable and likely to re-displace after closed manipulation

Percutaneous K wires are unlikely to maintain radial length

Extreme deformity is necessary before poor outcome is certain

Implant Evolution

Modern internal fixation implants have developed unrecognizably since the first scaled-down long bone plates and screws were used to stabilize wrist fractures before the mid 1990s. External fixators, K-wires and non-locking T-plates were the main implants employed before the advent of angular stability. The introduction of fixed angle locking plates revolutionized the management of many fractures, since it became possible to support the subchondral bone without engaging it with screws. The often fragmented bone (in the young) or fragile bone (in the elderly) in the distal radial metaphysis meant that standard fixation was simply not possible and the area was 'bridged' by an external fixator or multiple K-wires.

The first 'locking' plates employed smooth pins in the subchondral region and bridged the comminuted or softened metaphysis with a load bearing segment of the plate. The plate was secured to the bone in the diaphysis with non-locking screws, with the stability of the construct dependent on the screw-bone interface.

Further developments produced locking options on both sides of the fracture – a development which allowed even the most osteoporotic fracture to be managed by internal fixation if desired.

Many different implant producers began to develop their own shapes and surface finishes in what had become a lucrative marketplace. However, the principles remained – fixed-angle stability with a selection of pins or screws to support the subchondral bone.

Most of these fractures involve dorsal comminution with effective bone loss. It was logical to stabilize these fractures with locking plates applied on the dorsal surface after open, direct reduction of the displaced fragments and bridging of the bone defect with the implant.

Emphasis then moved to inserting the implants on the palmar surface of the radius, driven by the complications of tendon rupture caused by the adjacency of implant to the extensor tendons. This drove the development of plates that were pre-shaped to match the volar surface of the distal radius, so-called 'anatomic' plates. Anatomic palmar locking plates could now be used to aid reduction of dorsally displaced distal radius fractures. The subchondral pins, or screws, would be inserted just beneath the joint surface and the longitudinal limb of the plate 'pushed down' onto the palmar surface of the radius, correcting the anatomy as the plate neared the bone surface. This 'indirect reduction' maneuver has now become the procedure of choice for many fracture patterns.

Yet issues relating to extensor tendon contact still existed with the tips of the screws. The curved dorsal surface of the distal radius meant that screw tips often over-penetrated the cortex and entered the extensor compartments (II, III and IV), irritating the tendons and risking attritional rupture (see Fig. 13.15). Fixed-angle locking plate screws do not need to engage both cortices to function. Indeed the 'near cortex', in which a locking screw or pin engages, is actually the threaded locking hole of the plate. The screw/pin is then stable to deforming forces and further contact with cortical bone adds nothing to this stability. Locking screws and pins can (and should) be left short of the far cortex. In this way, a smooth locking pin functions identically to a locking screw. The thread of the screw confers no advantages and is functionally redundant.

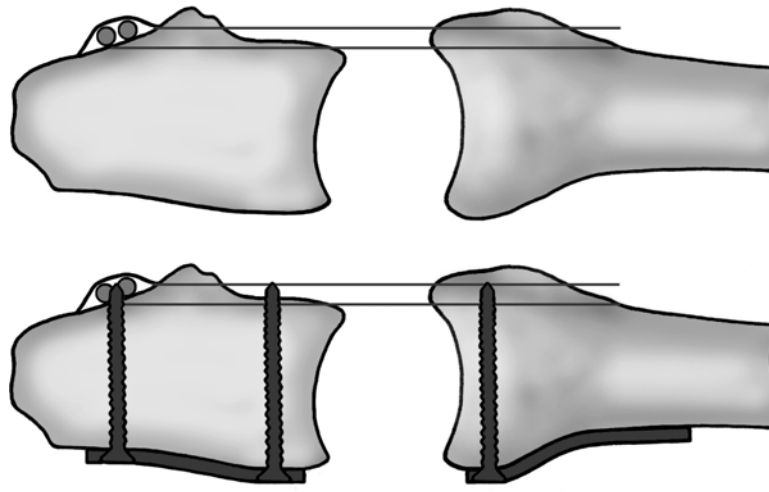


Fig. 13.15 Overpenetration of screw tips can remain unrecognized on lateral x-rays

The shape of the palmar surface of the distal radius is flat, so the application of a flat metal plate to a flat surface of bone automatically corrects any rotational deformities. The surface is also curved in an AP direction, which is why anatomic implants were required, but the distal edge of this surface can easily be broached by a palmar implant, giving rise to a metal 'edge' which can irritate and/or rupture the deep flexor tendons. Careful placement of such implants before this 'watershed' line will avoid this problem.

Implants are most useful when a single design will be able to stabilize a variety of fracture patterns. Certain fracture patterns, however, do demand a specific plate design. The key to reduction and stabilization is the intermediate column, so palmar locking plates must provide an option for a minimum of 2 locking screws to engage in a fractured palmar lunate facet, preferably in the subchondral bone. Volar rim avulsion fractures demand a very distal placement of the implant, so this area of the plate should extend as far distally as is safe. Radial styloid fractures can be stabilized with screw trajectories from a more proximal starting point, but angled to engage in the tip of the styloid. This avoids the need to place the plate on the curved, most radial area of bone that lies directly underneath the radial artery. Small holes in the implant to allow temporary placement with K-wires will help the process of insertion, but are unnecessary for stability or final fixation.

Finally, it must be remembered that not all distal radius fractures can be stabilized by an implant applied on the palmar surface. Certain fracture patterns, such as the displaced dorsoulnar fragment, will require a dorsally placed implant and, on certain unusual occasions, implants will be necessary on both dorsal and palmar surfaces to stabilize highly comminuted fractures. In these circumstances, the palmar plate should be applied first to recreate the anatomic palmar 'cortex', before the dorsal implants are used to buttress against this palmar implant and secure articular reduction and stability.

The surfaces of implants should be highly polished to reduce their micro-roughness and make them less likely to soft tissue and bone ingrowth. Their edges should be rounded and smooth.

Absorbable implants cannot yet provide the stability, strength and acceptable surface to be recommended. The technology of these may improve with development, but their absorption characteristics must become more predictable and less inflammatory before they are accepted for wide indications.

Clinical Pearl

Angular stability is available in almost all modern distal radius implants

The mechanical strength of modern implants makes primary bone grafting of distal radius fractures almost obsolete

Dorsally placed implants remain useful in certain fracture patterns

Summary

Distal radial fractures account for a significant workload in the daily practice of all trauma surgeons. The global shift towards internal fixation means a greater number of invasive, technically demanding procedures are being performed. This change is supported by very little hard evidence at present, but the majority of studies report a quicker return to function after internal fixation.

Two different demographic groups exist – the low energy, metaphyseal 'bending' fracture, frequently seen in the elderly and the high energy, fragmented and often articular fracture type seen in younger individuals. These are different injuries and demand a specific approach. The 3-column concept is valuable and should be used to assist in injury pattern recognition, reconstruction planning and to focus attention on the DRUJ. In addition, the presence of carpal malalignment should be identified and corrected to restore normal mechanical loading. In this way, these fractures are managed as 'wrist injuries' and not simply 'distal radius fractures'.

Recognition of the injury pattern is key to planning and executing the appropriate fixation technique. Soft tissue injuries are often missed. Ultimately, successful outcome is dependent upon identification and management of all injured bone and soft tissue components.

Adequate imaging is critical in understanding the injury pattern and the volar and dorsal rims of the distal radius should be well visualised. The volar rim of the lunate facet is a key area in fracture stability, due to the origin of the palmar extrinsic ligaments. CT scanning should be used judiciously only in those cases where doubt exists after assessment of plain radiographs.

Most articular fractures are unstable and likely to require stabilisation in some form. Comminuted metaphyseal fractures lack a stable support for the articular fragments and will likely require bridging fixation. Extra-articular fracture management is less clear and consideration should be given to the extent of shortening, DRUJ disruption, tilt and translation, as well as to the specific characteristics of each patient. Treatment choice should be made on an individual basis. No clear guidelines can, or should, be given.

When internal fixation is chosen, the 'correct' implant should be selected for its mechanical characteristics, rather than manufacturer and those features should be employed in a manner adhering to the principles of the implant's design. Familiarity with the chosen device is obligatory, to ensure adequate placement and appropriate use of fixed locking, variable angle locking and non-locking technology. Imaging during surgery is important to assess screw placement and prevent tendon injuries.

Radiological parameters are not as predictive of functional outcomes as was once thought. Each patient's individual needs and goals should be assessed, before a management plan is conceived and treatment is undertaken.

References

1. Frykman G. Fractures of the distal radius, including sequelae – shoulder and finger syndrome, disturbance in the distal radioulnar joint and impairment of nerve function: a clinical and experimental study. Acta Orthop Scand Suppl. 1967;108:1–155.
2. Rikli DA, Regazzoni P. Fractures of the distal end of the radius treated by internal fixation and early function. A preliminary report of 20 cases. J Bone Joint Surg (Br). 1996;78B:588–92.
3. Pechlaner S, Kathrein A, Gabl M, Lutz M, Angermann P, Zimmermann R, Peer R, Peer S, Rieger M, Freund M, Rudisch A. Distal radius fractures and concomitant injuries: experimental studies concerning pathomechanisms. J Hand Surg (Br). 2002;28B:609–16.
4. Tanabe K, Nakajima T, Sogo E, Denno K, Horiki M, Nakagawa R. Intra-articular fractures of the distal radius evaluated by computed tomography. J Hand Surg (Am). 2011;36A:1798–803.
5. Melone CJ. Distal radius fractures: patterns of articular fragmentation. Orthop Clin North Am. 1993; 24:239–53.
6. Knirk JL, Jupiter JB. Intra-articular fractures of the distal end of the radius in young adults. J Bone Joint Surg (Am). 1986;68A:657–9.
7. Haus BM, Jupiter JB. Intra-articular fractures of the distal end of the radius in young adults: re-examined as evidence based and outcomes medicine. J Bone Joint Surg (Am). 2009;91A:2984–91.
8. Mandziak DG, Watts AC, Bain GI. Ligament contribution to patterns of articular fractures of the distal radius. J Hand Surg (Am). 2011;36A:1621–5.
9. Lochmüller EM, Matsuura M, Bauer J, Hitzl W, Link TM, Müller R, Eckstein F. Site-specific deterioration of trabecular bone architecture in men and women with advancing age. J Bone Miner Res. 2008;23:1964–73.

10. Stoffelen D, De Smet L, Broos P. The importance of the distal radioulnar joint in distal radial fractures. J Hand Surg (Br). 1998;23B:507–11.
11. Müller ME, Nazarian S, Koch P, Schatzker J. The comprehensive classification of fractures of long bones. Berlin: Springer; 1990.
12. Altissimi A, Azzara A, Mancini GB, Pierdominici P. The reliability of classification of articular fractures of the distal radius. J Hand Surg (Br). 1996;21B Suppl 1:31.
13. Jupiter JB, Fernandez DL. Comparative classification for fractures of the distal end of the radius. J Hand Surg (Am). 1997;22A:563–71.
14. Naqvi SGA, Reynolds T, Kitsis C. Interobserver reliability and intraobserver reproducibility of the Fernandez classification for distal radius fractures. J Hand Surg (Eur). 2009;34E:483–5.
15. Lafontaine M, Hardy D, Delince P. Stability assessment of distal radius fractures. Injury. 1989;20:208–10.
16. Altissimi M, Mancini GB, Azzara A, Ciaffoloni E. Early and late displacement of fractures of the distal radius: the prediction of instability. Int Orthop. 1994;18:61–5.
17. Barton T, Chambers C, Bannister G. A comparison between subjective outcome score and moderate radial shortening following a fractured distal radius in patients of mean age 69 years. J Hand Surg (Eur). 2007;32E:165–9.
18. Abbaszadegan H, Jonsson U, von Sivers K. Prediction of instability of Colles' fractures. Acta Orthop Scand. 1989;60:646–50.
19. Young CE, Nanu AM, Checketts RG. Seven year outcome following Colles' type distal radial fracture: a comparison of two treatment methods. J Hand Surg (Br). 2003;28B:422–6.
20. Batra S, Gupta A. The effect of fracture-related factors on the functional outcome at 1 year in distal radius fractures. Injury. 2002;33:499–502.
21. Gliatis JD, Plessas SJ, Davis TRC. Outcome of distal radial fractures in young adults. J Hand Surg (Br). 2000;25B:535–43.
22. McQueen MM, Hajducka C, Court-Brown CM. Redisplaced unstable fractures of the distal radius: a prospective randomized comparison of four methods of treatment. J Bone Joint Surg (Br). 1996;78B:404–9.
23. Ng CY, McQueen MM. What are the radiological predictors of functional outcome following fractures of the distal radius? J Bone Joint Surg (Br). 2011;93B:145–50.
24. Gartland J, Werley C. Evaluation of healed Colles' fractures. J Bone Joint Surg (Am). 1951;33:895–907.
25. Kwok IHY, Leung F, Yuen G. Assessing results after distal radius fracture treatment: a comparison of objective and subjective tools. Geriatr Orthop Surg Rehabil. 2011;2:155–60.
26. Beaule PE, Dervin GF, Giachino AA, Rody K, Grabowski J, Fazekas A. Self reported disability following distal radius fractures: the influence of hand dominance. J Hand Surg (Am). 2000;25A:476–82.
27. Fujii K, Henmi T, Kanematsu Y, Mishiro T, Sakai T, Terai T. Fractures of the distal end of radius in elderly patients: a comparative study of anatomical and functional results. J Orthop Surg (Hong Kong). 2002;10:9–15.
28. Karnezis IA, Fragkiadakis EG. Association between objective clinical variables and patient-rated disability of the wrist. J Bone Joint Surg (Br). 2002;84B:967–70.
29. Grewal R, MacDermid JC. The risk of adverse outcomes in extra-articular distal radius fractures is increased with malalignment in patients of all ages but mitigated in older patients. J Hand Surg (Am). 2007;32A:962–70.
30. Robertson GA, Robertson BF, Thomas B, McEachan J, Davidson DM. Assessing angulation on digital images of radiographs of fractures of the distal radius: visual estimation versus computer software measurement. J Hand Surg (Eur). 2011;36E:230–5.
31. Yang Z, Mann FA, Gilula LA, Haerr C, Larsen CF. Scaphopisocapitate alignment: criterion to establish a neutral lateral view of the wrist. Radiology. 1997;205:865–9.
32. Capo JT, Accousti K, Jacob G, Tan V. The effect of rotational malalignment on x-rays of the wrist. J Hand Surg (Eur). 2009;34E:166–72.
33. Kaempfe FA, Walker KM. External fixation for distal radius fractures: effect of distraction on outcome. Clin Orthop. 2000;1(380):220–5.
34. Lundy DW, Quisling SG, Lourie GM, Feiner CM, Lins RE. Tilted lateral radiographs in the evaluation of intra-articular distal radius fractures. J Hand Surg (Am). 1999;24A:249–56.
35. Pruitt DL, Gilula LA, Manske PR, Vannier MW. Computed tomography scanning with image reconstruction in evaluation of distal radius fractures. J Hand Surg (Am). 1994;19A:720–7.
36. Cole RJ, Bindra RR, Evanoff BA, Gilula LA, Yamaguchi K, Gelberman RH. Radiographic evaluation of osseous displacement following intra-articular fractures of the distal radius: reliability of plain radiography versus computed tomography. J Hand Surg (Am). 1997;22A:792–800.
37. Adams B. Effects of radial deformity on distal radioulnar joint mechanics. J Hand Surg (Am). 1993;18A:492–8.
38. Short WH, Palmer AK, Werner FW, Murphy DJ. A biomechanical study of distal radial fractures. J Hand Surg (Am). 1987;12A:529–43.
39. Kihara H, Palmer AK, Werner FW, Short WH, Fortino MD. The effect of dorsally angulated distal radius fractures on distal radioulnar joint congruency and forearm rotation. J Hand Surg (Am). 1996;21A:40–7.
40. Bong MR, Egol KA, Leibman M, Koval KJ. A comparison of immediate postreduction splinting constructs for controlling initial displacement of fractures of the distal radius. J Hand Surg (Am). 2006;31A:766–70.
41. Stewart HD, Innes AR, Burke FD. Functional cast-bracing for Colles fractures. J Bone Joint Surg (Br). 1984;66B:749–53.

42. Wahlstrom O. Treatment of Colles fractures. Acta Orthop Scand. 1982;53:225–8.
43. Koenig KM, Davis GC, Grove MR, Tosteson ANA, Koval KJ. Is early internal fixation preferred to cast treatment for well-reduced unstable distal radial fractures? J Bone Joint Surg (Am). 2009;91:2086–93.
44. Hull P, Baraza N, Gohil M, Whalley H, Mauffrey C, Brewster M, Costa ML. Volar locking plates versus K-wire fixation of dorsally displaced distal radius fractures – a functional outcome study. J Trauma. 2011;70:E125–8.
45. Kreder HJ, Agel J, McKee MD, Schemitsch EH, Stephen D, Hanel DP. A randomized, controlled trial of distal radius fractures with metaphyseal displacement but without joint incongruity: closed reduction and casting versus closed reduction, spanning external fixation, and optional percutaneous K-wires. J Orthop Trauma. 2006;20:115–21.
46. Kristiansen TK, Ryaby JP, McCabe J, Frey JJ, Roe LR. Accelerated healing of distal radial fractures with the use of specific, low-intensity ultrasound. A multicenter, prospective, randomized, double-blind, placebo-controlled study. J Bone Joint Surg (Am). 1997;79A:961–73.
47. Trumble TE, Schmitt SR, Vedder NB. Factors affecting functional outcome of displaced intra-articular distal radius fractures. J Hand Surg (Am). 1994;19:325–40.
48. Altissimi M, Mancini GB, Ciaffoloni E, Pucci G. Comminuted articular fractures of the distal radius: results of conservative treatment. Ital J Orthop Traumatol. 1991;17:117–23.
49. Catalano LW, Cole RJ, Gelberman RH, Evanoff BA, Gilula LA, Borrelli J. Displaced intra-articular fractures of the distal aspect of the radius. Long-term results in young adults after open reduction and internal fixation. J Bone Joint Surg (Am). 1997;79A:1290–302.
50. Fernandez DL, Geissler WB. Treatment of displaced articular fractures of the radius. J Hand Surg (Am). 1991;16A:375–84.
51. Jupiter JB, Marent-Huber M. Operative management of distal radial fractures with 2.4 millimeter locking plates: a multi-center prospective case series. J Bone Joint Surg (Am). 2009;91A:55–65.
52. Lindau T, Arner M, Hagberg L. Intra articular lesions in distal fractures of the radius in young adults. J Hand Surg (Br). 1997;22B:638–43.
53. Mehta JA, Slavotinek JP, Krishnan J. Local osteopenia associated with management of intra-articular distal radial fractures by insertion of external fixation pins in the distal fragment: prospective study. J Orthop Surg (Hong Kong). 2002;10:179–84.
54. Salter RB, Simmonds DF, Malcolm BW, Rumble EJ, MacMichael D, Clements ND. The biological effect of continuous passive motion on the healing of full-thickness defects in articular cartilage: an experimental investigation in the rabbit. J Bone Joint Surg (Am). 1980;62A:1232–51.
55. Jakob M, Rikli DA, Regazzoni P. Fractures of the distal radius treated by internal fixation and early function. A prospective study of 73 consecutive patients. J Bone Joint Surg (Br). 2000;82B:340–4.
56. Leung F, Tu Y, Chew WYC, Chow SP. Comparison of external and percutaneous pin fixation with plate fixation for intra-articular distal radius fractures: a randomized study. J Bone Joint Surg (Am). 2008;90A:16–22.
57. Kreder HJ, Hanel DP, Agel J, McKee M, Schemitsch EH, Trumble TE, Stephen D. Indirect reduction and percutaneous fixation versus open reduction and internal fixation for displaced intra-articular fractures of the distal radius: a randomized, controlled trial. J Bone Joint Surg (Br). 2005;87B:829–36.
58. Grewal R, Perey B, Wilmink M, Stothers K. A randomized prospective study on the treatment of intra-articular distal radius fractures: open reduction and internal fixation with dorsal plating versus mini open reduction, percutaneous fixation, and external fixation. J Hand Surg (Am). 2005;30A:764–72.
59. Chung KC, Watt AJ, Kotsis SV, Marqaliot Z, Haase SC, Kim HM. Treatment of unstable distal radial fractures with the volar locking plating system. J Bone Joint Surg (Am). 2006;88A:2687–94.
60. Arora R, Lutz M, Hennerbichler A, Krappinger D, Espen D, Gabl M. Complications following internal fixation of unstable distal radius fracture with a palmar locking plate. J Orthop Trauma. 2007;21:316–22.
61. Campbell DA. Open reduction and internal fixation of intra-articular and unstable fractures of the distal radius using the AO distal radius plate. J Hand Surg (Br). 2000;25B:528–34.
62. Orbay JL, Fernandez DL. Volar fixation for dorsally displaced fractures of the distal radius. J Hand Surg (Am). 2002;27A:205–15.
63. Tada K, Ikeda K, Shigemoto K, Suganuma S, Tsuchiya H. Prevention of flexor pollicis longus tendon rupture after volar plate fixation of distal radius fractures. Hand Surg. 2011;16:271–5.
64. Sügün TS, Karabay N, Gürbüz K, Özaksar T, Toros T, Kayalar M. Screw prominences related to plamar locking plating of distal radius. J Hand Surg (Eur). 2011;36:320–4.
65. Pichler W, Windisch G, Schaffler R, Rienmüller R, Grechenig W. Computer aided 3D analysis of the distal dorsal radius surface and the effects on volar plate osteosynthesis. J Hand Surg (Eur). 2009;34E:598–602.
66. Drobetz H, Bryant AL, Pokorny T, Spitaler R, Leixnering M, Jupiter JB. Volar fixed-angle plating of distal radius extension fractures: influence of plate position on secondary loss of reduction – a biomechanic study in a cadaveric model. J Hand Surg (Am). 2006;31A:615.e1–e9.
67. Harness NG, Jupiter JB, Orbay JL, Raskin KB, Fernandez DL. Loss of fixation of the volar lunate facet fragment in fractures of the distal part of the radius. J Bone Joint Surg (Am). 2004;86A:1900–8.
68. Ruch DS, Ginn A, Yang CC, Smith BP, Rushing J, Hanel DP. Use of a distraction plate for distal radial fractures with metaphyseal and diaphyseal comminution. J Bone Joint Surg (Am). 2005;87A:945–54.

69. May MM, Lawton JN, Blazar PE. Ulnar styloid fractures associated with distal radial fractures: incidence and implications for distal radioulnar joint instability. J Hand Surg (Am). 2002;27A:965–71.
70. Solgaard S. Function after distal radius fracture. Acta Orthop Scand. 1988;59:39–42.
71. Fernandez DL. Fractures of the distal radius: operative treatment. AAOS Instruct Course Lect. 1993;42:73–88.
72. Biyani A, Simison AJM, Klenerman L. Fractures of the distal radius and ulna. J Hand Surg (Br). 1995;20B:357–64.
73. Noda K, Goto A, Murase T, Sugamoto K, Yoshikawa H, Moritomo H. Interosseous membrane of the forearm: an anatomical study of ligament attachment locations. J Hand Surg (Am). 2009;34A:415–42.
74. Ring D, McCarty LP, Campbell DA, Jupiter JB. Condylar blade plate fixation of unstable fractures of the distal ulna associated with fracture of the distal radius. J Hand Surg (Am). 2004;29A:103–9.
75. Dennison D. Open reduction and internal locked fixation of unstable distal ulna fractures with concomitant distal radius fracture. J Hand Surg (Am). 2007;32A:801–5.
76. McKee MD, Waddell JP, Yoo D, Richards RR. Non union of distal radial fractures associated with distal ulnar shaft fractures: a report of four cases. J Orthop Trauma. 1997;11:49–53.
77. Hauck RM, Hershey PA, Shahen J, Palmer AK. Classification and treatment of ulnar styloid non union. J Hand Surg (Am). 1996;21A:418–22.
78. Richards RS, Bennett JD, Roth JH, Milne K. Arthroscopic diagnosis of intra-articular soft tissue injuries associated with distal radial fractures. J Hand Surg (Am). 1997;22A:772–6.
79. Scheer JH, Adolfsson LE. Radioulnar laxity and clinical outcome do not correlate after a distal radius fracture. J Hand Surg (Eur). 2011;36E:503–8.
80. Jupiter JB. Commentary: the effect of ulnar styloid fractures on patient-rated outcomes after volar locking plating of distal radius fractures. J Hand Surg (Am). 2009;34A:1603–4.
81. Forward DP, Lindau TR, Melsom DS. Intercarpal ligament injuries associated with fractures of the distal part of the radius. J Bone Joint Surg (Am). 2007;89A:2334–40.
82. Varitimidis SE, Basdekis GK, Dailliana ZH, Hantes ME, Bargiotas K, Malizos K. Treatment of intra-articular fractures of the distal radius: fluoroscopic or arthroscopic reduction? J Bone Joint Surg (Br). 2008;90B:778–85.
83. Campbell DA. Letter to the editor. J Hand Surg (Eur). 2007;32E:233–4.
84. Singer BR, McLauchlan GJ, Robinson CM, Christie J. Epidemiology of fractures in 15,000 adults: the influence of age and gender. J Bone Joint Surg (Br). 1998;80B:243–8.
85. Nesbitt KS, Failla JM, Les C. Assessment of instability factors in adult distal radius fractures. J Hand Surg (Am). 2004;29A:1128–38.
86. Beumer A, McQueen MM. Fractures of the distal radius in low-demand elderly patients: closed reduction of no value in 53 of 60 wrists. Acta Orthop Scand. 2003;74:98–100.
87. Kennedy C, Kennedy MT, Niall D, Devitt A. Radiological outcomes of distal radius extra-articular fragility fractures treated with extra-focal kirschner wires. Injury. 2010;41:639–42.
88. Jupiter JB, Ring D, Weitzel PP. Surgical treatment of redisplaced fractures of the distal radius in patients older than 60 years. J Hand Surg (Am). 2002;27A:714–23.
89. Orbay JL, Fernandez DL. Volar fixed-angle plate fixation for unstable distal radial fractures in the elderly patient. J Hand Surg (Am). 2004;29A:96–102.
90. Arora R, Lutz M, Fritz D, Zimmermann R, Oberladstätter J, Gabl M. Palmar locking plate for treatment of unstable dorsal dislocated distal radius fractures. Arch Orthop Trauma Surg. 2005;125(6):399–404.
91. Chung KC, Squitieri L, Kim HM. Comparative outcomes study using the volar locking plating system for distal radius fractures in both young adults and adults older than 60 years. J Hand Surg (Am). 2008;33A:809–19.
92. Walz M, Kolbow B, Auerbach F. Do fixed angle T-plates offer advantages for distal radius fractures in elderly patients? Unfallchirurg. 2004;107:664–70.
93. Roumen RM, Hesp WL, Bruggink ED. Unstable Colles fractures in elderly patients. A randomized trial of external fixation for redisplacement. J Bone Joint Surg (Br). 1991;73B:307–11.
94. Kelly AJ, Warwick D, Crichlow TP, Bannister GC. Is manipulation of moderately displaced Colles' fracture worthwhile? A prospective randomized trial. Injury. 1997;28:283–7.
95. Young BT, Rayan GM. Outcome following nonoperative treatment of displaced distal radius fractures in low-demand patients older than 60 years. J Hand Surg (Am). 2000;25A:19–28.
96. Anzarut A, Johnson JA, Rowe BH, Lambert RG, Blitz S, Majumdar SR. Radiologic and patient-reported functional outcomes in an elderly cohort with conservatively treated distal radius fractures. J Hand Surg (Am). 2004;29A:1121–7.
97. Azzopardi T, Ehrendorfer S, Coulton T, Abela M. Unstable extra-articular fractures of the distal radius: a prospective, randomized study of immobilization in a cast versus supplementary percutaneous pinning. J Bone Joint Surg (Br). 2005; 87B:837–40.
98. Hegeman JH, Oskam J, Vierhout PA, Ten Duis HJ. External fixation for unstable intra-articular distal radial fractures in women older than 55 years. Acceptable functional end results in the majority of the patients despite significant secondary displacement. Injury. 2005;36:339–44.
99. Jaremko JL, Lambert RG, Rowe BH, Johnson JA, Majumdar SR. Do radiographic indices of distal radius fracture reduction predict outcomes in older adults receiving conservative treatment? Clin Radiol. 2007;62:65–72.
100. McQueen MM, Caspers J. Colles fracture: does the anatomical result affect the final function? J Bone Joint Surg (Br). 1988;70B:649–51.

Hand Injuries in Children

14

David McCombe

Keywords
Hand injury • Children • Fingertip • Nail bed injury • Hand fracture • Wrist fracture • Joint injury • Flexor tendon • Extensor tendon • Nerve injury • Vascular injury • Replantation • Growth plate fracture

Introduction

Hand trauma in children is common, but often inadequately assessed and treated. The types of injury vary with age depending on the pattern of hand use. Assessment of the injury, particularly in the younger child, can be difficult, given the inability or reluctance of the child to co-operate, both in providing a history and allowing an examination of the injured hand. The established principles of treatment for hand trauma, detailed elsewhere in this text, apply equally to children, but the healing and growth potential of the child has specific implications for management. The management plan also has to take into account the varying capacity of the child for co-operation with active rehabilitation programmes.

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

St. Vincent's Hospital,
41 Victoria Parade, Fitzroy, Melbourne,
VIC 3065, Australia
e-mail: david.mccombe@vhsa.com.au

Epidemiology/Aetiology

The annual incidence of hand injuries has been estimated at between 20 and 45/10,000 children [1, 2]. The aetiology and, consequently, the nature of hand injuries in children vary according to the age and behaviour of the individual child.

The toddler is naïve to the hazards of their environment and the commonest injury in this age group is the crush of the distal fingertip, usually the result of a finger caught in the hinge side of a closing door or a drawer. Burns to the palmer surface of the digit or palm and injuries from various domestic appliances in the home are also common, as the hand is used to explore the toddler's surroundings.

In the older child, from 6 years to adolescence, fractures are the most common injury, as the child becomes involved in ball and contact sports. The proximal phalanges of the border digits are the most commonly fractured bones in most series [3, 4], with a peak incidence around the age of 12. Fracture patterns in children differ to fractures in the mature skeleton, due to biology and behaviour.

Mutilating injuries to the hand, with significant tissue loss or damage along with major amputations

I.A. Trail, A.N.M. Fleming (eds.), *Disorders of the Hand: Volume 1: Hand Injuries*,
DOI 10.1007/978-1-4471-6554-5_14, © Springer-Verlag London 2015

are uncommon in children, but present specific challenges considering the effects of injury and ischaemia upon the growth potential of the hand.

Assessment

The assessment of the injured hand in children can be a challenge, particularly in the acute setting. Most injuries are isolated and relatively minor, however, in the setting of a major or combined injury, it is important to assess the child as a whole, as issues such as blood loss in a small child can lead to significant hypovolemia relative to a similar injury in the adult.

Depending on the age of the child, the history may be difficult to obtain and often the injury may not have been witnessed. Second hand histories from siblings or parents can be valuable however and, despite the difficulty with age, it is worth attempting to engage with the child, to help build rapport for subsequent examination and treatment.

Examination of the injured hand in the younger child in the acute setting can also be difficult, as the child is usually in pain and often terrified at the prospect of the dressing being removed or the hand being touched by yet another stranger. It is important to observe, as much as possible, with the dressing intact, including the circulation of the exposed fingertips and movement, posture and alignment of digits before removing dressings. It may not be necessary to remove all adherent dressings to evaluate the injury, if the nature of the injury is obvious and it is clear that the injury will require further management under anesthetic in theatre. Examination requiring co-operation of the child is often difficult, particularly the assessment of sensation and motor function. It is important to observe for posture and spontaneous active movement and use the presence of sweat on the fingertips, as detected by resistance to running a plastic pen across the pulp of the affected finger to indicate intact innervation (Fig. 14.1). In the delayed presentation of nerve injury, trophic changes and loss of the glabrous skin ridges, result in a dry shiny fingertip pulp indicate a. Examination of flexor tendon function can also be difficult and again, posture is important. Other markers include, 'the tenodesis effect' of movement

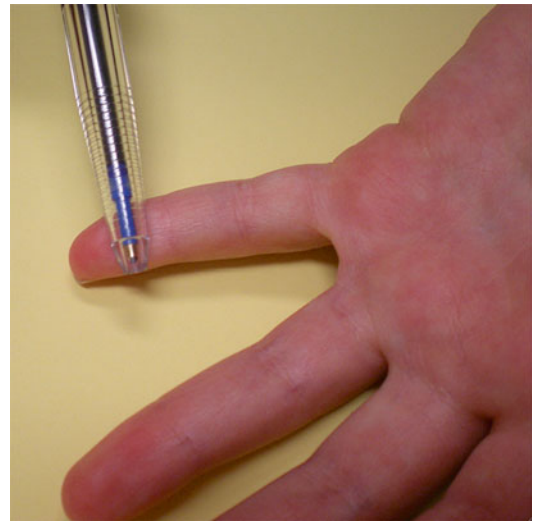


Fig. 14.1 Injury to the proximal nerve leads to loss of sensation and sweating of the pulp skin. The latter can be detected by the lack of resistance to dragging a plastic pen across the pulp in comparison to the normally innervated digit

of a proximal joint, and secondly, whether squeezing the forearm compartment results in flexion of the interphalangeal joints (Fig. 14.2).

Imaging forms an important part of examination in all hand injuries. In the child, the presence of cartilaginous physes and ossification centres complicates the imaging of skeletal injuries and requires knowledge of the anatomy of the developing skeleton. Ultrasound has a role in the assessment of a closed injury, particularly of dynamic structures, such as flexor tendons or vascular structures and for non-metallic foreign bodies. It has an advantage in younger children, compared to other modalities such as CT or MRI, as anaesthesia or sedation is not required to obtain adequate imaging.

Management

The principles, management and surgical technique do not vary widely from adult practice. There are, however, particular injuries that are more common in children and the biology of the child, with rapid healing and the effect of ongoing growth, needs to be considered in treating injury. The capacity for rapid healing is favourable for shortening immobilization periods post injury, but restricts the period in

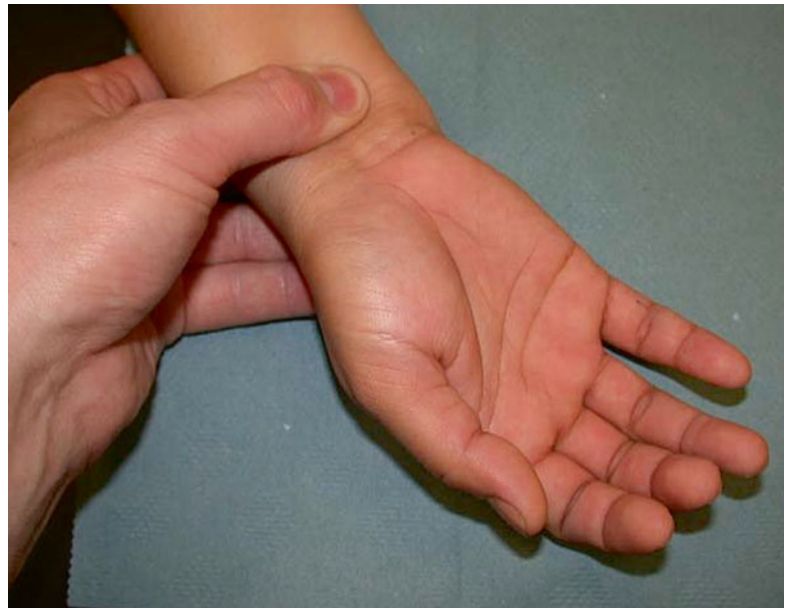


Fig. 14.2 Pressure applied to the flexor tendons in the distal forearm elicits interphalangeal flexion if the flexor tendon is intact

which closed manipulation of fractures is likely to be effective. The growing skeleton has the potential to remodel skeletal deformity, depending on the plane of the fracture's displacement. On the other hand, if growth plates are damaged by injury or treatment, progressive deformity can occur because of tethering of growth. Another factor that is important to consider in treatment plans is the often limited capacity of the child to co-operate with postoperative care. The dressing and splint protection of the injured hand or limb needs to take into account the age of the child, the capacity for co-operation, the need to protect skeletal and/or tendon repairs and the need to be able to observe the part for changes in circulation, particularly in the setting of revascularization or replantation. For most children under the age of 7, above elbow splinting is required to prevent the removal of the splint by the child either in a sugar tong type splint or cylindrical cast (Fig. 14.3). The rehabilitation programme for a child with a hand injury is age and maturity dependent and it is usually safer to immobilize the younger child for at least 4 weeks, rather than rely upon their co-operation. After this, depending on the nature of the injury and the child, ongoing protective splinting and directed play to encourage specific motion is usually used.

Fig. 14.3 Well padded above elbow cast applied in theatre after repair of a hand injury in a 3 year old child. The limb is then elevated in a stockingette sling usually worn under the outer clothes to protect the limb

The psychological impact of the injury upon the child and parents also needs to be considered as part of the treatment programme. This is particularly the case with significant or mutilating injuries, where the physical impact of the injury is compounded by postoperative pain, hospitalization and ongoing outpatient care.

Fingertip and Nail Bed Injury

The fingertip is the anatomically specialized component of the digit, responsible for the transmission of force in pinch, grasp and palpation, (an increasingly important function in the world of keyboards and touch screens), the transduction of sensation and is an important aesthetic component of the digit. The pulp is a richly innervated pad, stabilized over the skeleton by a complex fascial network, augmented by the rigid nail plate on the dorsum. Given its role in palpation and grasp and its position at the extremity of the digit, the fingertip is vulnerable to injury, particularly in the adventurous toddler. The most common mechanism of injury is a crush, which combines injury to the nail bed and pulp and in 50 % of cases there is a fracture of the distal phalangeal tuft. Separation of the injury into these components has been described by Evans and Bernadis in their PNB classification of fingertip injury [5].

Nail Bed Injury

A subungual haematoma, without avulsion or subluxation of the nail plate can be managed conservatively or with trephine alone. Results from exploration and repair of the nail bed are no better than with trephination alone [6]. Where the nail plate is damaged or subluxated from the eponychial fold, it will no longer splint the nail bed and will allow an irregular scar to form as the associated nail bed laceration heals and produces a nail deformity. In this circumstance, removal of the nail and accurate nail bed repair, using magnification and absorbable suture, is indicated (Fig. 14.4). An effective alternative to suturing is

Fig. 14.4 Laceration of the sterile matrix of the nail bed (**a**), requires removal of the nail and accurate repair of the matrix (**b**). The result at 3 months is shown in (**c**)

Fig. 14.5 Deformity of the nail of the ring finger after injury to the germinal matrix and scarring of nail fold

cyanoacrylate glue [7]. If the nail itself is lost or badly damaged, a silicone or artificial sheet is inserted under the nail fold to prevent adhesion and to maintain the contour of the nail matrix. The splint or nail should be fenestrated for drainage and fixed using glue or absorbable suture to prevent displacement. Nail regrowth begins a few weeks after injury and occurs over at least 4 months [8]. Secondary nail deformities occur due to scarring of the dorsal nail fold, resulting in dull streaking of the nail; scarring of the germinal matrix, resulting in splitting, ridging or absence of the nail (Fig. 14.5); scarring of the sterile matrix, resulting in loss of nail adhesion to the matrix (onycholysis) or adhesion of the nail fold, which produces a retention of the nail in the eponychial fold. These deformities can be addressed, though a normal nail is difficult to restore where there is significant scarring of the specialized perionychial tissues. The other common secondary deformity of the nail complex is the parrot-beak deformity, seen with loss of the tuft of the phalanx and consequently loss of nail bed support.

Distal Phalanx Injury

Most distal phalangeal fractures in the setting of a crush injury are comminuted fractures of the tuft and require splinting only for 3 or 4 weeks until the tip is stable. Unstable fractures of the shaft of the phalanx, associated with a fingertip injury, can be stabilized with a fine axial Kirschner wire. Basal fractures of the distal phalanx will be discussed below.

Fingertip Pulp Injury

Lacerations of the pulp, due to crush or avulsion, should be closed without tension or left open, as the pulp is likely to swell following injury and excessive suture tension can lead to tissue necrosis.

If tissue loss has occurred, in most circumstances, dressing alone allows for healing by secondary intention over a couple of weeks, and for most, an excellent result. If the tissue loss is significant however, the management depends on whether the amputated part is available and can be replanted or repositioned as a composite graft, or whether tissue will need to be imported to obtain wound closure.

Replantation of fingertip amputations provides the optimal reconstruction and is indicated where an artery can be identified in the distal part. The vascular anatomy of the fingertip has been defined and forms the basis of classifications of amputations [9, 10]. The distal digital arteries form an arcade at the level of the base of the nail and terminal arteries radiate distally from this with the largest usually in the midline. While technically challenging, it is usually possible to anastomose this vessel to an artery at the level of amputation. The access can be difficult in a small fingertip and, rather than fixing the skeleton initially, an open-book technique can be used, repairing the volar skin and then the vessel from the dorsum, before 'closing the book' of the fingertip by advancing a preplaced axial Kirschner wire across the osteosynthesis. The alternative is to suture a vein graft to the distal artery prior to skeletal fixation and proximal arterial anastomosis. Another difficulty in the distal replantation is obtaining adequate venous drainage. Survival of the replant is improved if venous outflow is achieved [11]. Replantations at the level or distal to the nail fold, do not allow for dorsal venous anastomosis. The alternative is to use a volar vein [11], or create an arteriovenous anastomosis [12] with a distal arterial branch anasatomosed to a proximal vein to relieve venous congestion.

Where no venous outflow is possible, the venous congestion that results from the arterial input can be relieved by controlled bleeding over the first 4–5 days, until adequate venous angiogenesis develops across the scar. In the adult, or perhaps the adolescent, this can be achieved with leeches, but this is difficult in children. Bleeding can be achieved with an incision or de-epithelialization of the pulp tip and either systemic or topical anticoagulation [13]. Survival of distal replants with controlled bleeding is better in children than with adults [14], but is limited to distal replants where the volume of tissue is smaller. In small children with small total blood volumes, the technique of controlled bleeding has the potential to lead to significant blood loss, requiring blood transfusion. This needs to be discussed with the parents before embarking on this process, as the benefit of fingertip survival has to be balanced against the risks of blood loss and transfusion, in addition to the trauma of regular bleeding of the fingertip.

Clinical Pearl

For distal replantation, anastomosing a vein graft to the distal artery before attachment of the amputated part allows better access for what is usually the more difficult anastomosis. After the amputated part is repositioned on the stump the vein graft can then be anastomosed to the proximal artery out of the zone of injury.

If replantation of the amputated part is not feasible, reposition of the part as a composite graft is the preferred option for distal amputations of the pulp and distal nail bed. The technique requires defatting of the graft and precise dermal apposition to optimize the revascularization of the graft. The survival of the graft can be improved by cooling, Prostaglandin E1 [15] or hyperbaric treatment.

For cases where there is a substantial defect of the pulp or fingertip, tissue can be imported by graft, composite graft of glabrous skin from the palm or toe [16], local homodigital or regional flaps such as the thenar flap, which is well suited to the child's hand. Debridement and shortening of the distal fingertip, with excision of the nail bed, may be applied in the proximal amputation, where efforts at preservation of the fingertip may only produce a beaked nail, due to inadequate phalangeal length, though all attempts should be made to preserve enough phalangeal length for the flexor tendon insertion.

Fractures

The growing skeleton of the hand is a combination of the tubular bones of the digital rays, marked by cartilaginous growth plates and the carpal bones, whose ossification occurs sequentially through the first 8 years of life. The tubular bones of the phalanges and metacarpals have thick strong periosteum, confluent with the adjacent joints' capsules, but the growth plate is an area of relative weakness, particularly the hypertrophic precalcific zone of the physis. The Salter-Harris classification describes the pattern of fractures seen in relationship to the physis (Fig. 14.6). The growth potential of the physis is preserved in the most common injuries (Type I and II), where the plane of the fracture is through the hypertrophic zone of the physis, leaving the proliferative zone intact. Type III and IV fractures involve the joint surface and may need reduction and/or fixation to maintain the joint congruity and stability. The fractures that cross the physis, (Type IV), can bring the metaphysis into contact with the epiphysis and a bony bar can form across the growth plate, retarding growth and producing deformity. A crush injury of the physis (Type V) can damage the proliferative zone cells and lead to premature growth plate closure and retardation of longitudinal growth [17]. The structural weakness of the growth plate can be further exposed by the anatomy of the ligamentous attachments at the metacarpophalangeal joint, where the ligaments insert on the epiphysis and hence the propensity for fractures of the base of the proximal phalanx at the growth plate with lateral force. At the interpha-

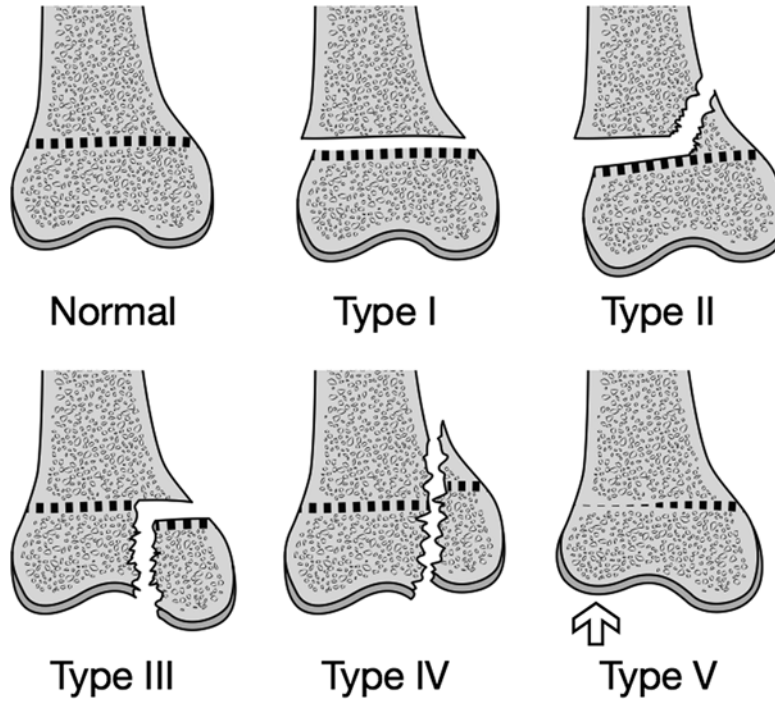


Fig. 14.6 The Salter Harris classification of growth plate injuries. The *Type I* injury is a fracture through the hypertrophic zone of the growth plate. *Type II* fractures follow the same plane but at a lateral margin the fracture involves the metaphysis and a portion of this remains attached to the growth plate. The *Type III* fractures involve the epiphysis and joint surface but don't cross the growth plate. *Type IV* fractures extend from the joint surface through the growth plate and with displacement, the metaphysis can come into contact with the epiphysis and fuse across the growth plate producing deformity. *Type V* injuries involve a crush injury to the growth plate with injury to the proliferative zone of the physis and premature closure of the growth plate

langeal joints, the collateral ligament insertion spans the physis providing support and lateral forces tend to produce fractures proximal to the joint of the head or neck of proximal or middle phalanges [18]. Given the relative strength of the joint capsule and ligaments, dislocations and significant ligamentous injuries in the child's hands are uncommon.

In general, displaced fractures are difficult to reduce after 7–10 days in children as they heal quickly. Malunion is thus common as presentation is often delayed, as the injury and deformity is attributed to soft tissue sprain only. Early osteotomy and reduction is reasonable for significantly displaced intra-articular and diaphyseal fractures, but is difficult to recommend for epiphyseal injuries given the potential for remodeling here, if the deformity is in a plane of the adjacent joint's motion and the risk of physeal injury by osteotomy in this position. Immobilization for 3–4 weeks is adequate for most injuries, but further protection may be required for the playground or schoolyard for another 2 weeks, while the fracture consolidates.

While the whole gamut of fracture patterns in the hand and forearm seen in the adult can occur in the child, there are particular types of fractures that are common and worth further discussion.

Phalangeal Fractures

Distal phalangeal fractures of the tuft and distal shaft have been discussed above. Fractures at the level of the growth plate of the distal phalanx are seen after an axial or crush injury and are usually angulated palmarward because the flexor tendon inserts distal to the growth plate. This pattern of

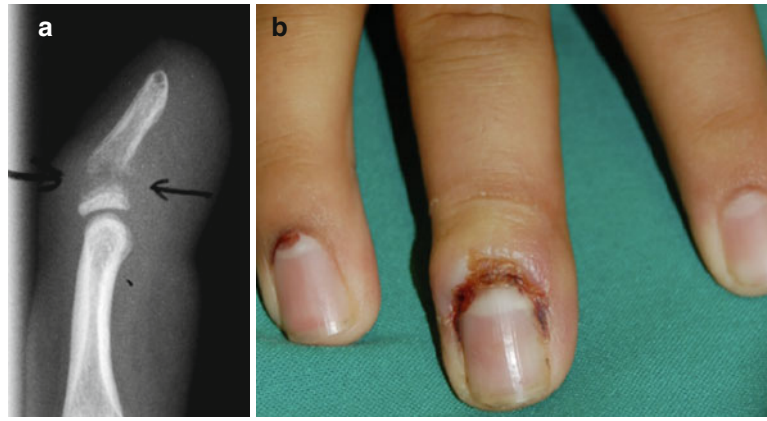


Fig. 14.7 (**a**) Delayed presentation after Seymour fracture, (Salter-Harris Type 1 Fracture of distal phalanx). The *arrows* indicate lysis of fracture margins consistent with osteomyelitis, which can complicate this fracture as it is often not initially recognised as a compound injury. (**b**) The fracture is associated with laceration of the overlying germinal matrix and subluxation of the nail plate from the nail fold

injury was described by Seymour [19]. The fracture is usually associated with a laceration at the base of the nail bed presenting with an apparent mallet deformity and the nail plate is often subluxed from the nail fold, with a subungual haematoma (Fig. 14.7). As the fracture is compound, there is a risk of infection, subsequent growth plate injury and growth arrest. The child often presents late, by which time the fracture is at least contaminated, if not frankly infected. The management of the wound is paramount, with thorough lavage and debridement of haematoma required with repair of the nail bed laceration. Adequate stability can be obtained with laceration repair, nail plate relocation and splinting. However, a K-wire may be required on occasion, though there is a risk of pin site infection and osteomyelitis [20]. Antibiotics are required where the presentation and treatment have been delayed.

Middle and Proximal phalangeal fractures of the head or neck are common injuries and can be unicondylar, bicondylar, in either a T or Y pattern, or a subcondylar fracture. Intra-articular fractures with displacement can produce angular and/or rotation deformity and require reduction and fixation. The diagnosis can be difficult with swelling at the joint, obscuring the deformity and with the head being largely cartilaginous in the younger child, fractures can be missed on X-ray. A true lateral X-ray can reveal a double condylar shadow, indicating volar displacement of one condyle, relative to the other. Interfragmentary screw or pin fixation is effective, but there is a risk of devascularizing the fragments with excessive mobilization and consequent osteonecrosis. A subcondylar fracture occurs when the digit is caught in a door and there is a shearing force across the joint, producing a dorsal displacement and rotation of the condylar fragment into extension (Fig. 14.8). The consequence of this is a loss of flexion, as the subcondylar fossa of the phalanx is deformed, leaving no room for the flexing phalanx to rotate into. It is recommended that as little remodeling occurs at the distal end of the phalanx (as it is remote from the growth plate). Displaced subcondylar fractures need reduction and K wire fixation, whether by closed [21] or open techniques [22]. Established malunions with loss of flexion can be addressed by ostectomy, to clear the subcondylar fossa [23] allowing flexion without impingement.

Diaphyseal fracture of the phalanges can occur and are treated according to established principles. It is important to note that the often held belief that remodeling will cure most ills in paediatric fractures does not apply to rotation deformities and these injuries need reduc-

tion and appropriate fixation (Fig. 14.9). Basal fractures of the phalanges, particularly the proximal phalanx of the border digits, are very common and usually are Type II Salter-Harris injuries. There is usually lateral angular deformity combined with extension at the fracture site. These fractures can be reduced, with the deforming force of intrinsic muscle tension eliminated by metacarpophalangeal joint flexion during reduction and post reduction splinting. For most, the fractures can be stabilized with buddy strapping and an appropriate splint, but unstable fractures can be stabilized with a percutaneous K-wire.

Fig. 14.8 Subcondylar fracture of the proximal phalanx, with rotation of the distal fragment into extension

Clinical Pearl
Beware of the child with the mallet deformity in combination with the subluxed nail or subungual haematoma as this is the common presentation of a Seymour fracture or open physeal fracture of the distal phalanx.

Metacarpal Fractures

Fracture of the metacarpal(s) is a common injury [24], particularly in the adolescent, where combat and sport can lead to axial loading of the metacarpal head, producing the angulated fracture of the neck with the apex dorsal, or dorsal fracture dislocation at the carpometacarpal joint. Direct injuries to the head of the metacarpal can occur with direct blows or bite injuries. The management of these injuries parallels the management in the adult hand. Open joint injuries sustained against a tooth or other sharp surface must be treated aggressively to prevent infection with arthrotomy and washout. In the closed injuries, joint alignment and stability must be attained to prevent ongoing symptoms and dysfunction. Extra-articular injuries are more common and usually involve the small finger ray with fracture of the neck. Rotation deformity can occur, but is difficult to assess in the acute setting with

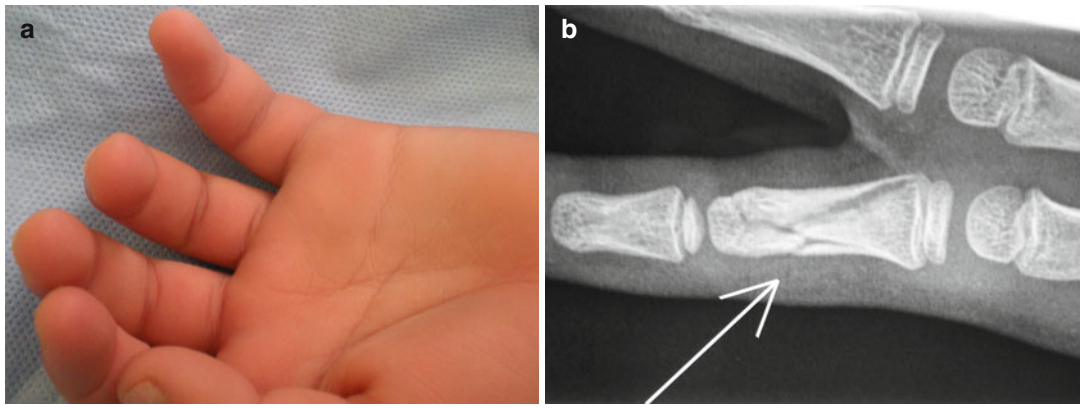


Fig. 14.9 Comminuted fracture of the diaphysis of proximal phalanx with significant rotation deformity seen on clinical examination (**a**). While the fracture is seen on X-ray (**b**), as indicated by the *white arrow*, the rotation deformity is often not obvious. This fracture requires reduction as remodelling in the plane of rotation will not occur

swelling of the hand and limitation of active flexion of the finger. Where identified, rotation deformity needs to be reduced, as it leads to a functional problem with interference of digital motion with flexion. On the other hand, a degree of flexion deformity is functionally acceptable, though may leave a visible deformity. It is reasonable to accept a residual deformity of up to 30° in the small finger metacarpal, but less in the radial digits, where there is less mobility in the carpometacarpal joints. Closed reduction of the fracture is usually adequate and stabilization with a splint, aided by maintaining the metacarpophalangeal joint in less flexion than usual in the adult to obtain leverage on the fracture [25]. A percutaneous wire or intramedullary nail may be required in the unstable fracture. Epiphyseal injuries are less common in the metacarpal, apart from the thumb, where the physis is at the base of the metacarpal and falls onto the thumb can produce a Salter Harris II or III fracture of the base, with volar angulation or dorsal translation as the metacarpal is pushed dorsally and the epiphysis, or at least part of it, is held by the carpometacarpal ligaments in situ. Reduction and percutaneous pinning to reduce the joint is appropriate.

Carpal Fractures

Carpal fractures are rare in children younger than ten and, if seen, usually are the result of a high-energy injury. Carpal injuries are seen in the adolescent with the fall onto the outstretched hand. The scaphoid is the most commonly fractured carpal bone and the most common scaphoid fracture is through the wrist [26] or distal third [27] and have a good prognosis. The diagnosis is sometimes difficult, but if the index of suspicion is high based on focal tenderness, swelling and pain with axial loading, imaging with X-ray is required. Where initial X-rays are not helpful, either a period of immobilization with repeat assessment and X-ray or further investigation with Magnetic Resonance Imaging (MRI) may be required to exclude a significant injury. Magnetic Resonance Imaging, where available, has replaced Technetium Bone Scan as a second line investigation for the screening of carpal injury. MRI (or Bone Scan) can demonstrate an occult fracture or bone bruise in the presence of normal X-rays [28]. The bone bruise is encountered more commonly in the era of MRI investigation (Fig. 14.10) and its significance in the scaphoid is unclear but probably does not lead to ongoing problems [29] and can be managed with a period of splinting until pain resolves.

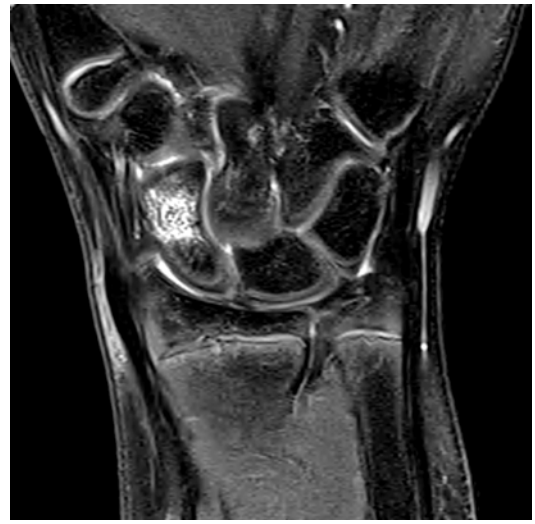


Fig. 14.10 Bone bruise of scaphoid demonstrated on MRI showing increased signal within distal pole of scaphoid following a fall onto the outstretched hand. No fracture was seen on X-rays and CT scan

The management of the acute scaphoid injury depends on the position of the fracture and benefits from imaging with a CT scan to assess displacement and fracture location and hence prognosis. Open reduction and internal fixation is required for displaced fractures or non-union. Non-union is rare in the child, provided early diagnosis and appropriate immobilization has been provided.

Distal Radius and Ulna Fractures

Fractures of the distal radius and ulna are very common. The forearm is the site of 25 % of all pediatric fractures [30] and 75 % of forearm

fractures occur in the distal third of the radius [31]. The fractures are either of the metaphysis or involve the distal radial physis. Given the proximity to a physis responsible for significant growth in the child, displaced fractures have significant remodeling potential, and this has to be taken into account with the management of the fracture.

The fractures occur due to a fall onto the limb. The peak incidence is in the early teenage years, corresponding with the period of maximum growth. There is usually swelling and often an obvious deformity, with the child reluctant to move the affected wrist. X-rays taken in two planes, including the wrist and elbow, are appropriate.

The fractures of the distal radius may be either unicortical or bicortical fractures.

The unicortical fracture (or torus) has a buckle on the compression surface, but minimal displacement. The fracture is stable and is managed with a cast or splint for 3 weeks until non-tender.

The bicortical fractures of the metaphysis of the radius may be transverse or oblique and can be angulated, translated and or rotated and represent two thirds of distal radius fractures. The remaining third involve the physis and are usually Salter Harris Type II fractures, with the physis displaced dorsally. Depending on the degree of displacement, there may be associated distal ulna or TFCC injury, or neurovascular compromise in the most significantly displaced injuries of the distal radius. While the need for reduction of grossly displaced fractures will be self-evident, the capacity for remodeling of the distal radius means that conservative treatment of moderately displaced fractures may be appropriate, depending on assessment of the remodeling capacity in the individual case. Remodeling will occur in the younger child and with fractures close to the physis and with deformity in the plane of motion of the adjacent joint. There is argument as to what constitutes an acceptable deformity, but a reasonable approach is that in the child with at least 2 years of growth remaining, 20° of angulation in the sagittal plane and up to 50 % of translational displacement is likely to remodel and can be accepted [32, 33] (Fig. 14.11). If the deformity is greater than these guidelines, or there is an associated soft tissue or neurovascular injury, operative management is indicated. Closed reduction is appropriate for most injuries, although, if initial attempts are unsuccessful, an open approach to avoid further neurovascular or soft tissue injury is used. Fracture stability can be assessed at the time of reduction. Where the reduction appears stable, circumferential cast immobilization with either above [32] or below elbow [34] cast is used for a period of 6 weeks. During this period repeat X-rays are required, as up to one third of these fractures will redisplace to an unsatisfactory position and require further reduction [35]. An alternative is to pin the fracture at the time of reduction, using either a single wire or crossed K-wires, with the pins positioned through the metaphysis rather than crossing the growth plate, if possible. This approach is used if circumferential casting is contraindicated by soft tissue swelling or neurovascular injury, or if the initial reduction has failed to maintain position. If the fracture is stabilized by a wire, the wrist is protected with a splint and the wires removed at 4 weeks.

Fractures of the distal radius can be associated with injury to the median or ulnar nerves [36]. These injuries are usually a neuropraxia, related to traction or direct contusion and improve with reduction of the fracture and stabilization. Neuropathic symptoms may also relate to acute carpal tunnel syndrome or compartment syndrome, which require surgical decompression.

In the longer term, distal radius fractures can be complicated by malunion or growth arrest if the growth plate is damaged. The latter complication occurs in approximately 4 % of cases [32]. The consequence of malunion or growth arrest can be manifest as ulnocarpal impaction, distal radioulnar joint instability or deformity. These problems can be difficult to treat, particularly as further maladaptive growth can complicate the initial deformity and there may be a co-existent ligamentous injury. A range of procedures can be used, including radial and/or ulnar osteotomy or epiphysiodesis depending on the deformity and age of the patient to symptoms and correct deformity [37].

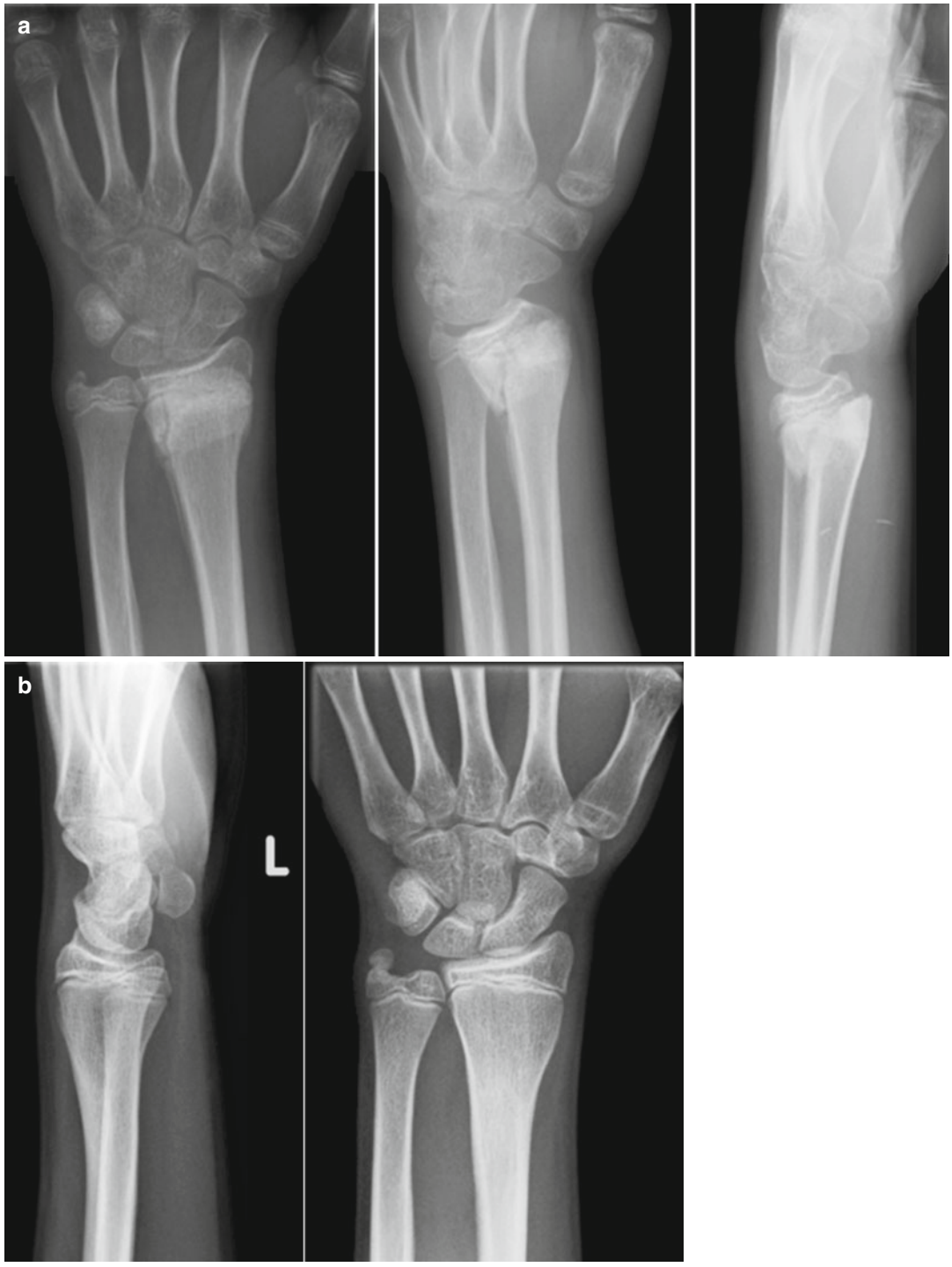


Fig. 14.11 Remodelling of an angulated distal radius fracture. The fracture was initially treated with a cast alone after the initial position of dorsal angulation was accepted (**a**). At 12 months after the fracture, the deformity has improved to an acceptable position (**b**)

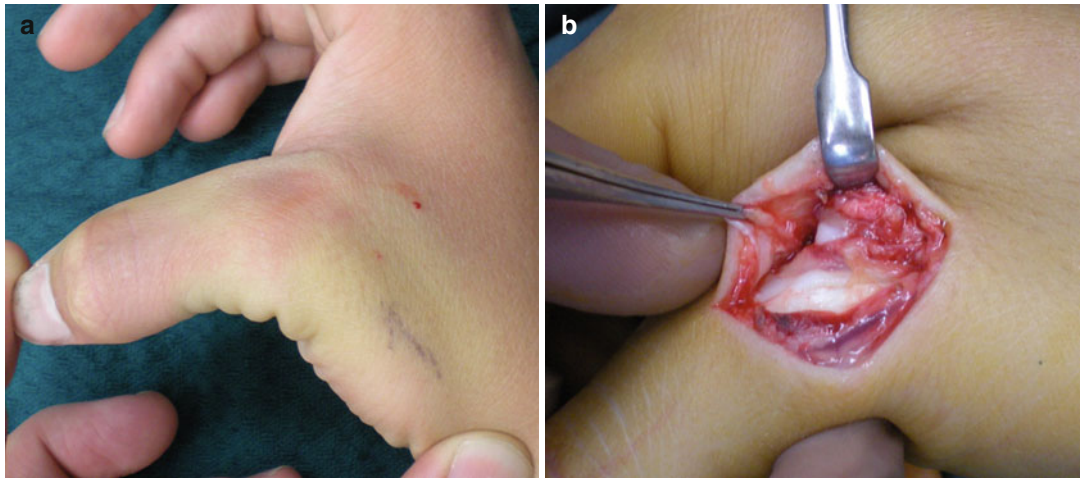


Fig. 14.12 Complete rupture of Ulnar Collateral ligament of the Metacarpophalangeal Joint of the thumb following a football injury with instability of joint demonstrated with stress of the ulnar ligament (**a**). The diagnosis of avulsion of the ligament from the proximal phalanx was confirmed on exploration (**b**). The joint was repaired by reattachment of the ligament using a bone anchor

Joint Injury

Joint injuries without fracture are uncommon in children, given the relative strength of the joint capsule and ligaments. Joint dislocations and ligamentous injuries that occur in children present with local swelling, tenderness, restriction of motion and deformity in the case of persistent dislocation. Associated avulsion fractures may be seen on X-ray and ultrasound can be useful in establishing whether a specific ligament is intact.

Interphalangeal joint dislocations are rare compared to the adult populations, with projectile injuries usually producing fractures in the child. Metacarpophalangeal joint dislocations are the most common dislocation in the child's hand [38], with the thumb most commonly involved. In the digital rays, the proximal phalanx dislocates dorsally and the metacarpal head is prominent in the palm. These dislocations can be difficult to reduce. The volar plate usually remains attached to the phalanx and can become interposed into the joint. The metacarpal head can become entrapped between the lumbrical and flexor tendon, with longitudinal traction on the digit exacerbating the entrapment as described by Kaplan [39]. Closed reduction can be attempted by hyperextension and palmar displacement of the phalanx, rather than longitudinal traction. If unsuccessful, open reduction is required with both dorsal [40] and palmar approaches described. From the palmar approach, the digital nerves are displaced into a superficial position by the prominent metacarpal head and are at risk whilst the volar plate can be difficult to retrieve if dorsal to the metacarpal head. However, the flexor tendon and lumbrical can be retracted to allow the joint to be reduced and the volar plate can be repaired, if necessary. A short period of dorsal block splinting is required post reduction. Joint stiffness, growth arrest and osteonecrosis can complicate the injury in the longer term [38].

In the thumb, the metacarpophalangeal joint can dislocate dorsally, with injury to the volar plate and collateral ligaments. The child presents with deformity at the joint, usually hyperextension, and swelling. The injuries have been classified into incomplete, complete or complete complex dislocations, depending on the extent of the ligamentous injury and the extent of the phalangeal displacement. Reduction of the metacarpophalangeal joint requires longitudinal traction and flexion but, in complete injuries, the volar plate may be interposed in the joint at presentation (a complete complex dislocation), or during attempted reduction of the complete dislocation.

Interposition of the volar plate will prevent closed reduction and open reduction, either with a palmar or dorsal approach is required. The reduced joint needs at least 3 weeks of dorsal block splinting for stabilization of the joint's soft tissues.

Clinical Pearl
The thumb deformity associated with a trigger thumb can be mistaken for an incomplete dorsal metacarpophalangeal joint dislocation. The trigger thumb should be at least passively flexible at the metacarpophalangeal joint and a nodule is often palpable in the flexor tendon.

The thumb is commonly injured by a valgus force across the thumb metacarpophalangeal joint. The effect on the child is usually a Salter Harris type II fracture, at the base of the proximal phalanx. In the adolescent with a maturing skeleton, the injury is more likely to produce a sprain or avulsion of the ligament from its distal attachment or an avulsion fracture (a Salter Harris Type III fracture) at the base of the proximal phalanx. The distal end of the avulsed ligament can be caught by the adductor aponeurosis as the joint reduces and remain dorsally displaced superficial to the aponeurosis, as described by Stener [41]. The diagnosis of a complete injury is made clinically on the basis of a lack of an end point to abduction stress (Fig. 14.12) or if an avulsion fracture is demonstrated on X-ray. An incomplete ligament injury and an undisplaced fracture is treated with a splint for 4–6 weeks. A complete ligament injury with instability or a significantly displaced fracture requires surgery. A dorsal approach is used, with care taken to look for the ligament adherent to the proximal margin of the adductor aponeurosis. An incision is made in the margin of the thumb extensor tendon at the insertion of the adductor aponeurosis and this is reflected ulnarward to expose the ulnar joint capsule. The acute ligamentous injury is evident by this point, but the joint capsule may be intact if the injury is a fracture and capsulotomy required. Fixation of fractures, or the reinsertion of the phalangeal attachment of the ligament, needs to take the adjacent physis into account and care is required to avoid pins, screws or anchors crossing or damaging the growth plate. Chronic injuries are more difficult to manage in children as tendon graft reconstructions run the risk of injury to the physis and chondrodesis has been advised in this circumstance to stabilize the joint [42].

Significant ligament injuries in the wrist are very rare and, when they occur, they do so in the setting of a high energy injury [43]. Scapholunate ligament injury is, however, misdiagnosed frequently on X-ray because the delay in proximal pole ossification gives rise to the appearance of a widened scapholunate interval. Usually this can be excluded on clinical grounds or using lateral X-rays or other imaging modalities.

Distal radioulnar joint (DRUJ) ligament injury and or joint instability is seen in children and adolescents. The stability of the DRUJ depends on joint congruity and the integrity of the radioulnar ligaments and their attachment to the distal ulna. Distal radius fractures with residual deformity leads to altered mechanics of rotation and loss of joint congruity and stability. Injury to the radioulnar ligaments, with peripheral tears of the triangular fibrocartilage complex or avulsion of their insertion to the fovea of the ulnar, also leads to instability. Fractures involving the base of the ulnar styloid disrupts the point of ligament attachment and hence joint stability. Symptomatic DRUJ instability requires investigation with X-rays and, where indicated, MRI. Treatment is directed to the cause of the joint instability, by either corrective osteotomy of the radius, repair of the TFCC, (either by arthroscopic or open techniques) and/or fixation of ulnar styloid fractures. Apart from producing instability, injury to the soft tissues of the ulnocarpal and distal radioulnar joints may be symptomatic with ulnar sided wrist pain, particularly with loading of the wrist in extension or rotation or with grip. While most of these injuries settle with rest and strapping or splint protection, persistent pain may indicate a tear of the TFCC, which can be addressed arthroscopically, either by debridement or repair with good results reported in children and adolescents [44].

Tendon Injury

Tendon injuries in children, particularly the infant and young child, present challenges of diagnosis and management, as their capacity to co-operate with the surgeon and hand therapist varies from moment to moment and child to child.

Injuries to the tendons of the hand are either to the extensor or flexor tendons, closed or open injuries and either isolated tendon injuries or part of a more complex combined injury. Injuries to the flexor and extensor systems are classified according to the anatomical site of injury, with management varying, in part, according to the level of injury. The principles of management of children's tendon injuries are essentially the same as those that guide the management of injuries in the adult, with consideration of age specific issues of diagnosis, the technical challenges of the smaller calibre tendons and postoperative rehabilitation. The following text will focus on these points of difference rather than reiterate the principles of tendon injury management that will be discussed elsewhere.

The diagnosis of tendon injury depends on the circumstances of the injury. Lacerations related to the course of flexor or extensor tendons place these structures at risk and significant lacerations require exploration. While an absence of active motion in the affected tendon can be expected, this can be difficult to demonstrate without co-operation and the deficit can be concealed by trapping the affected digit with an adjacent digit and producing apparent active motion. Children are also vulnerable to closed injuries, either by forced extension of the flexing digit, producing avulsion of the FDP tendon (a jersey finger) (Fig. 14.13) or forced flexion of the extended digit by an axial injury with a ball or fall, producing avulsion of the extensor tendon from

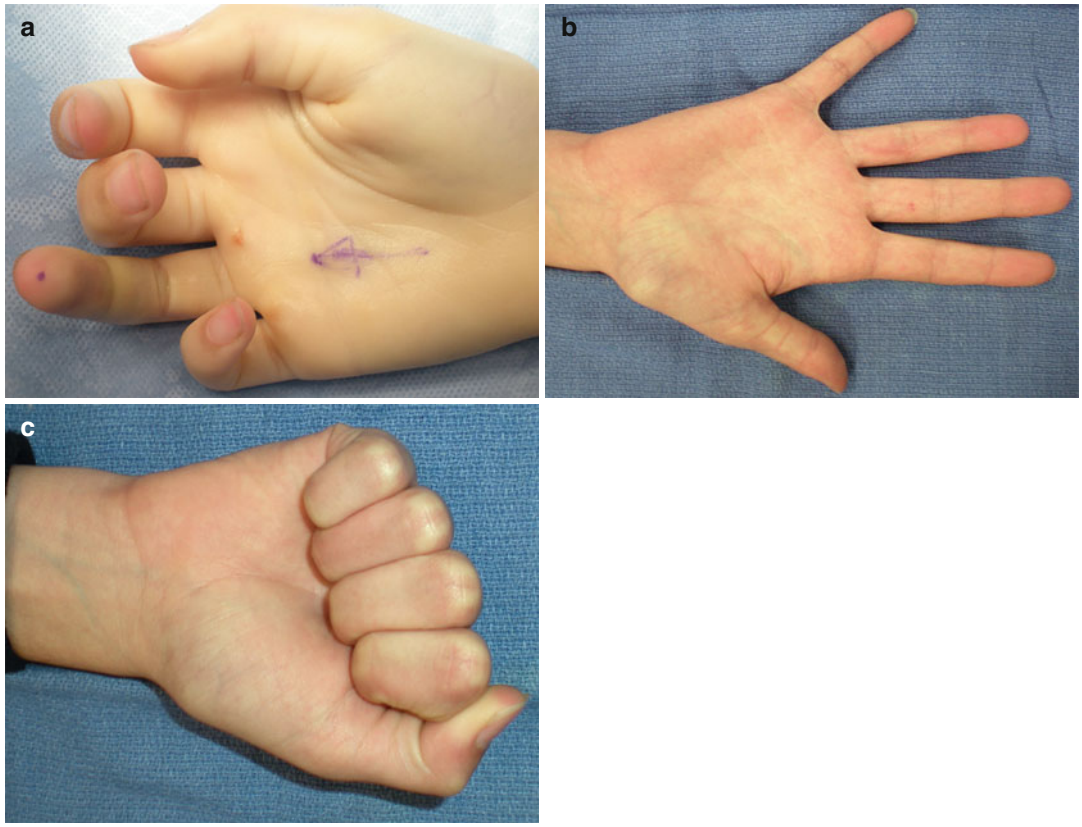


Fig. 14.13 Avulsion of the Flexor Digitorum Profundus (FDP) tendon from the distal phalanx following hyperextension injury of ring finger. The cascade of the digits was disrupted and there was no active flexion of the distal joint (**a**). Following repair of the FDP tendon, active flexion is restored (**b**, **c**)

either the PIP joint (a Boutonniere injury) or DIP joint (a mallet injury). Closed tendon injuries can be misdiagnosed as joint sprains and a patient's clinical examination, aided by imaging with X-ray and Ultrasound, is required where the potential for this diagnosis exists. Clinical examination should include assessment of the posture of the affected digit or the hand as a whole, assessment of whether the tenodesis effect is intact or for flexor tendons in particular, whether there is digital flexion elicited by compression of the flexor tendon mass in the distal forearm. In the older child active motion can be elicited with the co-operation of the patient.

Flexor Tendon Injury

The management of tendon injury in the child is the same as the adult. Acute flexor tendon injuries are managed, where possible, with direct repair using the same techniques as used in the adult, with preservation of the pulley system, atraumatic handling of the tendon and a combination of core and epitendinous suture to achieve a robust coaptation of the tendon, without excessive bulk that glides in the fibro-osseous tunnel of the flexor sheath. The size of the tendon does constrain the suture technique and judgement is required as to core suture calibre and the core suture purchase length should be approximately 1.5 times the tendon width [45]. A number of core suture techniques have been described, from 2 to 8 strand techniques. No difference in outcome has been demonstrated between 2 and 4 strand techniques in young children [46]. The advantage of multistrand techniques is an increase in tensile strength, that allows active motion rehabilitation protocols [47]. However, immobilization is a most common postoperative protocol in children and considering the calibre of the young child's flexor tendon, a 2 strand modified Kessler core suture is the most commonly used technique in this surgeon's practice. Multistrand techniques have a place in the older child, where active rehabilitation protocols may be appropriate. The epitendinous suture is a continuous suture that aims to produce a smooth surface to the repair for gliding. In the young child, an absorbable suture should be used, to prevent constriction at repair site as the remainder of the tendon grows [48]. There is debate as to whether both flexor tendons should be repaired in Zone II injuries. The FDS tendon provides a dynamic pulley and a gliding surface for the FDP tendon, but a bulky repair of the FDS tendon can obstruct the motion of the repaired FDP tendon, leading to rupture or adhesion. In children, repair of both FDP and FDS in Zone II have been associated with worse results than FDP repair only [49, 50].

Rehabilitation of flexor tendon repair is complex because of the competing interests of preventing tendon adhesion and allowing the tendon repair to heal without gapping or dehiscence. Rehabilitation protocols vary from immobilization, to Kleinert's rubber band controlled motion programme, to passive motion programmes, to active motion programmes. A Cochrane review of available evidence was unable to conclude which programme is best [51], but it is accepted that motion protocols are preferred to immobilization and there is a trend towards early active motion programmes. In young children, however, motion based protocols are difficult to implement because of the co-operation required. Fortunately good outcomes are achieved with immobilization in children, though this should be limited to 4 weeks and an above elbow splint used in younger children to limit the risk of splint displacement and rupture of the repair [52]. Following the initial immobilization period, where the wrist is held flexed to 30° and MP joints flexed to 70°, a dorsal blocking splint is applied for 2 weeks to allow protected motion and then further rehabilitation is tailored to the child, as persisting flexion contracture may need to be addressed with splinting or passive stretching [53]. The decision as to when a child is old enough to cope with a mobilization programme is difficult and depends upon the individual child.

While good to excellent results can be achieved in children with flexor tendon repair, poor outcomes do occur with rupture rates of 7–12 % reported [50, 52] and adhesions limiting active tendon excursion in a small proportion as well. Tendon repair rupture is difficult to diagnose in the younger child and, while re-exploration and

repair is normally indicated, delay in diagnosis may preclude this. Tendon adhesions are another challenge in children. Improvement in active motion, post repair, can occur over the longer term with growth [52] and it has been suggested that tenolysis be delayed until at least 11 years of age, when co-operation with a rehabilitation programme is more likely [54].

Unfortunately, there is a higher risk of delayed diagnosis of tendon injury in the child [50, 55]. This presents a management challenge, particularly in the flexor tendon injury because muscle and tendon contracture, with loss of excursion and collapse of the flexor sheath, make primary tendon repair difficult and increases the risk of joint contracture after repair. Delayed primary repair, up to at least 6 weeks, is reasonable and probably better tolerated in the child than the adult, but judgment is required at the time of exploration. The patient and parent should be prepared for the alternative of a tendon graft reconstruction, either in one or two stages, depending on the condition of the digit and the flexor sheath. It is tempting to delay tendon reconstruction in younger children until they reach the point where they can co-operate with rehabilitation. However, this risks placing the digit at risk of further injury as it remains extended with grasp and also, because of the absence of the dynamic stimulus of motion and force, the growth of the digit may be retarded [56, 57]. The results reported after either one or two stage tendon reconstructions vary [58, 59], but reasonable results can be achieved, although they require close postoperative supervision to avoid complications including infection, tendon rod exposure, graft adhesion or rupture.

Extensor Tendon Injury

Given the anatomy of the extensor hood in the digit, closed avulsion injuries at the DIP and PIP joints can be managed with splinting alone, as significant tendon retraction does not occur and if the joint can be maintained in an extended position, the tendon insertion can effectively heal. Open injuries warrant repair, though with care not to shorten or unbalance the complex extensor mechanism in the finger. Injuries at the level of the DIP or PIP joint usually need to be supplemented by temporary K wire stabilization of the joints to prevent dehiscence and extensor lag [60]. Injuries to the extensors proximal to the MP joint have a better prognosis as the conformation of the tendon is better suited to a robust core suture repair. Rehabilitation usually requires 4 weeks of immobilization of the hand, with wrist extension and MPJ flexion of less than 30°.

Nerve Injury

The anatomy of the peripheral nerve and the pathophysiology of nerve injury are well described. Ongoing research into the biology of nerve injury, regeneration and recovery has made significant progress [61]. It is clear that nerve injury has broad implications with changes in the nerve itself, distally in the muscle or sensory end organ and centrally in the brain. Distal to the injury, the nerve axon is degraded and muscle or sensory receptors degenerate while waiting for reinnervation. Proximal to the level of injury, there is a change in the nerve cell body, due to the loss of the neurotrophic effect of factors transported from the periphery back to the cell body. This process leads to the structural changes in the nerve cell known as chromatolysis and converts the cell to a regenerative phenotype, though a significant proportion of the proximal cell bodies fail to survive the peripheral injury to their axon. For the surviving population, axonal sprouting occurs and provided axons are maintained and directed appropriately by the connective tissue and Schwann cell network of the distal nerve. It is now recognized that changes occur centrally in the somatosensory and motor cortex, with initial disruption and then with peripheral reinnervation, reorganization of the cortical response [62]. Children make a better recovery from peripheral nerve injury than adults with similar injuries [63, 64]. Experimental work shows that the process of peripheral nerve regeneration is slower and less complete in older animals and also that the younger child has better central plasticity, allowing

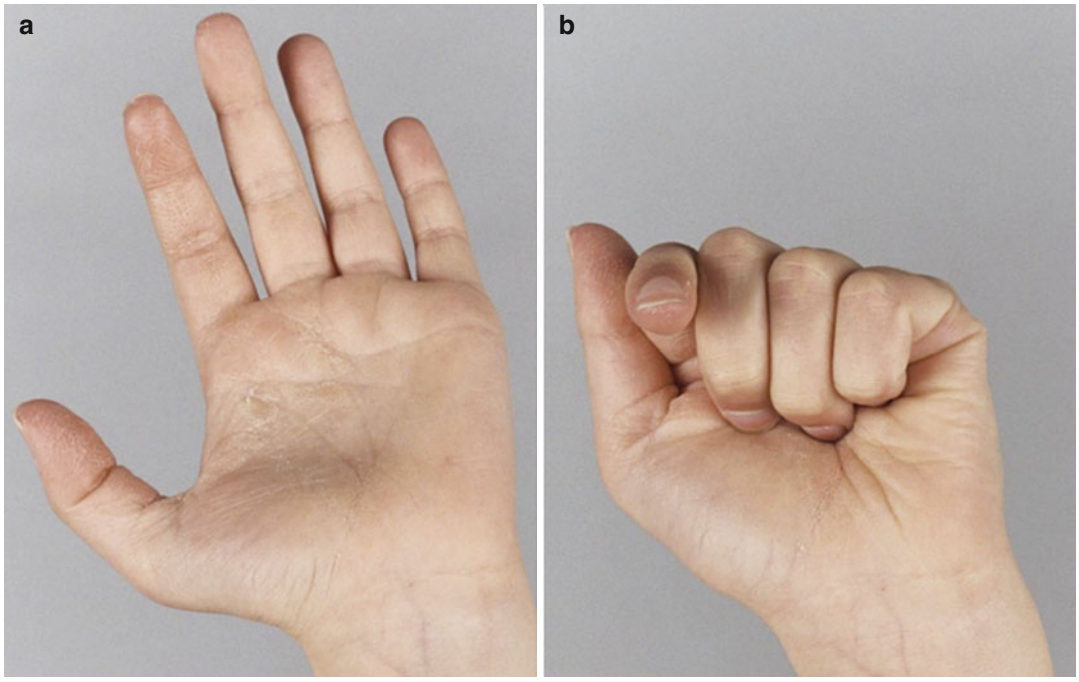


Fig. 14.14 The consequences of a proximal median nerve injury are shown with trophic changes of the skin in the distribution of the median nerve, (**a**) and the characteristic 'benediction' posture with loss of thumb and index flexion (**b**)

more complete reorganization of the cortical responses as peripheral reinnervation proceeds [62, 65].

While the biology of nerve regeneration is favourable in the child, the clinical situation can be difficult. The classifications of nerve injury by Seddon [66] and Sunderland [67] describe the spectrum of injury and the potential for recovery of nerve function and they are the basis of the algorithm for treatment of nerve injury. Closed injuries that leave the peripheral nerve or its connective tissue structure intact have potential for spontaneous recovery, either by recovery from the neuropraxia or axonal regeneration, within an intact supporting framework. Injury that disrupts the nerve structure by either closed traction or crush, or an open laceration, has poor or no potential for spontaneous recovery and requires surgical repair or reconstruction.

In the acute setting, a laceration in the vicinity of a significant nerve warrants exploration. The management of the closed injury is more difficult. Determining the prognosis of the closed nerve injury relies on an adequate history to determine risk of nerve injury alongside relevant symptoms and serial examination to assess whether there is evidence of recovery. The standard sensory and motor testing can be attempted where the child is co-operative. For the younger child, the examiner can establish whether innervation is intact by testing for the presence of sweating, with the tactile adherence test or the wrinkling response of the glabrous skin of the fingertip to immersion in warm water for 5 min. If the child's presentation is delayed, the effects of established denervation, such as dry skin and muscle wasting, in the distribution of the affected nerve or typical postural changes such as clawing of the ulnar nerve injury, the simian hand posture of the distal median nerve injury and the 'benediction' posture of the proximal median nerve injury (Fig. 14.14), are seen. Digital tip ulceration with atrophy or signs of chewing of the fingertips may also be seen with severe sensory impairment.

Advances in imaging with high definition ultrasound and MRI allow an assessment of the

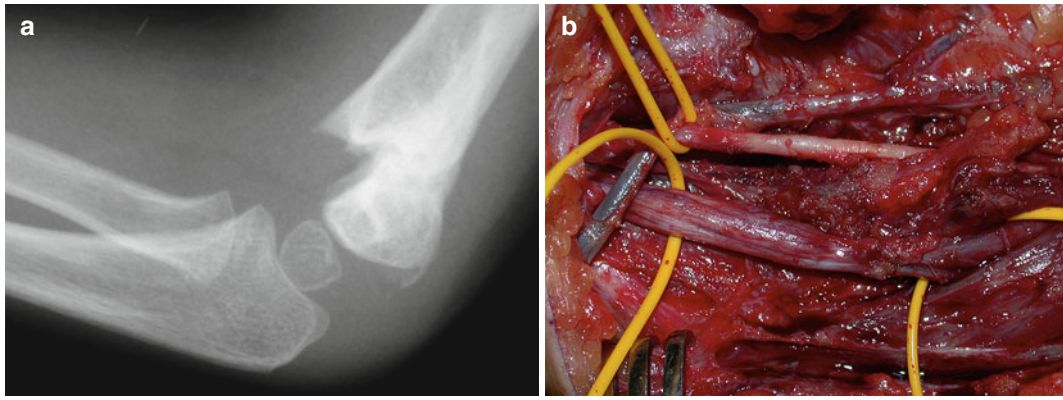


Fig. 14.15 Malunion of a supracondylar fracture of the humerus with a residual step deformity (**a**). This deformity was associated with a median nerve palsy and exploration demonstrated compression of the median nerve and brachial artery, (marked by vessel loops in **b**)

peripheral nerve [68], and nerve conduction studies can be used to document the extent of nerve injury and reinnervation [69]. However, clinical assessment remains the most important tool in the management of peripheral nerve injury.

A goal of management of the peripheral nerve injury is early identification of the injury and repair, or reconstruction of the nerve. Early exploration of the transected nerve usually allows for direct coaptation, rather than facing a nerve gap that is likely after fibrosis and resection of the stump neuromas formed during any delay after injury. There is evidence that early repair of the nerve favours neuron survival because of the early re-introduction of the neurotrophic effect of factors transported from the distal nerve stump [70] and also minimizes the period of denervation and its dystrophic effects upon the muscle and sensory end organ. Where early exploration is undertaken and the nerve is contused, but in continuity, the nerve is left intact, as there is potential for good recovery of lower grade injuries, particularly in children. The closed injury to the nerve, either by crush or traction, is usually observed on the presumption that the nerve is intact and the injury is of lower grade and, hence, spontaneous recovery is likely. Exploration is indicated if there is no evidence of reinnervation or recovery by 3 months. Earlier exploration of these injuries may be indicated, where the circumstances of injury make a higher grade injury more likely, such as a significantly displaced supracondylar humeral fracture that has been incompletely reduced and there is potential for ongoing nerve entrapment or impingement (Fig. 14.15).

Repair of the lacerated nerve is usually performed with direct coaptation of the nerve ends by epineural suture in smaller nerves, or by grouped fascicular suture in larger mixed nerves with the intention of trying to match the fascicular topography of the nerve ends. While the alignment of a single fascicle nerve is usually straightforward, cues such as the fascicular pattern and epineural vessels help in aligning larger nerves. Nerve ends can be trimmed to limit fascicular extrusion at the repair site and the epineural or perineural sutures are tensioned so as not to bunch the nerve ends at the repair and disrupt fascicular alignment. Fibrin glue applied around the repair can augment repair strength. The repair should be tension free, so as to minimize ischaemia of the nerve ends. This can be achieved by judicious mobilization of the nerve ends and positioning of the limb. Care should be taken if an extreme position is required to achieve direct coaptation, as the child is unlikely to maintain this post surgery, even with protective splinting and it may be better to accept that a nerve graft is necessary rather than risk disruption of the repair.

If there has been delay in exploration, with consequent fibrosis and retraction of nerve ends or nerve loss at the time of injury or there is a non-conducting neuroma in continuity that

requires resection, reconstruction of the nerve with a conduit or graft will be required. The optimal conduit is autogenous nerve graft, usually as either a single length or as multiple cables interposed between fascicles. For short defects, particularly of sensory nerves, alternative conduits can be effective and are attractive in minimizing donor site morbidity in children, who have the advantage of greater potential for functional recovery. These alternative conduits include the biological conduits of vein [71], muscle [72] or a vein filled with muscle [73], or biodegradable engineered conduits of polyglycolic acid [74], collagen [75] or caprolactone [76].

For proximal nerve injuries, where the prognosis for recovery is limited given the delay in reinnervation, transfer of an expendable functioning nerve adjacent to the denervated target has some advantages [77]. The greater capacity for nerve regeneration and cortical plasticity of children may limit the relative advantage of the nerve transfer, apart from situations where the proximal nerve stump is unavailable, or of poor quality. Nerve transfers have been shown to be effective in obstetrical brachial plexus palsy, but there have been few reports of their use in children in nerve injuries distal to the plexus. Another technique that has been used in circumstances where the proximal nerve stump is unavailable and there is no expendable donor nerve is end to side nerve repair. The distal nerve stump is coapted to the side of an intact nerve via a perineural window, with reinnervation occurring, due to the sprouting of collateral nerves from the intact neurons into the distal stump [78]. While both sensory and motor sprouting has been demonstrated in experimental models, clinically it would appear to be more reliable in reconstruction of sensory nerves, such as digital nerve lesions [79].

Vascular Injury

Injury of the axial arteries of the upper limb can occur by open or closed trauma, or as an iatrogenic injury by puncture or cannulation of the artery. The consequences of arterial injury in the upper limb may be gangrene with tissue loss, critical ischaemia with contracture, nerve injury and trophic changes, or claudication with exercise or cold exposure. In the paediatric age group, arterial injury with ongoing relative ischaemia also can lead to growth retardation with limb length discrepancy [80].

Significant open trauma often involves a significant vascular injury, which may be evident by a history of significant blood loss or peripheral ischaemia or, perhaps more commonly, may be an occult injury and identified at the time of exploration for co-existent injuries. Children have a small circulating blood volume and the effect of significant blood loss, either as a retained haematoma or external blood loss, should not be underestimated. Control of active bleeding can usually be obtained by direct pressure, with care taken not to produce a venous tourniquet effect with an inexpertly applied bandage proximal to the injury. Early exploration is indicated, rather than proceeding with investigations apart from an X-ray, which may be indicated for assessment of co-existent skeletal injury. Ongoing bleeding is often an indication of a partial laceration rather than complete division, particularly of the distal arteries. Complete division of an axial artery is usually associated with retraction of the ends of the vessel, particularly of the proximal vessels and extensile exposure of the neurovascular bundles may be required to identify the stumps of the divided arteries, with care taken not to divide collateral branches. If the artery can be mobilized without tension, direct repair is preferable. However, commonly, because of retraction or damage to the artery, a reversed vein graft is commonly required to repair a segmental defect.

If the limb is ischaemic, it is important to minimize the interval from injury to restoration of circulation, particularly with a proximal injury, where the risk of distal muscle ischaemia is significant. Muscle ischaemia of more than 4 h has systemic consequences of acidosis, hypotension and myoglobinuria and the local consequences of swelling and potentially compartment syndrome. These issues need to be monitored or addressed prophylactically.

Closed injuries of the upper limb arteries are usually associated with fractures or dislocations,

particularly about the elbow and less commonly due to direct crush or blast injury. The artery can be contused, stretched, entrapped within the fracture or joint, or ruptured. Reduction of the skeletal deformity is often enough to restore adequate flow, though if the limb remains pulseless after fracture reduction and fixation, particularly if there is compromise of distal perfusion or an associated nerve palsy, exploration of the neurovascular bundle is warranted [81, 82], so as to avoid the disastrous complication of Volkmann's ischaemic contracture. At operation the artery may require arteriolysis only, thrombectomy or, where the artery is damaged over a significant segment and there is evidence of intramural haemorrhage and dissection, resection of the damaged segment and reconstruction with vein graft. Following surgery, the artery repair or reconstruction is protected together with any nerve, tendon or skeletal repairs, with a splint for 3–4 weeks. Anticoagulants, thrombolytics and antiplatelet drugs, such as Aspirin, have been used perioperatively, though in the author's practice they are generally reserved for patients requiring thrombectomy or vein grafts.

Iatrogenic injuries to arteries of the upper and lower limbs are seen in paediatric practice, as a consequence of arterial cannulation or puncture, particularly in the neonatal age group. The artery can be occluded by the cannula or by associated spasm or thrombus that persists or develops after the cannula is removed. Pseudoaneurysms can also develop after arterial puncture, or even arteriovenous fistulas, if there has been puncture or cannulation of both artery and vein.

Management of these injuries requires accurate assessment with Doppler ultrasound to determine the extent of injury and quality of distal flow and clinical assessment of the distal tissue perfusion. An algorithm for management of these injuries has been developed [83]. The most common scenario is of relative limb ischaemia, with some distal flow reconstituted by collateral vessels and it is appropriate to institute anticoagulation and/or thrombolytic therapy and monitor the limb closely. If the condition of the limb deteriorates, or is completely ischaemic at presentation with no distal flow demonstrated, the risk of limb loss is significant and exploration and arterial reconstruction is required (Fig. 14.16).

Complex and Mutilating Hand Injuries

A child with a mutilating injury of the upper limb with devascularisation, amputation or a significant complex injury requires a plan formulated for repair or reconstruction of the injury. Fortunately, these injuries are relatively uncommon in children. However, the implications of tissue loss or damage for long term hand function and acceptance, requires the surgeon to, in the words of del Pinal, bring some organization to the chaos of these injuries [84]. Injury outcome scores have been applied in the management and estimation of prognosis of adult lower limb [85] and, more specifically, hand injuries [86]. While these have been applied to the child, the value is arguable and decisions regarding salvage or reconstruction of these complex injuries are made on an individual basis, with the benefit of the doubt in favour of reconstruction rather than amputation.

For these complex injuries, preoperative assessment is difficult as exposure of the part will usually be painful but, optimally, some estimate made of whether tissue loss has occurred and, hence, whether the child and parents need to be consented for use of donor sites remote to the injury, or whether the reconstruction is likely to be achieved in a single or multiple stages. A significant compound injury, particularly with associated contamination, tissue loss or devascularisation requires broad-spectrum antibiotic therapy, commencing in the emergency room and maintained perioperatively. The status of the child's tetanus immunization must also be confirmed and addressed if indicated.

The initial operative management is debridement of non-vital tissue, so as to allow a thorough evaluation of the wound. Debridement should be conservative, particularly of tendons and nerves, but foreign material needs to be removed by sharp dissection and lavage to limit the risk of infection in contaminated wounds. Where there are

Fig. 14.16 Critically ischaemic upper limb with thrombotic occlusion of the brachial artery following cannulation (**a**). Circulation was restored by excision and vein graft reconstruction of the brachial artery and the limb has been largely preserved (**b**)

amputated parts, these can prepared simultaneously on a side table with an evaluation made as to whether the parts can be replanted orthotopically or whether they can be used for spare parts. The next step is to obtain skeletal stability and then repair or reconstruction of the injured tendons or neurovascular structures, the priority of repair depending on the vascular status of the limb or hand. Adequate soft tissue coverage may be available for direct closure, or tissue can be imported in the form of graft or flap depending on the wound.

The psychological aspects of the injury for the child and parents can be compounded by the trauma of treatment and the stigma of the disability and disfigurement of the hand. It is easy to underestimate this effect on the child and parents, both acutely and as an ongoing effect for hand function and for the child's and parent's future interaction with the treating surgeon specifically and doctors in general. It is important to address these issues with counseling of both child and parents, beginning in the acute setting, as early attention to the psychological trauma and anxiety can help with acceptance of and adaptation to the physical effects of the injury [87, 88].

References

1. Ljungberg E, Dahlin LB, Granath F, Blomqvist P. Hospitalized Swedish children with hand and forearm injuries: a retrospective review. Acta Paediatr. 2006;95(1):62–7.
2. Vadivelu R, Dias JJ, Burke FD, Stanton J. Hand injuries in children: a prospective study. J Pediatr Orthop. 2006;26(1):29–35.

3. De Jonge JJ, Kingma J, van der Lei B, Klasen HJ. Phalangeal fractures of the hand. An analysis of gender and age-related incidence and aetiology. J Hand Surg Br. 1994;19(2):168–70.
4. Valencia J, Leyva F, Gomez-Bajo GJ. Pediatric hand trauma. Clin Orthop Relat Res. 2005;432:77–86.
5. Evans DM, Bernardis C. A new classification for fingertip injuries. J Hand Surg Br. 2000;25(1):58–60.
6. Roser SE, Gellman H. Comparison of nail bed repair versus nail trephination for subungual hematomas in children. J Hand Surg Am. 1999;24(6):1166–70.
7. Strauss EJ, Weil WM, Jordan C, Paksima N. A prospective, randomized, controlled trial of 2-octylcyanoacrylate versus suture repair for nail bed injuries. J Hand Surg Am. 2008;33(2):250–3.
8. Johnson M, Shuster S. Continuous formation of nail along the bed. Br J Dermatol. 1993;128(3):277–80.
9. Ishikawa K, Ogawa Y, Soeda H. A new classification of the amputation level for the distal part of the finger. J Jpn SRM. 1990;3:54–62.
10. Tamai S. Twenty years' experience of limb replantation–review of 293 upper extremity replants. J Hand Surg Am. 1982;7(6):549–56.
11. Hattori Y, Doi K, Ikeda K, Abe Y, Dhawan V. Significance of venous anastomosis in fingertip replantation. Plast Reconstr Surg. 2003;111(3):1151–8.
12. Hsu CC, Lin YT, Moran SL, Lin CH, Wei FC. Arterial and venous revascularization with bifurcation of a single central artery: a reliable strategy for Tamai Zone I replantation. Plast Reconstr Surg. 2010;126(6):2043–51.
13. Han SK, Chung HS, Kim WK. The timing of neovascularization in fingertip replantation by external bleeding. Plast Reconstr Surg. 2002;110(4):1042–6.
14. Dautel G, Barbary S. Mini replants: fingertip replant distal to the IP or DIP joint. J Plast Reconstr Aesthet Surg. 2007;60(7):811–5.
15. Eo S, Hur G, Cho S, Azari KK. Successful composite graft for fingertip amputations using ice-cooling and lipo-prostaglandin E1. J Plast Reconstr Aesthet Surg. 2009;62(6):764–70.
16. Hong JP, Lee SJ, Lee HB, Chung YK. Reconstruction of fingertip and stump using a composite graft from the hypothenar region. Ann Plast Surg. 2003;51(1):57–62.
17. Salter RB, Harris WR. Injuries involving the epiphyseal plate: instructional course lectures of the AAOS. J Bone Joint Surg Am. 1963;45:587–622.
18. Papadonikolakis A, Li Z, Smith BP, Koman LA. Fractures of the phalanges and interphalangeal joints in children. Hand Clin. 2006;22(1):11–8.
19. Seymour N. Juxta-epiphysial fracture of the terminal phalanx of the finger. J Bone Joint Surg. 1966;48(2):347–9.
20. Al-Qattan MM. Extra-articular transverse fractures of the base of the distal phalanx (Seymour's fracture) in children and adults. J Hand Surg Br. 2001;26(3):201–6.
21. Waters PM, Taylor BA, Kuo AY. Percutaneous reduction of incipient malunion of phalangeal neck fractures in children. J Hand Surg Am. 2004;29(4):707–11.
22. Al-Qattan MM. Phalangeal neck fractures in children: classification and outcome in 66 cases. J Hand Surg Br. 2001;26(2):112–21.
23. Simmons BP, Peters TT. Subcondylar fossa reconstruction for malunion of fractures of the proximal phalanx in children. J Hand Surg Am. 1987;12(6):1079–82.
24. Hastings 2nd H, Simmons BP. Hand fractures in children. A statistical analysis. Clin Orthop Relat Res. 1984;188:120–30.
25. Cornwall R. Finger metacarpal fractures and dislocations in children. Hand Clin. 2006;22(1):1–10.
26. Waters PM. Operative carpal and hand injuries in children. J Bone Joint Surg Am. 2007;89(9):2064–74.
27. Elhassan BT, Shin AY. Scaphoid fracture in children. Hand Clin. 2006;22(1):31–41.
28. Sferopoulos NK. Bone bruising of the distal forearm and wrist in children. Injury. 2009;40(6):631–7.
29. La Hei N, McFadyen I, Brock M, Field J. Scaphoid bone bruising–probably not the precursor of asymptomatic non-union of the scaphoid. J Hand Surg Eur Vol. 2007;32(3):337–40.
30. Joseph B. Fractures of the forearm bones. In: Gupta A, Kay S, Scheker L, editors. The growing hand. London: Mosby; 2000. p. 567–81.
31. Blount WP. Forearm fractures in children. Clin Orthop Relat Res. 1967;51:93–107.
32. Bae DS, Waters PM. Pediatric distal radius fractures and triangular fibrocartilage complex injuries. Hand Clin. 2006;22(1):43–53.
33. Younger AS, Tredwell SJ, Mackenzie WG, Orr JD, King PM, Tennant W. Accurate prediction of outcome after pediatric forearm fracture. J Pediatr Orthop. 1994;14(2):200–6.
34. Chess DG, Hyndman JC, Leahey JL, Brown DC, Sinclair AM. Short arm plaster cast for distal pediatric forearm fractures. J Pediatr Orthop. 1994;14(2):211–3.
35. Miller BS, Taylor B, Widmann RF, Bae DS, Snyder BD, Waters PM. Cast immobilization versus percutaneous pin fixation of displaced distal radius fractures in children: a prospective, randomized study. J Pediatr Orthop. 2005;25(4):490–4.
36. Waters PM, Kolettis GJ, Schwend R. Acute median neuropathy following physeal fractures of the distal radius. J Pediatr Orthop. 1994;14(2):173–7.
37. Waters PM, Bae DS, Montgomery KD. Surgical management of posttraumatic distal radial growth arrest in adolescents. J Pediatr Orthop. 2002;22(6):717–24.
38. Light TR, Ogden JA. Complex dislocation of the index metacarpophalangeal joint in children. J Pediatr Orthop. 1988;8(3):300–5.
39. Kaplan EB. Dorsal dislocation of the metacarpophalangeal joint of the index finger. J Bone Joint Surg Am. 1957;39-A(5):1081–6.
40. Bohart PG, Gelberman RH, Vandell RF, Salamon PB. Complex dislocations of the metacarpophalangeal joint. Clin Orthop Relat Res. 1982;164:208–10.
41. Stener B. Displacement of the ruptured ulnar collateral ligament of the metacarpo-phalangeal joint of the thumb. J Bone Joint Surg Br. 1962;44-B(4):869–79.

42. Kozin SH. Fractures and dislocations along the pediatric thumb ray. Hand Clin. 2006;22(1):19–29.
43. Graham TJ, Hastings 2nd H. Carpal injuries in children. In: Gupta A, Kay S, Scheker L, editors. The growing hand. London: Mosby; 2000. p. 583–90.
44. Terry CL, Waters PM. Triangular fibrocartilage injuries in pediatric and adolescent patients. J Hand Surg Am. 1998;23(4):626–34.
45. Tang JB, Zhang Y, Cao Y, Xie RG. Core suture purchase affects strength of tendon repairs. J Hand Surg Am. 2005;30(6):1262–6.
46. Navali AM, Rouhani A. Zone 2 flexor tendon repair in young children: a comparative study of four-strand versus two-strand repair. J Hand Surg Eur Vol. 2008; 33(4):424–9.
47. Lalonde DH. An evidence-based approach to flexor tendon laceration repair. Plast Reconstr Surg. 2011; 127(2):885–90.
48. Favetto JM, Rosenthal AI, Shatford RA, Kleinert HE. Tendon injuries in children. In: Gupta A, Kay S, Scheker L, editors. The growing hand. London: Mosby; 2000. p. 609–27.
49. Kato H, Minami A, Suenaga N, Iwasaki N, Kimura T. Long-term results after primary repairs of zone 2 flexor tendon lacerations in children younger than age 6 years. J Pediatr Orthop. 2002;22(6):732–5.
50. Fitoussi F, Lebellec Y, Frajman JM, Pennecot GF. Flexor tendon injuries in children: factors influencing prognosis. J Pediatr Orthop. 1999;19(6):818–21.
51. Thien TB, Becker JH, Theis JC. Rehabilitation after surgery for flexor tendon injuries in the hand. Cochrane Database Syst Rev. 2004;(4):CD003979.
52. O'Connell SJ, Moore MM, Strickland JW, Frazier GT, Dell PC. Results of zone I and zone II flexor tendon repairs in children. J Hand Surg Am. 1994;19(1): 48–52.
53. Havenhill TG, Birnie R. Pediatric flexor tendon injuries. Hand Clin. 2005;21(2):253–6.
54. Birnie RH, Idler RS. Flexor tenolysis in children. J Hand Surg Am. 1995;20(2):254–7.
55. Gilbert A, Masquelet A. Primary repair of flexor tendons in children. In: Tubiana R, editor. The hand. Philadelphia: Saunders; 1998. p. 359–63.
56. Cunningham MW, Yousif NJ, Matloub HS, Sanger JR, Gingrass RP, Valiulis JP. Retardation of finger growth after injury to the flexor tendons. J Hand Surg Am. 1985;10(1):115–7.
57. Gaisford JC, Fleegler EJ. Alterations in finger growth following flexor tendon injuries. A clinical and laboratory study. Plast Reconstr Surg. 1973;51(2):164–8.
58. Amadio PC. Staged flexor tendon reconstruction in children. Ann Chir Main Memb Super. 1992;11(3): 194–9.
59. Courvoisier A, Pradel P, Dautel G. Surgical outcome of one-stage and two-stage flexor tendon grafting in children. J Pediatr Orthop. 2009;29(7):792–6.
60. Fitoussi F, Badina A, Ilhareborde B, Morel E, Ear R, Pennecot GF. Extensor tendon injuries in children. J Pediatr Orthop. 2007;27(8):863–6.
61. Lundborg G. A 25-year perspective of peripheral nerve surgery: evolving neuroscientific concepts and clinical significance. J Hand Surg Am. 2000;25(3): 391–414.
62. Navarro X, Vivo M, Valero-Cabre A. Neural plasticity after peripheral nerve injury and regeneration. Prog Neurobiol. 2007;82(4):163–201.
63. Tajima T, Imai H. Results of median nerve repair in children. Microsurgery. 1989;10(2):145–6.
64. Onne L. Recovery of sensibility and sudomotor activity in the hand after nerve suture. Acta Chir Scand Suppl. 1962;(Suppl 300):1–69.
65. Lundborg G, Rosen B. Sensory relearning after nerve repair. Lancet. 2001;358(9284):809–10.
66. Seddon HJ. A classification of nerve injuries. Br Med J. 1942;2(4260):237–9.
67. Sunderland S. A classification of peripheral nerve injuries producing loss of function. Brain. 1951;74(4): 491–516.
68. Tagliafico A, Altafini L, Garello I, Marchetti A, Gennaro S, Martinoli C. Traumatic neuropathies: spectrum of imaging findings and postoperative assessment. Semin Musculoskelet Radiol. 2010;14(5):512–22.
69. Papazian O, Alfonso I, Yaylali I, Velez I, Jayakar P. Neurophysiological evaluation of children with traumatic radiculopathy, plexopathy, and peripheral neuropathy. Semin Pediatr Neurol. 2000;7(1):26–35.
70. Ma J, Novikov LN, Kellerth JO, Wiberg M. Early nerve repair after injury to the postganglionic plexus: an experimental study of sensory and motor neuronal survival in adult rats. Scand J Plast Reconstr Surg Hand Surg. 2003;37(1):1–9.
71. Chiu DT, Strauch B. A prospective clinical evaluation of autogenous vein grafts used as a nerve conduit for distal sensory nerve defects of 3 cm or less. Plast Reconstr Surg. 1990;86(5):928–34.
72. Fawcett JW, Keynes RJ. Muscle basal lamina: a new graft material for peripheral nerve repair. J Neurosurg. 1986;65(3):354–63.
73. Brunelli F, Spalvieri C, Rocchi L, Pivato G, Pajardi G. Reconstruction of the distal finger with partial second toe transfers by means of an exteriorised pedicle. J Hand Surg Eur Vol. 2008;33(4):457–61.
74. Mackinnon SE, Dellon AL. Clinical nerve reconstruction with a bioabsorbable polyglycolic acid tube. Plast Reconstr Surg. 1990;85(3):419–24.
75. Bushnell BD, McWilliams AD, Whitener GB, Messer TM. Early clinical experience with collagen nerve tubes in digital nerve repair. J Hand Surg Am. 2008; 33(7):1081–7.
76. Bertleff MJ, Meek MF, Nicolai JP. A prospective clinical evaluation of biodegradable neurolac nerve guides for sensory nerve repair in the hand. J Hand Surg Am. 2005;30(3):513–8.
77. Tung TH, Mackinnon SE. Nerve transfers: indications, techniques, and outcomes. J Hand Surg Am. 2010;35(2):332–41.
78. Viterbo F, Trindade JC, Hoshino K, Mazzoni A. Two end-to-side neurorrhaphies and nerve graft with

removal of the epineural sheath: experimental study in rats. Br J Plast Surg. 1994;47(2):75–80.
79. Artiaco S, Tos P, Conforti LG, Geuna S, Battiston B. Termino-lateral nerve suture in lesions of the digital nerves: clinical experience and literature review. J Hand Surg Eur Vol. 2010;35(2):109–14.
80. Whitehouse WM, Coran AG, Stanley JC, Kuhns LR, Weintraub WH, Fry WJ. Pediatric vascular trauma. Manifestations, management, and sequelae of extremity arterial injury in patients undergoing surgical treatment. Arch Surg. 1976;111(11): 1269–75.
81. Mangat KS, Martin AG, Bache CE. The 'pulseless pink' hand after supracondylar fracture of the humerus in children: the predictive value of nerve palsy. J Bone Joint Surg Br. 2009;91(11):1521–5.
82. Noaman HH. Microsurgical reconstruction of brachial artery injuries in displaced supracondylar fracture humerus in children. Microsurgery. 2006; 26(7):498–505.
83. Coombs CJ, Richardson PW, Dowling GJ, Johnstone BR, Monagle P. Brachial artery thrombosis in infants: an algorithm for limb salvage. Plast Reconstr Surg. 2006;117(5):1481–8.
84. del Pinal F. Severe mutilating injuries to the hand: guidelines for organizing the chaos. J Plast Reconstr Aesthet Surg. 2007;60(7):816–27.
85. Howe Jr HR, Poole Jr GV, Hansen KJ, Clark T, Plonk GW, Koman LA, et al. Salvage of lower extremities following combined orthopedic and vascular trauma. A predictive salvage index. Am Surg. 1987;53(4): 205–8.
86. Campbell DA, Kay SP. The hand injury severity scoring system. J Hand Surg Br. 1996;21(3):295–8.
87. Grob M, Josty IC, Soldin MG, Dickson WA. Paediatric friction hand injuries caused by domestic vacuum cleaners-a review from one unit. Burns. 2003;29(7): 714–6.
88. Meyer TM. Psychological aspects of mutilating hand injuries. Hand Clin. 2003;19(1):41–9.

Acute Thumb and Finger Instability

15

Sharifah Ahmad Roohi and Caroline Leclercq

Keywords
Capsular injuries • CMC dislocations • Collateral ligament tear • Digital dislocations • DIP dislocations • Hand dislocations • Instability • Ligamentous injuries • Mallet finger • MCP dislocations • PIP dislocations • Sagittal band disruptions • Sports hand injuries • Stener lesion • Stiffness (digital) • UCL tear • Volar plate injury

General Introduction

Instability is defined as a loss of equilibrium attributable to an unstable situation in which some forces outweigh others, which if applied to orthopaedics simply means an inability to maintain a reduced joint [1]. In the hand alone there are 19 joints which help to perform a myriad of intricate movements all balanced by opposing forces. Instability results from a disruption of this balance. An acute disruption is most commonly due to physical injury be it to the **bone** (Chap. 5) or **soft tissue** (ligaments, tendons) but may also present due to a **nerve** dysfunction causing loss of flexor (anterior interosseous) or extensor (posterior interosseous) function and distal joint instability. While fractures are dramatic, commonly seen in the emergency room and certainly frequent in the hand surgeon's clinic, ligamentous and soft tissue injuries, unless obvious or anticipated, are frequently missed especially at the general practitioners' or in a busy emergency room. They inevitably present later as instability or inability to perform a certain task.

In a study of a range of musculoskeletal soft tissue injuries, where all acute fractures and dislocations were excluded from the study population, it was found that 33.9 % were hand tendon injuries [2]. In another study of injuries to American professional footballers, it was found that 6 % involved the hand. Of these, metacarpal fractures were the most common followed closely by proximal interphalangeal joint (PIPJ) dislocations, ulnar collateral ligament sprains and distal interphalangeal joint (DIPJ) dislocations [3].

This chapter will start from the distal most joint and move up proximally dealing with the anatomical uniqueness of the digital joints, the causes of instability (dorsal, volar or lateral),

S.A. Roohi, MD, FRCS, MCh Orth (✉)
Department of Orthopaedics, Faculty of Medicine and Health Sciences, Universiti Putra Malaysia, 43400 Serdang, Selangor, Malaysia
e-mail: roohihandsurgery@gmail.com

C. Leclercq, MD
Consultant, Institut de la main, 6, Square Jouvenet, 75016 Paris, France

I.A. Trail, A.N.M. Fleming (eds.), *Disorders of the Hand: Volume 1: Hand Injuries*,
DOI 10.1007/978-1-4471-6554-5_15, © Springer-Verlag London 2015

their presentation, diagnosis and differentiation with a review of the treatment options and complications thereof.

The Distal Digital Joint (DIPJ of the Fingers and IPJ of the Thumb)

Introduction

The fraction of distal joint motion although small compared to the entire arc of digital flexion, plays an important role functionally, for the radial three digits are responsible for precision pinch while the ulnar two fingers are part of the power grip. A number of clinical conditions disrupting this function of the distal joint may present to the clinician with instability, the most common of which is a mallet finger (or thumb). Others include damage to the collateral ligaments, a disruption of the flexor mechanism, or a complete dislocation of the joint (dorsal or volar). Each of these will have a specific presentation and differential diagnosis and management.

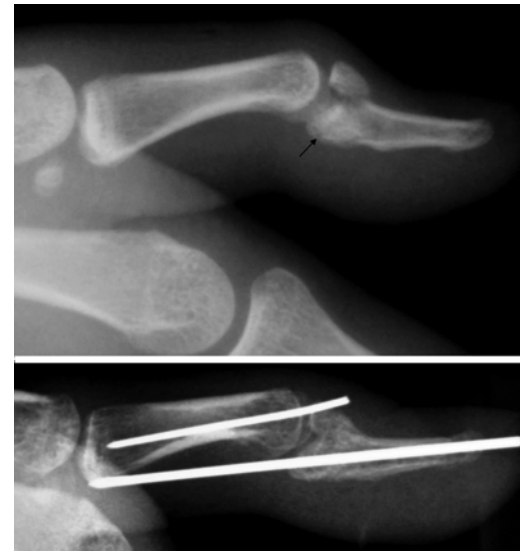


Fig. 15.1 In this 15 year old girl, the faint epiphyseal margin can be seen just distal to the IP joint (*arrow*). The fracture is fixed after a delay of 2 weeks by closed manipulation and insertion of a k-wire (*below*)

Anatomy and Clinical Implications

The Distal Interphalangeal Joint (DIPJ) of the Fingers

Bony Architecture

The DIPJ is a hinged ginglymus articulation formed by the concentric opposition of the concave base of the distal phalanx (P3) and the convex head of the middle phalanx (P2). An intercondylar depression divides the bicondylar head which has a slightly different arrangement from index to small fingers to accommodate the precise positioning requirements of each digit. In the index finger (IF), the radial condyle is slightly longer causing the finger to deviate ulnarly upon flexion. The condyles of the middle finger (MF) are aligned symmetrically and there is no rotation during flexion. In the ring (RF) and small (SF) fingers the arrangement of the condyles is reversed, the ulnar being longer to allow radial deviation in flexion facilitating rotational tip to tip contact with the thumb [4]. An elevated antero-posterior ridge on the base of the P3 extends dorsally like a hood over the P2 intercondylar groove which has a complementary extended engaging articular surface enabling a passive hyperextension of approximately 45°. The thin extensor tendon occupies a small attachment on this hood merging with the flimsy capsule that encloses the joint dorsally. Of interest is that this prominence in a skeletally immature child houses the epiphysis of the bone – which if injured may result in growth disturbance (Fig. 15.1).

Joint Capsule

The capsule holding the joint together is a complex structure and is strengthened by contributions from both ligaments and tendon attachments which serve to stabilise the joint, on all sides. **Laterally**, reinforcement is provided by the *collateral ligaments* which are thick bands arising from the head of P2, running in a distal and volar direction to insert into the laterovolar tubercle of the base of the distal phalanx [5]. *Accessory collateral ligaments* arise slightly more volar from the P2 head and insert distally to the sides of the palmar plate. The *lateral bands* join to form the terminal extensor tendon and provide secondary support. **Volarly**,

the *palmar plate* is a thick fibro-cartilaginous structure providing volar stability to the joint and a smooth floor for the **Flexor Digitorum Profundus** (**FDP**) **tendon** to glide on. Distally it merges with the FDP fibres and inserts into the periosteal surface of P3. Proximally, the volar plate is loosely attached to the neck of the middle phalanx in a swallow-tail fashion, although there are no "check rein" ligaments and a lack of firm bony attachment. Whilst this is controversial, clinical evidence would support the latter as in most cases the volar plate detaches proximally [6]. Due to this less rigid proximal fixation, hyperextension of the joint is possible and may result in less dorsal dislocation than its proximal counterparts [6]. This may also be due to the enhanced stability provided by the tendinous insertions and the shorter lever arm of the distal phalanx holding the joint tight. When dislocations do occur, due to the comparatively stronger volar component they are usually dorsal or lateral.

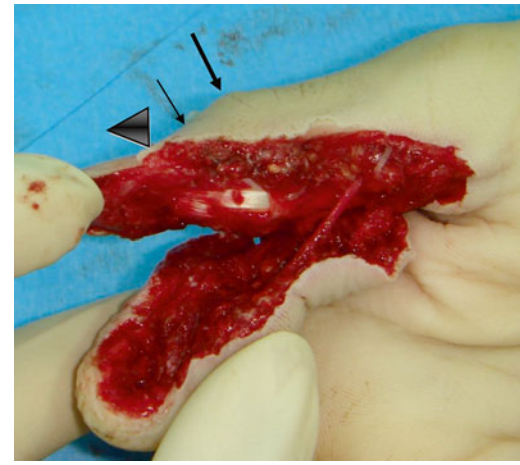


Fig. 15.2 Twenty six year old chef was involved in a roller machine accident at work when the whole pulp of the thumb fortunately still supplied by the radial digital artery, was avulsed. The DIP joint is shown by the *bold arrow*, the extensor pollicis longus distally up to the *small arrow* and the flexor pollicis longus is seen attached to the distal phalanx right up to the level of the *triangle*

The Thumb Inter Phalangeal Joint (IPJ)

Bony Architecture

The thumb IPJ is also a hinge joint and anatomically similar to the DIPJ, although the articular surfaces are held together tightly by a strong capsule and ligaments that allow minimal lateral motion. The primary motion arc is flexion and extension, however, because the ulnar condyle of the proximal phalanx protrudes more distally and volarwards than its radial counterpart, between 5° and 10° pronation occurs on thumb flexion, permitting pinch opposition with the fingers [7]. The extensor tendon inserts into a dorsal crest on the distal phalanx just distal to its articular surface while the thick flexor pollicis longus (FPL) tendon creates a depression on the volar surface for its insertion [8]. The broader firm insertion of the FPL is slightly more distal than the extensor (Fig. 15.2) giving it stability and more force in flexion than in extension. Of course, severance of either one results in a deforming force and significant loss of function.

Joint Capsule

Laterally, the capsule is reinforced by two *collateral ligaments (CL)* and their extensions, the *accessory collateral ligaments* inserting in a similar fashion as at the DIP joints. The *Volar Plate* sometimes houses a sesamoid bone which may resemble a chip fracture on radiographs. The thin *Dorsal Capsule* is reinforced by the *extensor pollicis longus (EPL)* tendon [8].

The Mallet Finger

Epidemiology

The mallet finger is a common entity occurring mostly at work or during sports due to a sudden flexion force (which may even be trivial) applied to the extended finger [9]. Most commonly affected are the long, ring and small fingers of the dominant hand, particularly in young to middle-aged males [10]. In women, it tends to occur in an older age group [11]. Jones & Peterson proposed a genetic predisposition when they reported 20 mallet fingers in 7 members of a 3-generation family; 85 % of which occurred spontaneously or with minimal trauma [12]. Of interest is the microvascular study performed by Warren et al. in which they alluded to a "critical zone" 11–16 mm from the terminal

insertion site of the lateral bands corresponding to the area that is compressed against the head of the P2 on full DIP flexion [13]. This vascularly compromised segment may be predisposed to rupture with minor trauma (eg. in the elderly) or affected adversely by ill-fitting dorsal splints.

Clinical Features and Diagnosis

A mallet finger is a clinical diagnosis presenting with an obvious flexion or 'lag' of the DIP joint, usually caused by the forced flexion of an extended finger as in a ball hitting a finger end-on. It presents acutely but may have a delayed presentation [9, 11]. This delay may be explained by an initial spurious extension made possible by the tenodesis effect exerted by the still intact oblique retinacular fibres activated via the flexor system, which in time fatigues [14]. The cause may be a soft tissue discontinuity of the extensor mechanism or a bony avulsion (one-third of mallets) which can be seen on plain x-ray (Fig. 15.1) [15]. Doyle divided mallets into four types (Table 15.1) [16]. The four main symptoms upon consultation are deformity (100 %), pain (56 %), hindrance of ADLs (32 %) and incapacity at work (17 %) [9]. The features to look out for are *tenderness on the dorsum, loss of passive extension* of the DIPJ (possible bony block), *ligament laxity* which can cause swan neck deformity and *deviation of the joint in the coronal or sagittal plane* delineating associated collateral ligament damage and joint subluxation. The range of motion should be recorded. If pain does not permit this then a digital block may help in the acute setting to complete the examination.

Table 15.1 Doyle's classification of mallet finger injuries

Type	Description
I	Closed injury +/− small dorsal avulsion fracture
II	Open injury (laceration)
III	Open injury (deep abrasion involving skin, soft tissues and tendon)
IV	Mallet fracture
A	Distal phalanx physeal injury
B	Fracture fragment is 20–50 % of articular surface
C	Fracture fragment >50 % of articular surface

Radiographs should be obtained in both AP and lateral views to help in assessing the extent of damage and joint subluxation. Oblique views enable detection of small flecks of avulsed fragments of bone [17]. The advent of the mini C-arm has allowed real-time fluoroscopy to detect such small flecks as well as accurately assess instability.

Treatment Options

Traditionally mallet fingers have been treated with various types of splint, both volar and dorsal (Fig. 15.3), although various operative techniques, both minimally invasive and open (open reduction, internal fixation – ORIF) have also been advocated and will be discussed.

A soft tissue lesion or even one with a minor bone fleck responds well to splinting which should be carried out for a minimum of 6–8 weeks continuously; the extended position should not be 'dropped' even for an instant. The ideal position is one of slight hyperextension. It is important, however, any skin maceration is detected early. After this period, the DIPJ is tested for stability and ROM exercises are commenced – we usually advocate 10° per week – with a further period of 2–4 weeks of night splinting. Final assessment is at 12 weeks post splinting.

Surgical Treatment

Surgery is indicated in three situations – failed conservative treatment, individuals who are not able to comply with splinting or those who would have difficulty performing their jobs in a splint (such as doctors, musicians etc.). There is a plethora for techniques in managing this injury; however in these simple cases a simple transarticular pin is sufficient. In those with a significant fracture fragment, we prefer surgical treatment, although it is important to avoid stripping the insertion of the collateral ligament off the fracture fragment [17].

Outcome and Lit Review

Those who do well with splinting are usually in the younger age group, seek treatment early and

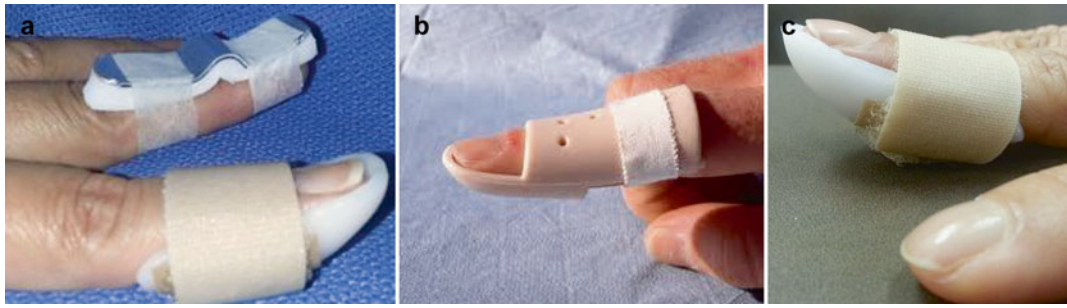


Fig. 15.3 The various types of mallet splints: (**a**) dorsal Zimmer as described by Kleinert (**b**) Stack and (**c**) mallet. **Instructions to patients**: The thermoplastic splint can be washed in cold water with soap and towel-dried. Discontinue use if rashes develop. Please loosen the strap if finger turns bluish or feels tight

have only a mild to moderate extensor lag [9]. McFarlane and Hampole believed however that good results (<15° extensor lag) may even be achieved 3 months out from the injury, although the patient must be compliant with the splinting [18]. The type of splint used does not seem to matter [19].

Complications

In the publication with the highest complication rates, Stern & Kastrup give a detailed review of 123 mallet fingers, noting complications from splinting (45 %) are short-term whereas surgical complications (53 %) took a longer time to settle (38 months) [20]. Splinting may result in maceration, ulceration and tape allergy, which can be minimised with early and frequent follow-up. Post-operative problems included nail deformity, joint incongruity and infections. Seven required re-operation and 4 ended up with either an arthrodesis (2), amputation (1) or a more severe mallet deformity (1). Reconstructive operations for chronic ligamentous instability will be considered in Chap. B4. Chronic Instability of the fingers & thumb by Carlos Herus Palou.

Clinical Pearl

For closed injuries, we always offer a 6–8 week period of uninterrupted immobilisation with great care in explaining what that means.

For open injuries, we usually repair the tendon, undertake a transarticular fixation with an oblique 1.0 mm Kirschner wire buried under the skin, Tubiana style and protect it with an external splint [21].

To quote a succinct summary, "patients should be informed of the potential for a residual DIP extensor lag and swan neck deformity with all methods of treatment" [22].

The Mallet Thumb

Epidemiology

As compared to mallet finger, mallet thumb is a less common condition. In describing this entity, Din and Meggitt treated 48 mallet fingers as opposed to 4 mallet thumbs over a period of four years [23]. The authors defined a mallet as one in which the site of injury is distal to the **MCPJ** of the thumb resulting in loss of only IP extension as opposed to a 'dropped' thumb in which the lesion is proximal to the MCPJ and has loss of both IP and MCP extension.

Clinical Features and Diagnosis

Mallet thumb presents in pretty much the same way with an inability to actively extend the IP joint. There may be pain and tenderness with some

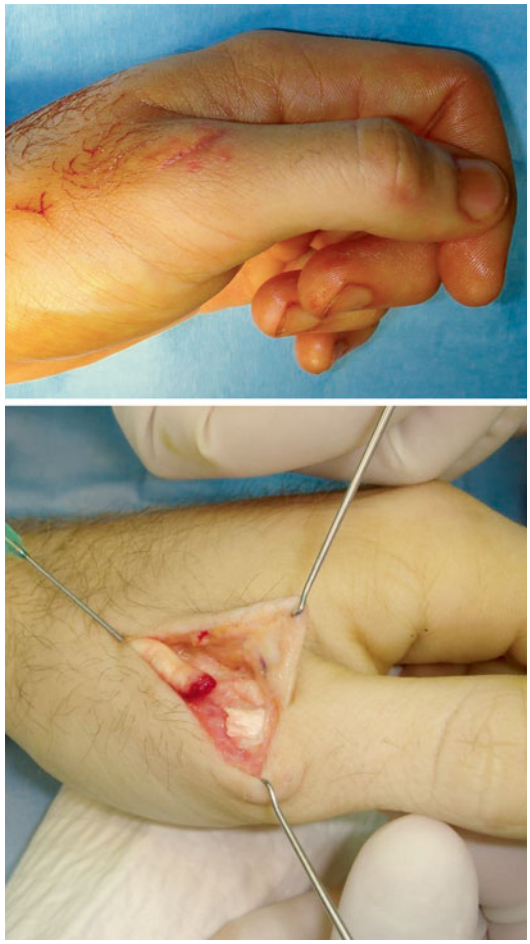


Fig. 15.4 The EPL has been cut just distal to the MCP joint of the thumb (healed scar on *left*). This went undiagnosed and was referred 3 weeks later. The proximal end had retracted >5 cm away

bruising. Radiological examination may or may not reveal a fracture of the distal phalanx or rarely, of the proximal phalanx [23]. The difference to note is the longer distance for potential injury; that is from the insertion site to the MCP joint of the thumb (the length of the proximal phalanx) (Fig. 15.4) as opposed to just the 1 cm or so around the DIPJ in the fingers. An MRI or ultrasound scan may be useful to identify and quantify any gap between the proximal and distal ends of the EPL [24].

Treatment Options

Initially, surgical treatment was the advocated option, however more recently, numerous articles

Fig. 15.5 There are a number of looped non-absorbable sutures available in the market. We prefer the Supramid with outer nylon coating and a reverse cutting needle (making it smooth and easy to go through the tendon with minimal damage)

have advised conservative treatment – even in open injuries – with the thumb (IPJ) held in extension for a period of 6–8 weeks followed by night splinting from anywhere between 2 and 6 weeks. We tend to favour non-operative treatment in younger patients who have closed injuries, who present early (less than 2 weeks' history), with mainly soft tissue component. Splinting should be carried out with the IP in full extension.

Surgical Treatment

In open injuries, those with a delayed presentation of more than 2 weeks and in older patients especially those where 2 months of splinting is not an option – surgery is the first choice. For injuries close to the insertion, closed reduction and pinning is preferable. More proximally the EPL tendon is more rounded and able to accept an intratendinous suture. We prefer a modified Kessler or 4-strand core repair using a non-absorbable looped suture (Fig. 15.5). Ideally, this should allow early active supervised mobilization. The repair is otherwise protected in a splint at all times for 6 weeks, after which night splinting is advised followed by strengthening exercises.

Outcome and Lit Review

In the largest series of thumb mallets Miura et al. used splinting in 25 and surgical treatment in 10. 16 % of their patients treated conservatively

could not extend to 0° whereas all surgical ones could. They concluded that surgical repair is the preferred option, although conservative treatment must be offered in selected cases such as following a closed soft tissue injury, on early presentation or in a younger patient (<30 years) [25]. Of the other 7 articles, 4 described good results with splinting whereas 2 advocated surgery. The remaining article had one case of each.

Complications

Lack of full extension, failed treatment and prolonged deformity are sometimes seen. In bony mallets, arthritis and inclusion cysts may occur.

Clinical Pearl

We feel a mallet thumb should be treated slightly differently from a mallet finger for persistent deformity is unacceptable in a thumb. In our practice, we have treated three mallet thumbs in the last five years as compared to over 40 mallet fingers. Most of our patients presented late (>2 weeks) or with failure of conservative treatment. Thus in all 3 cases, we treated them surgically either with closed pinning (Fig. 15.1) or open repair with good results. Surgical repair is indicated in open injuries, those where there is delay resulting in the EPL ends being retracted and more proximal injuries.

Collateral Ligament Injury

Clinical Features, Diagnosis and Treatment

Isolated collateral ligament injuries of the DIP or thumb IP joint are not as common as their proximal counterpart. The classical presentation would be pain and swelling on the lateral aspect of the digit. There might be some lateral deviation (Fig. 15.6) and tenderness on pressure at either insertion point. As in all trauma, concomitant volar plate tears

Fig. 15.6 (**a**) Dominant left thumb of an engineer after he traumatised it twice. Obvious injury to the ulnar collateral ligament with radial deviation of distal phalanx and damage to germinal matrix. He also had a malunited fracture but was able to flex 0–45°. What was troubling him was instability on pinch grip. (**b**) Post-op appearance after reconstruction with a small osteochondral bone graft and bone anchoring of UCL ligament. Deviation is corrected and the thumb was stable but ROM improved only slightly. The germinal matrix was simultaneously grafted

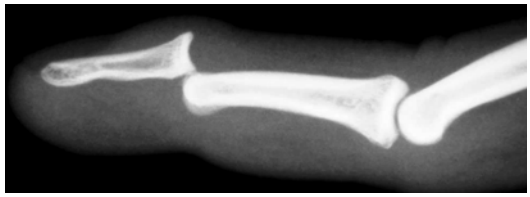


Fig. 15.7 Closed dorsal dislocation of the DIP joint. This is reduced by first hyperextending the joint, applying traction then reducing it. Volar plate interposition may require open reduction

should be identified in the form of palmar tenderness. Two radiographic views are essential and may show a small fleck of bony avulsion.

Almost all collateral ligament injuries are amenable to splinting, if diagnosed early, with good results. We find a radial gutter for ulnar collateral ligament injuries and vice versa very useful. Associated injuries are dealt with below.

There is very little written on collateral ligament injuries of the distal joint let alone the treatment outcomes.

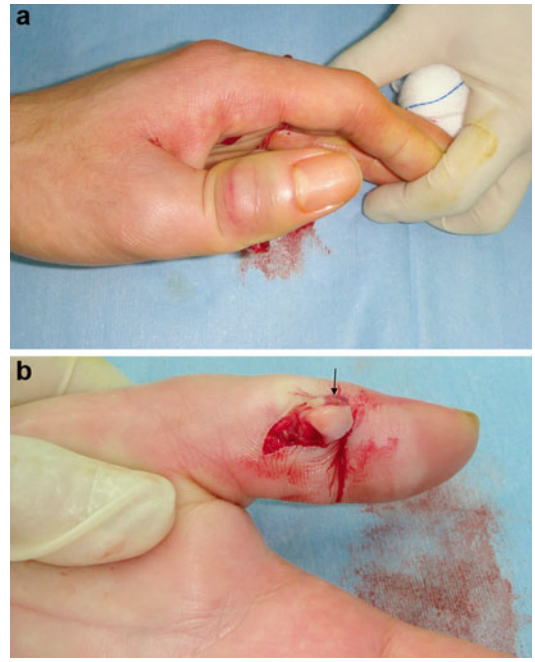


Fig. 15.8 (**a**) Gross deformity of the left dominant thumb with a reduced 2 point discrimination distally on the radial side. Circulation intact but areas of the thumb slightly pale. (**b**) The radial digital nerve (*arrow*) is stretched over the radial condylar head of the proximal phalanx

Joint Dislocation: Dorsal

Epidemiology

Dislocations are "not uncommon", are usually dorsal (Fig. 15.7) and open due to the thinner dorsal envelop and unyielding attachments of skin and subcutaneous tissue to the underlying distal phalanx which split open when forces are exerted [26].

Clinical Features and Diagnosis

Pain, deformity and an open wound are the usual hallmarks of presentation. Handedness, occupation and the mechanism of injury are important initial questions to be answered especially the latter to determine the amount of force involved. Assessment should first be carried out on the wound itself, followed by a neurovascular examination, including 2 point discrimination. Sometimes the digital nerve can be stretched. Radiological views AP, lateral and oblique are usually sufficient. Under a local block flexion and extension can be assessed.

Treatment Options

For closed dislocations reduction may be attempted under digital or regional block anaesthesia. However, open injuries should receive appropriate surgical wound care and we usually take the patient to theatre for a thorough assessment and débridement. Sometimes the digital nerve is stretched over the protruding phalanx (Fig. 15.8) or there may be an associated unicondylar fracture which may need fixation [27]. After reduction, the integrity of the collaterals and volar plate is assessed and the joint pinned if unstable. Otherwise, a dorsal extension block splint is applied in 10° of flexion.

Joint Dislocation: Volar

These are rare injuries and occur when there is a dorsal lip fracture involving more than 50 % of the articular surface resulting in

volar joint subluxation. They will be dealt with in Chap. 5.

The Proximal Interphalangeal Joint: (PIPJ)

Introduction

The PIP joint is different in both form and function from the other two finger joints. Whilst the metacarpophalangeal joint (MCP) joint is utilised for global positioning and the distal interphalangeal joint (DIP) for precision, the PIP is the pivot which most influences function. As a consequence, it not only has to provide lateral and rotational stability, much like the MCP of the thumb, but also have a wide range of flexion-extension. However, due to its exposed location and relative 'stiffness' it is prone to injury and is in fact the most commonly dislocated joint in the hand [28].

Injury to the PIPJ of the long finger, although the commonest is the one least understood and prone to mismanagement and thus complications. Benke and Stapleforth in a retrospective review of 96 PIP joint injuries found a poor outcome in 30 % (almost half of which had open injuries), as characterized by pain, poor function, joint instability, or stiffness in the form of flexion deformities [29].

The first pitfall occurs as it is commonly assumed to be a 'minor' injury by the patient (usually an athlete), the coach or even the primary physician and either not treated medically or not examined adequately at initial presentation. The second pitfall happens when the family physician or orthopaedic surgeon does not realise the potential complexity of the injury and either immobilises the finger for a prolonged period or operates on it inappropriately causing stiffness in both situations. Early and appropriate management of these injuries is key to preventing chronic pain, stiffness, deformity, or premature degenerative arthritis.

Understanding the anatomy, biomechanics and recovery patterns forms the basis of treating these injuries with minimal adverse effects and maximal functional outcome.

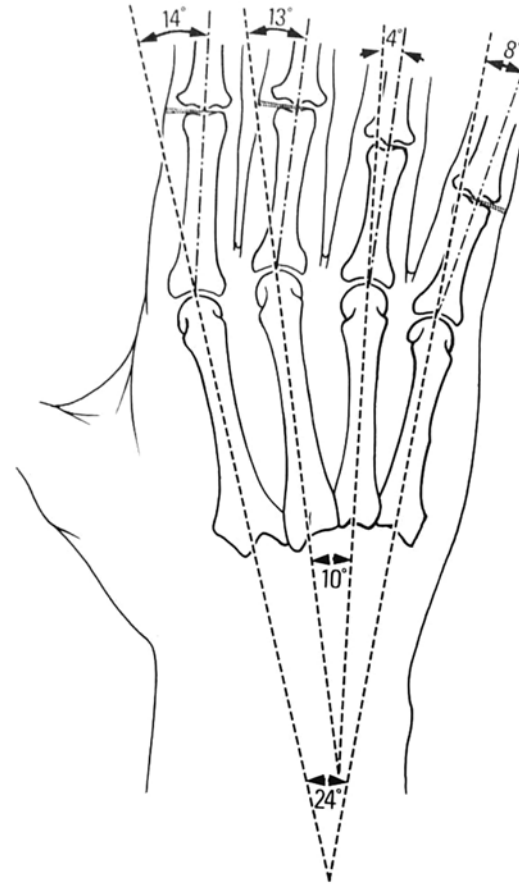


Fig. 15.9 The placement of the metacarpals and phalanges of the fingers in relation to the hand. At the PIP joint alone is there no sagittal angulation but transaxial rotation (see next figure) (Taken with permission from Dubousset [30])

Anatomy and Clinical Implications

Bony Architecture and Angles in the Hand

The longitudinal axes of the 2nd to 4th metacarpals are radially spaced at 24° with the proximal phalanges at a slight ulnar deviation (4–14°) whilst the middle phalanges maintain the longitudinal alignment (Fig. 15.9) [30]. The transaxial tilt of the condyles of the P1 heads are towards the ring finger resulting in supination at the PIPJ of the index and middle fingers and pronation of the small finger [31] which all then converge pointing towards the scaphoid tubercle when the

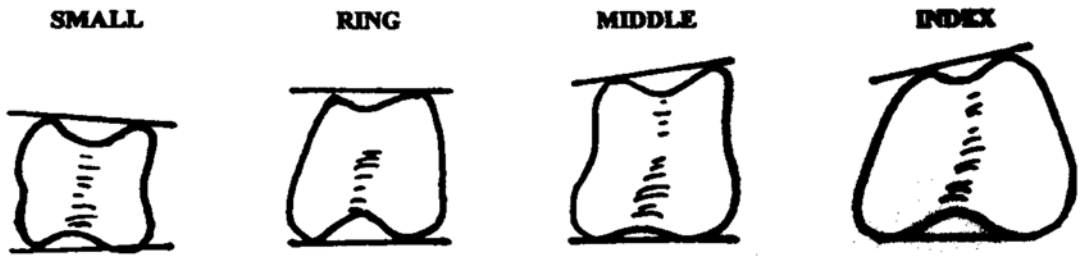


Fig. 15.10 In the transaxial plane, the condyles of the fingers are all angled towards the ring finger which imparts a rotational component to motion at the PIP joint (supination for index and middle and pronation for the small fingers) (Taken with permission from Kuczynski [91])

hand is clenched [30]. This assists the clinician in determining the correct alignment after primary or reconstructive surgery (Fig. 15.10) [32]. In all, these intricate differences allow varying degrees of fine precision and opposition movements to achieve fluidity of form and function.

The Proximal Interphalangeal Joint (PIPJ) of the Fingers

The PIP joint is a box like structure held together tightly by the capsule which is strengthened by the extensor expansion dorsally, the collateral and accessory collateral ligaments laterally and the volar plate palmarly. This boxing permits movement in the sagittal plane only: ie flexion and extension while other degrees of freedom – in the coronal (radial/ulnar deviation) and transaxial (pronation/supination) planes – are limited [31]. It is this restriction we believe that makes this joint particularly susceptible to injury.

Bony Architecture

The PIPJ is a hinged ginglymus articulation formed by the concentric opposition of the concave base of the middle phalanx (P2) and the convex head of the proximal phalanx (P1). The bicondylar head of the P1 is shaped like a trapezoidal trochlea, divided by a shallow anteroposterior intercondylar groove ($\leq$1 mm) that has a congruent elevation on the middle phalanx base into which it fits. The condyles have a near perfect radius of curvature – thus differing from the metacarpal heads – with a fixed transverse axis in all positions of flexion-extension. As a consequence little stretching of the collateral ligament complex occurs [30].

The articular surface of the proximal phalanx extends further over the volar surface (than the dorsal) including the retro-condylar area, which articulates with the volar lip of the base of the middle phalanx in full flexion, pushing the volar plate proximally. This permits the PIP joint's wide arc of motion from 0° to 110°, although some may have hyperextension and flexion to give an increased range of −30° to 120°. Lateral stress of some amount is possible (7–8°) in midrange of motion when the lateral capsular structures are under least tension [6]. This is possible due to incomplete congruency of the joint leading to a degree of "sloppiness" [31].

The middle phalanx (P2) is as wide as the P1 in the transverse diameter; however it is only half of the P1 in its AP diameter. Dorsally, it has a central tubercle to which the central slip of the extensor tendon attaches. Laterally the bone surfaces angle upwards at 60° on either side towards the lateral tubercle which is a roughened surface for the attachment of the proper collateral ligament. Volarly the palmar plate gains a firm attachment thereby increasing the glenoid cavity and articular area [33].

Joint Capsule

This complex structure is strengthened by contributions from both ligamentous and tendinous attachments which stabilise the joint, on all four sides.

Dorsally, the capsule is thin, pliable and doubles in length during its ROM. It is inseparable from the central slip of the extensor tendon which protects the PIP joint while allowing a wide range of flexion. The latter is joined by two lateral slips

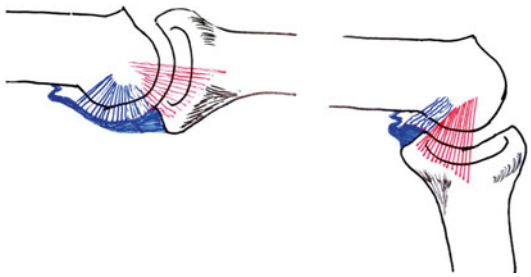


Fig. 15.11 The VP (in *solid blue*) is smoothed over the volar surface of the neck of P1 in extension forming a recess between it and the head. The Collateral ligament is seen here in *red lines* while the accessory collateral ligament (which is taut in extension) is depicted in *blue lines*. As the P2 flexes, the flimsy VP sheath moves out of the way and the volar lip fits snugly into this recess

on either side which merge into its fibres preventing their volar subluxation as occurs in a boutonnière deformity. More detailed anatomy is described in the extensor tendon chapter.

Laterally, reinforcement is provided by the *collateral ligaments* which are 2–3 mm thick bands arising from a lateral fossa on the head of P1, running in a distal direction parallel to P2, to insert into the laterovolar tubercle at the base of the middle phalanx [34]. Due to the fixed transverse axis, the length of these doesn't change in flexion-extension. *Accessory collateral ligaments* arise slightly more volar from the P1 head and insert distally into the sides of the palmar plate.

Volarly, the *palmar plate* (VP) is a thick fibrocartilaginous structure which has strong distal palmar insertions on the lateral aspects of P2 while the middle part is more lax. Proximally the central portion remains flimsy and membranous but is attached firmly in a u-shape formed by the two "*check rein*" ligaments on the lateral ridges on either side of the volar surface. This forms a deep (10–12 mm) palmar cul-de-sac: known as the *pretrochlear* bursa (between the proximal part of the VP and the head of the P1) which houses the volar lip of P2 as the flimsy VP is pulled away in full flexion of the PIPJ [30] (Fig. 15.11). It is bordered proximally by the A2 annular pulley and lies beneath the C1 cruciate pulley. The whole volar complex of VP, flexor tendons and their sheaths when healthy and acting together, prevent hyperextension.

Injuries to the Pip Joint

The PIPJ plays a pivotal role in power grasp (ulnar 2 fingers) and precision pinch (radial 2 fingers) with the thumb. It has the widest range of motion of all the finger joints being responsible for 85 % of the motion for grasping [35]. When injured it tends to cause more stiffness than instability thus resulting in significant morbidity. Injuries to the PIP joint may be broadly classified as dislocations, avulsions or articular fractures of which only the first shall be discussed in this chapter [36]. Most dislocations are reducible and may be dorsal (most common), lateral or volar (least common) referring to the displaced position of the middle phalanx [36].

Dorsal Dislocations

Epidemiology and Pathomechanics

This is the most common dislocation in the hand and presents as a 'jammed' finger on the field. It particularly occurs in ball-hitting or catching sports such as baseball, volleyball, basketball and rugby. The athlete's finger is hit head-on by the ball or suffers a hyper-extension injury with or without rotation. This inevitably starts with a tear in the volar plate (starting distally) and as the force continues, includes the collaterals (in between the proper and accessory) and as the middle phalanx is extended further dorsally may cause a volar lip fracture of the latter as it hits the P1 condyles (Fig. 15.12). For a complete dislocation to occur, two of the structures must be torn.

Classification

Bower initially classified these VP injuries based on the extent of damage and their stability, although we prefer the modification proposed by Liss and Green [37].

Type I: First degree sprains, ligament bruised but VP intact

Type II: Second and third degree sprains: hyperextension with some tearing but no subluxation of joint, congruity maintained.

Type III: Hyperextension with joint incongruity (Fig. 15.12b).

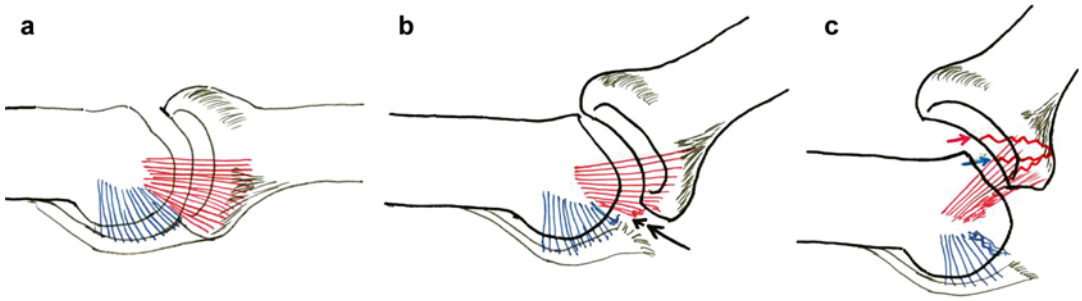


Fig. 15.12 (**a**) The PIP joint in extension – Collateral ligament in red and accessory collateral in blue. (**b**) As the joint is hyperextended, the VP tears distally away from the volar lip of P2 and the tear then splits between the proper and accessory collateral ligaments – as shown by the *black arrows*. (**c**) Further extension tears through in between the collaterals but the proper is usually still attached to the P1 head. Fractures may occur involving 30 % (*blue arrow* – stable) or 50 % (*red arrow* – unstable) of the articular surface which if in the former setting there is still some collateral attachment to the P2 base allowing the joint to maintain stability after reduction

Type IV: Dislocations and fracture-dislocations (Fig. 15.12c) may be compound and irreducible. Stable: those with articular involvement of ≤30 % and unstable: those with ≥50 % of the joint surface involved [33]. Those in between should be tested for stability post reduction. These are dealt with in Chap. 5 by Gray Giddins.

Clinical Features and Diagnosis

A detailed **history** must include the patient's age, occupation, handedness and mechanism of injury. The type of finger (long and slender vs. short and stubby) is also important for the latter heal well although they may become stiff, whilst the former often don't. If the finger is not obviously deformed, it is pertinent to ask whether any on-site reduction was performed.

During physical **examination** the degree of swelling, erythema and site of bruising should be noted. One should first check circulation (colour and capillary refill) followed by sensation (2-point discrimination). Next, points of tenderness in four areas (dorsal, volar, radial and ulnar) are assessed, especially the origins and insertions of the collaterals and VP. The central slip insertion is also palpated [38]. The finger is then put through an active range of motion (ROM) which is recorded. The patient is then asked to make a fist if possible and the rotational alignment is checked to make sure all the fingertips are pointing to the scaphoid and there is no overlap at the fingertips. We prefer to assess joint stability after radiological examination has been undertaken. In our setting a mini c-arm is useful and can be used simultaneously to reduce a subluxation or dislocation as well as examination time. If this is not available then radiographs should be taken in AP, lateral and oblique views for sometimes the volar fragment off the P2 may not be easily seen.

Treatment Options

Most injuries to the PIPJ may be treated in a closed manner. After reduction, splinting usually suffices. However, the subtle differences in the degree of injury will alter the duration and therapy regime. What is most important is patient education. Specifically and although the injury is mild, the PIP joint is notorious for becoming stiff and remaining swollen and that the key to recovery is motion. As a consequence, immobilisation should be kept to a maximum of 1 week in the milder cases and no more than 3 weeks in the more severe.

Type I and II Injuries

Although these are mild injuries, they should not be taken lightly. For type I injuries, dorsal splinting may be carried out in 20–30° of flexion and at 1 week gentle ROM exercises initiated. For type II injuries the duration of splinting is up to 4 weeks with progressive straightening of the dorsal block (Fig. 15.13). Alternatively buddy splinting may be used to allow earlier mobilisation.

At first follow-up it is essential to assess the integrity of the central slip by asking the patient to extend a (90°) flexed PIPJ against resistance [39]. Failure to extend at the PIPJ and fixed extension at the DIPJ reveals complete central slip disruption. Treatment is by immobilisation in as much extension as possible with close monitoring to prevent the development of a boutonniere deformity.

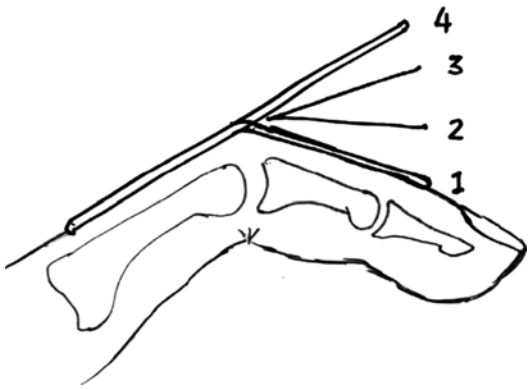


Fig. 15.13 A simple extension block splint using a padded aluminium dorsal splint or a custom made thermoplastic one for comfort may be used. Ten degree of extension per week (as numbered above) or every few days may be practiced

Type III Injuries

Joint subuxations can be reduced by closed means and if seen within the hour can be done under a digital or local block. The proximal phalanx is stabilised in one hand while applying pressure on the dorsal base of the middle phalanx. The other hand applies traction holding the finger while hyperextending the PIP joint. The P2 is pushed distally with volarly directed pressure. Reduction is quite easy and if it isn't, suggests soft tissue interposition. Radiographs are a must to ensure congruency (Fig. 15.14). Incongruency suggests failure of complete reduction. The reduction must then be tested for stability (Fig. 15.14b). The way to determine the degree of subsequent block is to note the degree of extension at which the reduction becomes unstable. A splint is then applied at this angle. Usually 10° of leeway is given each week.

Splinting, buddy taping and RICE (rest, ice, compression and elevation) are all part of the rehabilitation process. However, no more than 40° of flexion should be used as a position for immobilisation as this can result in a subsequent flexion contracture developing. Again, patient education with regard to persistent swelling as well as immobilisation, helps with the recovery.

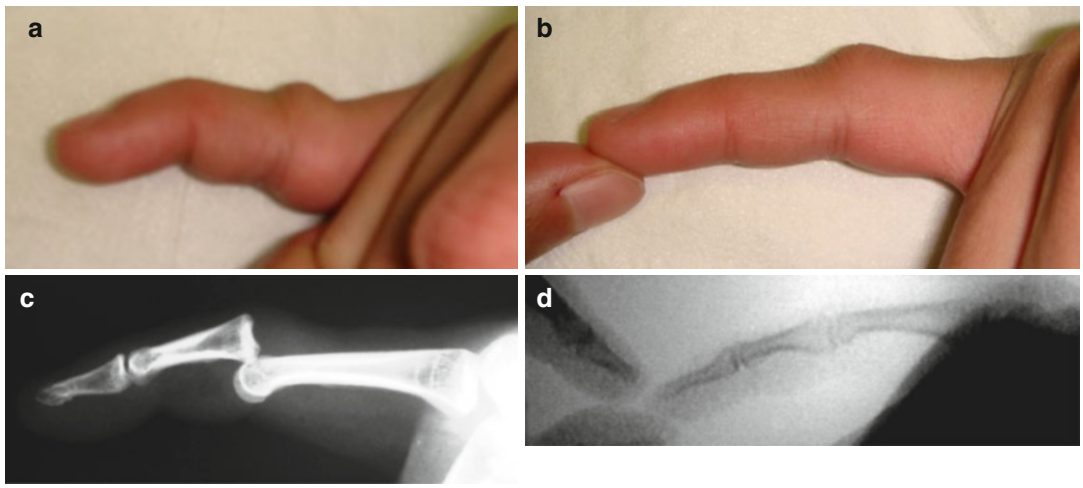


Fig. 15.14 (**a**) A 42 year old professional cricketer presented acutely with a swollen finger. (**b**) Marked difference can be seen in the clinical picture pre-reduction (**a**) and post-reduction (**b**). As explained in Fig. 15.12c, the proper collateral ligament (dorsal portion) usually remains intact, attached to the base of the P2 so after reduction, the joint is stable even when tested (**b**). (**c**) Bayonet position. (**d**) The joint is congruent post reduction

Surgical Treatment

Open injuries of course require formal lavage and meticulous debridement under magnification. Otherwise most dorsal dislocations (other than unstable fracture dislocations) do not need surgery. In chronic cases central slip deficiency may require repair or reconstruction.

Complications

Two main complications may occur from dorsal dislocations. Persistent hyperextension may be due to an unhealed volar plate. The patient typically presents with a "swan neck deformity" and snapping of the lateral bands or the inability to initiate flexion. Sometimes it simply requires a re-attachment of the volar plate. In others it may need reconstruction.

The other issue is a "pseudo-boutonniere deformity" where the PIPJ is flexed but the DIPJ is not extended. Again the cause is the volar plate but this time an undetected **proximal** avulsion. The flexion contracture needs to be released in the usual manner and splinted in extension for 3–4 weeks.

Outcomes, Personal Views and Conclusions

Literature shows more stiff than unstable PIP joints, the conclusion being that they have been over-treated rather than under-treated [36]. Thus it is more prudent to err on the side of mobility! Another interesting comment by Freiberg is "the shorter the digit and the older the patient, the less immobilisation is required". He found that the most commonly injured PIPJ is that of the small finger, and often results in the worst outcome. Their practice was to aim for a return of flexion (over extension) in the ulnar fingers and extension (over flexion) in the radial [38]. An inability to flex the little finger, is likely to be due to adhesions of the proximal portion of the volar plate to the base of the neck of the proximal phalanx, preventing the proximal phalanx access to the recess (Fig. 15.15). However, more recently ultrasound guided needle adhesiolysis has been shown to be helpful and may preclude the need for open surgery.

Clinical Pearl

The key to managing these simple yet challenging injuries is as in all hand injuries: early detection, appropriate management and the prevention of complications. A keen eye will pick up any failure, allowing the early implementation of alternate treatment saving the patient much anguish and hopefully the return of good function.

Collateral Ligament Ruptures

Epidemiology and Pathomechanics

Lateral collateral ligament ruptures of the PIPJ are a fairly common occurrence especially in athletes [40]. In an analysis of 18 cases Redler found the radial collateral of the border digits to be more commonly affected [41] (Fig. 15.16). For significant lateral instability to exist however, a tear in both the lateral collateral and volar plate must occur [40]. The disruption probably starts at the proximal phalanx and progresses through between the collateral and the accessory to finally tear at the volar attachment on the proximal phalanx [28]. Although this seems like a major disruption, most will heal as protected joint motion is commenced.

Clinical Features and Diagnosis

A detailed history and examination is a must. The joint is assessed by way of swelling, tenderness, range of motion and lateral stability. X-rays are also useful, particularly joint congruity [7]. It is important, however, that a true lateral is undertaken to identify subtle subluxation and/or (avulsion) chip fractures.

Treatment Options

Splinting in 10–20° of flexion is the mainstay of treatment especially if the joint is congruent and stable in motion. Adjunct ultrasound will soothe the digit and assists in healing. Buddy taping to the adjacent digit is an option but difficult in the small finger. Coban may be used to control swelling but should be used with caution.

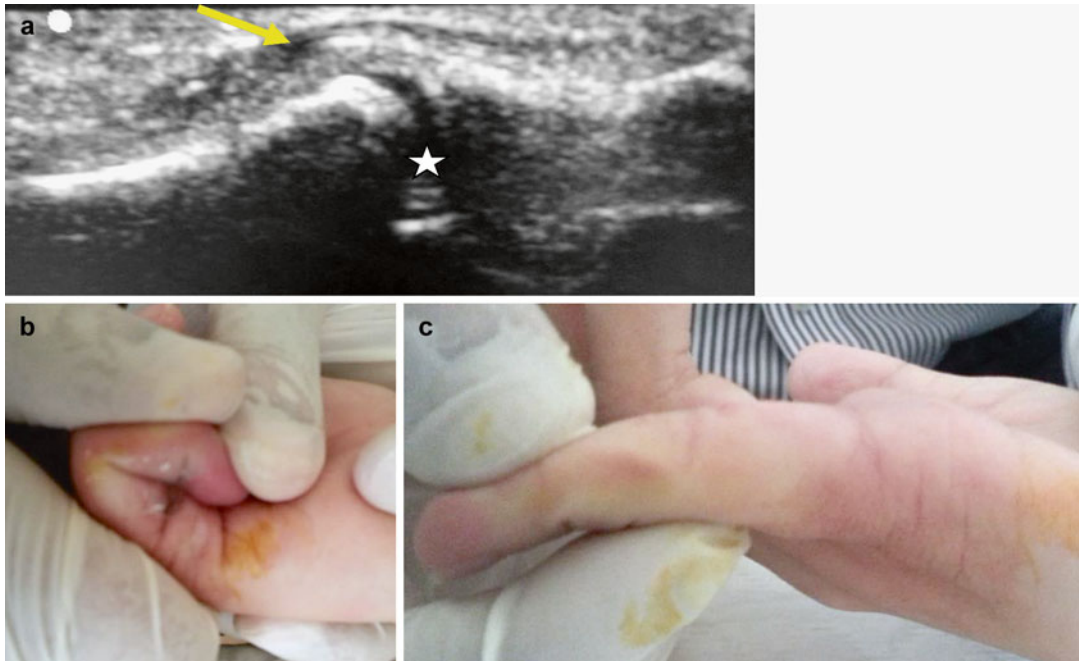


Fig. 15.15 Full flexion was markedly limited in a 9 year old's PIP joint after fixation of a malunited P1 neck fracture. (**a**) The US picture shows post-PRP injection (*white circle*) and needle adhesiolysis with the proximal part of the VP (*arrow*) now moving over the PIPJ (*star*). (**b**, **c**) Full flexion and extension was achieved

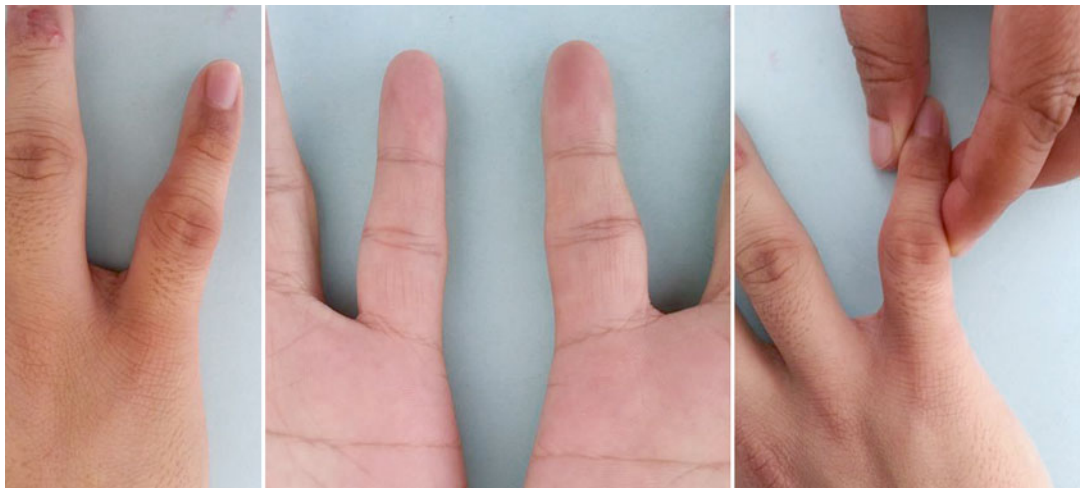


Fig. 15.16 A 23 year old medical student sustained a radial collateral ligament tear of his right small finger testifying to the epidemiological evidence. He did not seek treatment and 2 months later was still having laxity of more than 20° in extension. He was prescribed an ulnar gutter splint at night and buddy taping during the day. After 2 months the swelling subsided and the laxity reduced to less than 10°

Surgical Treatment

Instability after closed reduction, persistent laxity and an associated fracture fragment (Fig. 15.17) are indications for surgical treatment. However, these are rarely seen and most acute injuries will heal with splinting.

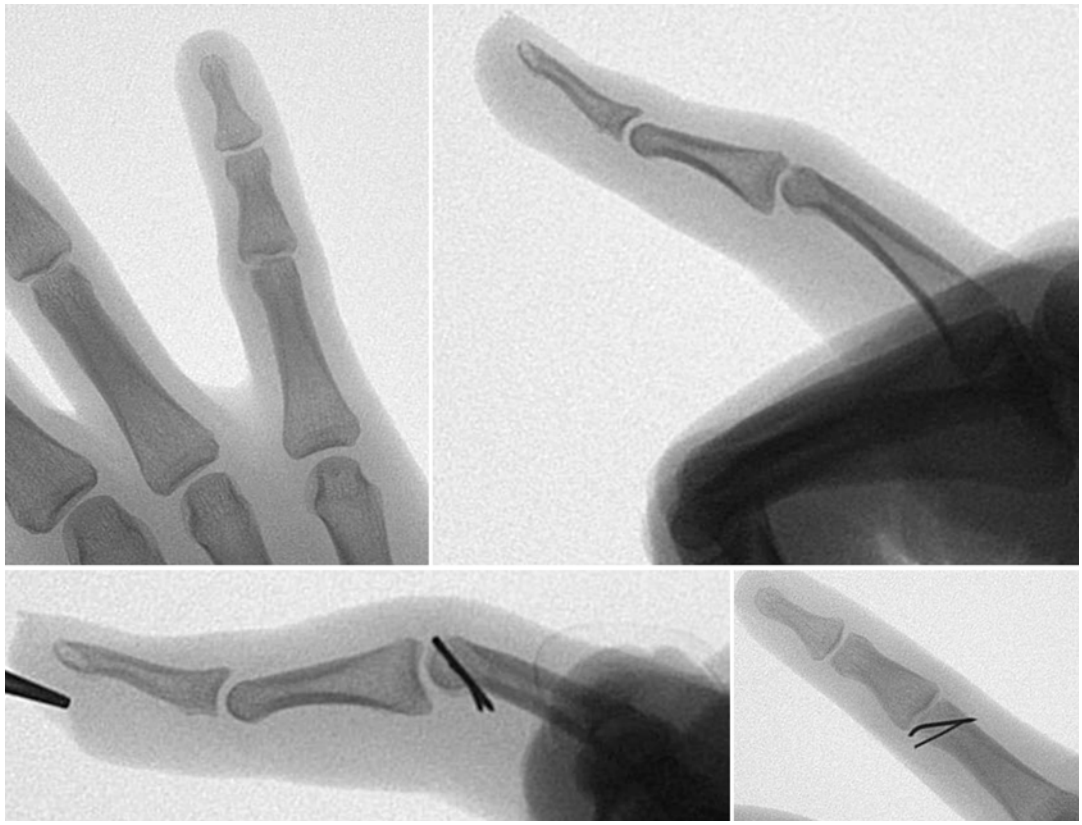


Fig. 15.17 AP and lateral views of an avulsion chip fracture of the radial collateral ligament of the PIPJ of the right small finger. It was fixed with 0.6 mm K-wires

Outcome and Lit Review

Most authors feel that the majority of these injuries will heal with conservative treatment. If however the joint is incongruent after reduction or functionally unstable, then open repair is indicated. Glickel advocates buddy taping but found radial collateral tears of the index finger may need open repair [28]. Redler suggested open repair in complete ruptures due to the frequent ligament interposition causing persistent instability after closed reduction [41].

Complications

Pain, swelling, chronic laxity and of course stiffness are the complications listed in the literature. Advice should be given at the outset that PIP joint injuries take months to heal and swelling may take up to a year or more to subside. As a result of this, it is inadvisable for patients to alter any rings for at least 12 months. Indeed in certain cases the swelling may be permanent. Chronic laxity may require exploration and either tightening (shortening) of the ligament or reinsertion into bone with a mini anchor [41]. Reconstruction with a sublimis tendon has been described.

Conclusions/Personal View

We believe that the PIPJ is prone to stiffness and therefore the mainstay of treatment in acute cases should be conservative with early controlled motion with a close eye kept on the more 'severe' cases. If progress is not being made or there is primary instability after closed reduction, then there is a place for open repair, bearing in mind surgery may lead to more complications including stiffness. Chronic lesions with persistent instability are better

candidates for surgery and have a greater likelihood of improvement.

Volar Dislocation of the PIPJ

Epidemiology and Pathomechanics

The dorsum of the PIPJ is composed of the extensor hood complex consisting of the central slip and the two lateral bands with the associated transverse and oblique retinacular ligament systems [33]. This 3-sided complex besides extending the PIPJ also prevents volar displacement of the P2 [33]. Therefore volar dislocations of the PIPJ are rare, but serious injuries. They may occur in isolation (volar dislocation) or sometimes with damage to one collateral ligament causing that side to 'drop' producing a rotatory component (volar rotatory subluxation) [28]. To complicate matters they may be open injuries usually sustained when the fingers get caught in a machine that hasn't stopped spinning (washer, dryer or blender) with associated contamination.

Clinical Features and Diagnosis

The mechanism of injury is a longitudinal compression force on the flexed digit which forces the P2 base to be pushed under the P1 head. The P1 condyles pierces through the extensor mechanism, stripping the central slip off the middle phalangeal insertion and sometimes buttonholing through the central slip and between the lateral bands. The head thus gets trapped between these two structures making closed reduction difficult.

On examination, the joint will be swollen, tender and deviated. Movement is limited and active extension is not possible. The most crucial part of any examination is to assess the integrity of the central slip and the rotatory component. Early detection will prevent dealing with a Boutonniere deformity and a contracted joint later.

Treatment Options

Radiographs will show rotation if present. Closed reduction under digital block, however, is most times not possible. Glickel et al. have described a special reduction tool which allows them to keep the MCP and PIP joints flexed and the wrist extended allowing traction to be applied to the finger [28]. This has the effect of relaxing the volar lateral band and with gentle rotatory movements the P1 condyles can be extricated from the 'noose'. Subsequently, the joint is immobilised in full extension for 4–6 weeks.

Surgical Treatment

Incongruency of the joint may be due to trapped structures preventing reduction. A mid-axial approach allows good visualisation of the damaged lateral bands [1]. Normally any lateral band should be repaired, although it is safe to excise if badly damaged since the contralateral lateral band is able to perform the extensor function. Post op splinting is in extension and early ROM may be started at 1–2 weeks' time.

Complications

A fixed flexion deformity similar to a chronic boutonniere is the most common sequelae often as a result of delay in diagnosis or inadequate reduction initially [33]. Secondary arthritis can also develop. Depending on the extent of the initial injury and damage to the structures, reconstruction may involve release and repair of the volar plate, repair of the collaterals, release of the lateral band and reconstruction of the extensor mechanism with restoration of the joint. Arthrodesis and arthroplasty are also viable options.

Clinical Pearl

We believe that the PIPJ is prone to stiffness and therefore the mainstay of any treatment in acute cases should be joint protection and early mobilisation, surgery being confined to the open or complex cases.

Open Dorsal Capsular Injuries

Epidemiology and Pathomechanics

The dorsum of the hand is constantly exposed to the elements and the PIP joint due to its prominence is particularly prone to injury. Minor scrapes

Table 15.2 Classification of the type of injury to the dorsal capsule of the PIP joint

Type	Description	Treatment	Case example
I	Full thickness dermal loss	Split skin graft or regenerative agent dressing	Fig. 15.18
II	Skin, subcutaneous and tendon tear or minimal loss	Meticulous repair in layers	Fig. 15.19
III	The above with dislocation of joint	Repair or fixation of structures with immobilisation (external fixator)	Figs. 15.20 and 15.21
IV	Composite loss of tissues in one finger or multiple digits involved	Repair, reconstruction in stages, possible immobilisation with external fixator or fusion. Joint replacement is a later option	Figs. 15.22, 15.23 and 15.24

are not an issue and any loss of dorsal skin can be dealt with in a variety of ways to prevent scarring and contracture. Composite loss of soft tissue and extensor tendon is more challenging and is usually not covered in standard texts. The typical patient is usually a young male who has been involved in a motorcycle (or car) accident, either gripping the handlebars (or flung out of the window) then scraping them along the road surface. The ulnar digits are more often affected presumably because the rider falls to one side. Less often, the injury may have been sustained in line of work especially those dealing with abrasive surfaces on a roller mechanism.

Classification

We have classified this based on the depth of injury and tissue involved (Table 15.2):

Type I: Third degree abrasive burn of the skin with full thickness dermal loss.

Type II: Break in the skin, and tearing of the extensor tendon with minimal or no loss of tissue or bone and no joint involvement.

Type III: Skin, tendon and joint capsule exposed with joint subluxed or dislocated.

Type IV: Composite loss of tissues in one finger or multiple digits involved

Clinical Features and Diagnosis

Getting to know the patient by taking a detailed **history** helps in the decision making in complex cases. The psyche of the patient helps the surgeon in determining whether compliance will be an issue, instructions are understood and followed or the patient would be difficult to manage. Other details will include the patient's age, occupation, handedness and mechanism of injury.

In the **examination** the key point to note is the depth of the abrasive injury and the number of tissues lost that need to be replaced, particularly assessing central slip attachment and the stability of the PIP joint. This is done by asking the patient to extend a flexed PIPJ (90°) against resistance. Failure to extend and fixed extension at the DIPJ reveals complete central slip disruption.

Treatment Options

Type I Injuries

Only Type I injuries may be managed conservatively. Partial thickness skin loss is not an issue, however full thickness loss may require grafting. We have found it extremely useful to treat dermal loss with a regenerating agent (Cacipliq®). The time taken to heal smaller wounds may be the same but the quality of skin that 'grows' back is more pliable and less prone to breakdown (Fig. 15.18).

Surgical Treatment

Open injuries of course require formal lavage and meticulous debridement under magnification.

Type II Injuries

Although these are relatively 'simple' injuries, they should not be taken lightly. According to Boyes, central slip excursion is only 2–3 mm at the PIPJ [42]. Thus this is the amount of skin and extensor tendon loss that can be tolerated yet still allow primary repair to be carried out. Therefore,

Fig. 15.18 (**a**) A 15 year old student sustained a full thickness burn to the dorsum of his right middle finger just distal to the PIP joint after an explosive chemistry experiment! (**b**) His mother opted for debridement and dressing. (**c**, **d**) We used a regenerating agent and the wound healed within a month. (**e**) The new skin was of good thickness and no limitation of movement or grip strength was noted

this repair must be done meticulously in layers, care being taken to bury the sutures under the extensor tendon and capsule to prevent scarring and adhesions (Fig. 15.19). Post-operative splinting should be in full extension for 2–4 weeks before gentle range of motion exercises are initiated.

Type III Injuries

When there is loss of dorsal capsular structures (extensor tendon, triangular ligament and the dorsal capsule), the joint is laid open and may result in dislocation. Reduction can usually be easily achieved, although maintenance often requires some form of fixation. We find the LINK® external fixator a good option being fast and easy to apply (Figs. 15.20 and 15.21).

Type IV Injuries

These are complex injuries of composite tissue types that may require more than one surgery. Thorough and meticulous debridement is the key to success. A systematic assessment of structural loss should be undertaken.

Any **bone damage** should be stabilised in the primary setting. In our experience if there is bone loss, it is better to stabilise it initially and address any loss secondarily by either osteo-chondral bone

Fig. 15.19 A 31 year old motorcyclist skidded and crashed to his right side when a tree branch fell on him. The ulnar two digits were scraped along the road. (**a**) An Ulnar based skin flap is lifted and the extensor loss is seen underneath. (**b**) Lifting up the extensor, the P1 has also been abraded but cortex is still intact. (**c**) There is enough laxity for a minimal loss to be overcome by fine suturing – PDS 5/0 is used here to approximate the extensor mechanism. (**d**) After skin closure, the joint is splinted in extension for 4 weeks

grafting (Fig. 15.22), joint transfer, arthroplasty or finally arthrodesis (Fig. 15.23) if all else fails.

Any injury to **the extensor mechanism** should be repaired or at least an attempt made to do so. Generally, we find it unnecessary to graft or reconstruct in the acute setting and healing occurs particularly if the joint is immobilised in extension. This usually results in the restoration of extensor function (Fig. 15.22).

Skin loss is dealt with depending on the amount of loss and the tissues exposed. If more than 2 cm^2 of tendon or bone is exposed, a rotational flap or venous flow through flap is required. However, we have found that in most cases the dorsal tissue can be approximated and allowed to heal by secondary intention (Figs. 15.22 and 15.24).

As in all PIPJ injuries, the patient is cautioned about the long recovery period and risk of stiffness and persistent swelling.

Complications

Failure to pick up a central slip avulsion or tear can result in a boutonnière deformity later.

Fig. 15.20 An 18 year old despatch rider who just obtained his licence skidded and fell off his motorcycle. (**a**, **b**) He sustained abrasion wounds on the hypothenar eminence and the three ulnar digits, of which the middle finger was the worst. (**c**) The central slip was avulsed and there was loss of the dorsal capsule. The joint had subluxed volarly but was easily reduced from this 'locked position' and the dorsal defect repaired. (**d**) The reduction was maintained with an external fixator. (**e**) After 6 weeks of immobilisation, the joint was stable. (**f**) Once the fixator was removed in clinic, the joint was put to motion and resulted in a good outcome

Fig. 15.21 (**a**) A similar case (Type III) with extensor tendon loss and some bone damage of the PIP joint in the (R) small finger causing a drop. (**b**) An external fixator holds the repair in extension for 6 weeks. (**c**) The skin heals by secondary intention. (**d**) Good extension (and flexion) of the PIPJ is achieved. (**e**) There is some loss of DIP flexion

Fig. 15.22 (**a**) A 32 year old engineer was trying to fix a roller belt at a client's factory and caught his (R) Index finger in the machine sustaining an abrasive loss of the dorso-radial aspect involving bone, tendon, ligaments and skin. (**b**) After debridement, the loss is clearer. (**c**) The finger drops due to absence of the dorsal extensor mechanism. (**d**–**f**) With the help of a k-wire and external fixator the phalanges are held in position and he is able to flex his MCP joint. The extensor mechanism was approximated but no formal reconstruction undertaken. The radial collateral ligament was attached back to the remnant P2. (**g**) The index finger 3 months later. He only had 30° of pain-free flexion at the PIP joint. (**h**) The DIP was fused and the PIPJ reconstructed with the help of an osteochondral graft held in place with a plate and screws, increasing PIP flexion to approximately 50°. An arthroplasty may be performed if pain becomes an issue

Prolonged immobilisation, however, will undoubtedly cause stiffness, although ultimately as the flexor mechanism is stronger than the extensor, this may be overcome with aggressive therapy. Instability due to ligamentous deficits or bone loss is also possible and should be addressed accordingly. Whatever, meticulous handling of tissues and thorough debridement will diminish the risk of infection.

Fig. 15.23 (**a**, **b**) A 55 year old chauffeur on his way to work fell off his motorcycle and sustained composite loss of skin, tendon and bone of the PIPJ of his left small finger. (**c**) The extent of comminution and damage pointed towards fusion which was performed secondarily after initial debridement. (**d**, **e**) Function is not bad

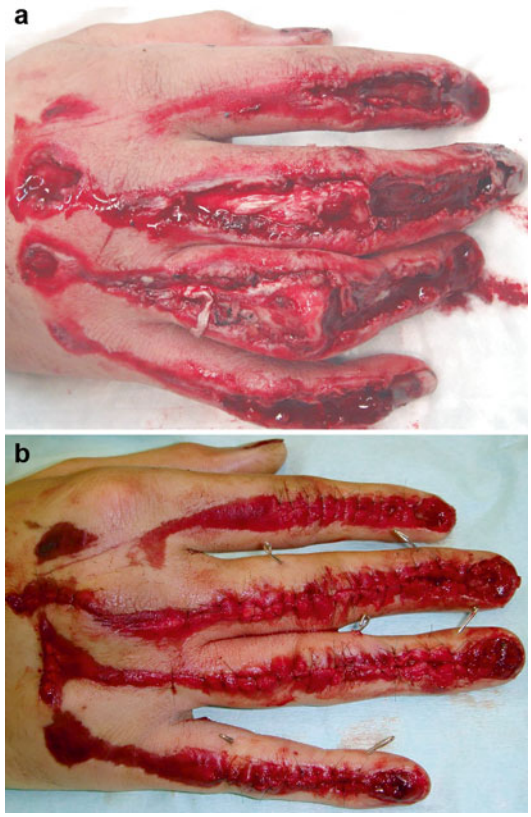


Fig. 15.24 (**a**) An F1 driver whose hand was flung out of his car and hit the gravel. There is complete composite loss of tissue including the dorsal cortex of the phalanges. (**b**) Approximation and closure is possible with all the joints of the ulnar 3 digits immobilised with fine 0.8 mm k-wires. He was lost to follow-up in Japan (Photos courtesy of Dr. Ranjit Singh Gill)

Outcomes, Personal Views and Conclusions

Unfortunately, there is little in the literature with regard to the treatment or outcome of these injuries. What is known is that their preponderance is to the ulnar digits particularly as these are the first to hit the ground [38]. Treatment should be based on the severity of the injury and the surgeon's comfort in dealing with it.

Clinical Pearl

The basic principles of management include salvage, followed later by procedures to improve function.

The Thumb Metacarpophalangeal Joint: (MCPJ)

Introduction

The term "sticks out like a sore thumb" is not without reason; with it's 90° anteverted position and wide 1st web space place, the thumb is often in a more exposed position, making it particularly vulnerable to injury more so since it is involved in almost all hand function. It is thus unique in that it has to impart both mobility and stability. While the former is afforded by the distal IP and proximal CMC joints, it is the MCP that confers stability, allowing the thumb to be used simultaneously for power grip and precision pinch and providing a stable post against which the finger pulps oppose to perform their myriad functions [43]. Therefore functional disability following injury (the thumb being allocated 40 % of hand function [44]) to this important joint is not due to stiffness but more of instability, deformity, or pain [45].

Anatomy and Clinical Implications

The Metacarpophalangeal Joint (MCPJ) of the Thumb

Bony Architecture

Stability of the 1st MCPJ is due to a number of factors:

- The shape of the metacarpal head is more quadrangular (Fig. 15.25). The flatter head decreases the range of motion [in both the flexion-extension (0–100°) and abduction-adduction (0–20°) planes] more so in extension (6°) than when in 15° of flexion (12°) [46]. This rigidity purportedly reduces the chances of injury [28].
- There is a wider distribution of cartilage on the volar surface (as opposed to dorsal for the finger MCs) encompassing the sesamoids and the more prominent condyles, ulnar longer than radial, which promotes a certain degree of pronation in flexion to assist in thumb opposition [8].

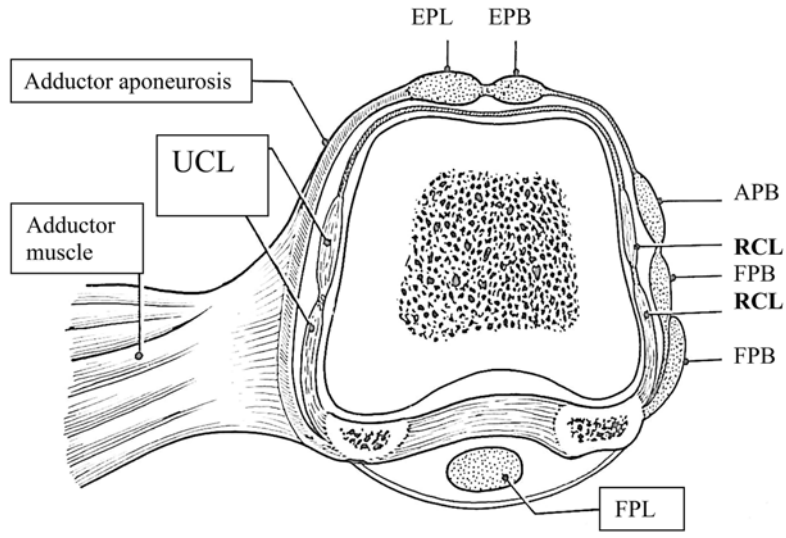


Fig. 15.25 Aubriot described the quadrangular shape of the Thumb MC and the structures that formed the surrounding superficial and deep layers of support (Taken with permission from Aubriot [43])

- Additional constraint and stability is provided by the consistent presence of two sesamoid bones that are embedded in the lateral margins of the volar plate and also are incorporated into the tendinous insertions of the FPB and Adductor Pollicis on the radial and ulnar sides respectively [43] (Fig. 15.25).

Table 15.3 Stabilisers of the thumb MP joint

Static	Dynamic
Dorsal capsule	Extrinsic muscles
Ulnar collateral ligament	Extensor pollicis longus (EPL)
Radial collateral ligament	Extensor pollicis brevis (EPB)
Volar plate	Flexor pollicis brevis (FPB)
Sesamoids	Intrinsic muscles
	Adductor pollicis brevis (APB)
	Flexor pollicis brevis (FPB)
	Adductor pollicis

Joint Capsule

The di-arthrodial hinge joint, which is similar to the finger PIPJ, has little intrinsic stability and depends on a number of static and dynamic stabilisers for additional support (Table 15.3). The static restraints are the volar plate, the collateral ligaments (ulnar and radial) and the dorsal capsule. **The dorsal capsule** consists of a *superficial and deep* fibrotendinous layer: the superficial one houses the extrinsic extensor tendons – Extensor Pollicis Longus (EPL) and radial to it the Extensor Pollicis Brevis (EPB), whereas the *deeper* layer consists of the joint capsule itself which is quite thin covering the articular surfaces to within 2 mm (Fig. 15.25). It is adherent to the extensor tendon sheaths.

Lateral surfaces–Ulnarly, the *superficial* layer continues to enclose the stout Adductor Pollicis tendon enveloping the whole ulnar side from its attachment to the medial sesamoid in the volar plate and volar rim of the proximal phalanx to the extensor expansion dorsally (and EPL) distally forming a triangular expansion. On the radial side the *superficial* layer is not as well-developed but encloses the thenar muscle insertions of Flexor Pollicis Brevis (FPB) and Abductor Pollicis Brevis (APB) in a sheath joining the larger lateral sesamoid to the extensor expansion dorsally [43]. The FPB has two heads, a proximal deeper one attaching to the sesamoid and volar plate and a superficial head which attaches slightly distal and more dorsal to the proximal phalanx volar base (Fig. 15.25). The APB lies more superficial to these and also is more dorsal in its attachment on the proximal phalanx [43]. The **collateral ligament complex** is the main structure stabilising the joint, ensheathed within the *deeper fibrotendinous layer on both sides*. It is made up of the proper

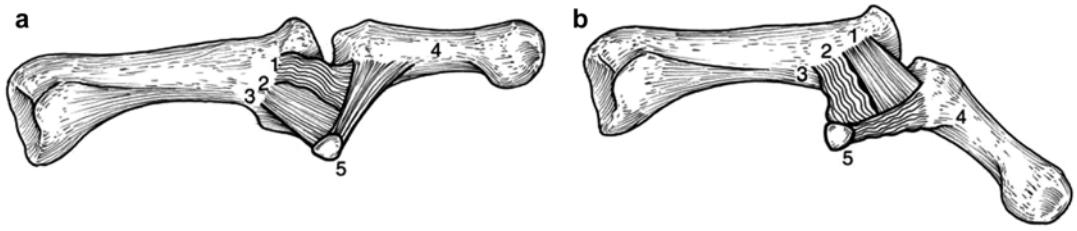


Fig. 15.26 Illustration of the collaterals in the thumb MCPJ. (**a**) In extension, the accessory collaterals (*2*) and volar plate (*3*) are taut whereas (**b**) in flexion, the proper collateral (*1*) is tight, maintaining stability throughout the range of motion (Taken with permission from Tang [47]), where 4 is the proximal phalanx and 5 is the 1st metacarpal

and accessory collateral ligaments (CL and ACL), the former being positioned more dorsal than the latter, measuring 4–8 mm in width and 12–14 mm in length. The thicker Ulnar Collateral Ligament (UCL) arises from a tubercle just dorsal to the mid-axis of the metacarpal head and descends palmar and distal to attach to the volar rim of the proximal phalanx just beside and below the adductor insertion, which overlies it. Similar to the fingers but to a lesser degree, because both condyles are prominent volarly, in flexion the proper collateral ligaments stretch over the condyles making them taut, while the accessory collateral ligaments and volar plate get bunched up underneath. The reverse occurs in extension with the accessory collaterals and VP becoming taut and the proper collateral relaxing (Fig. 15.26) [47]. The ever-present sesamoids provide the much-needed lateral constraint, which, in the fingers is afforded by the flexor sheath forming a pulley to prevent bowstringing [48]. The **volar plate** of the thumb MCPJ is similar to the finger PIP joints, although there are important differences. As in the PIPJ, the plate is 2–3 mm thick distally and attaches firmly to the proximal volar margin of the proximal phalanx. Proximally however it tapers to become membranous centrally with a flimsy attachment to the MC neck with no reinforcement by check rein ligaments, hence dislocation tends to be dorsal [45].

Extrinsic Joint Support

The dynamic stabilisers include the intrinsic muscles: Flexor Pollicis Brevis (FPB) and Abductor Pollicis Brevis (APB) on the radial side and Adductor Pollicis on the ulnar side. Their tendinous insertions are thicker and stronger than in the fingers, this is especially evident on the medial side extending dorsally in the form of the aponeurotic expansion, which along with the sagittal bands, keep the EPL balanced in the midline. The latter not only extends the IP joint, but also the MCP, although the EPB is the prime extensor. The latter, however, is only a thin tendon inserting into the dorsal capsule and base of the dorsal proximal phalanx. As a consequence, the dorsal integrity at the MCP joint depends very much on the dorsal capsule.

Dorsal Capsular Injuries (Volar Dislocations)

Epidemiology

Pure volar dislocations are rare injuries occurring ten times less often than their more common dorsal counterpart, although when they do occur they result in tearing of both the dorsal capsule and avulsion of the thin EPB tendon with or without volar subluxation [49]. Inevitably the dorsal parts of the collaterals may also be torn, although isolated capsular tears have been reported [49]. The mechanism of injury is a dorsal force applied with the MCP in flexion, either pushing in an anteromedial direction, tearing the dorsal capsule together with the RCL or less often anterolaterally injuring the UCL [50, 51].

It must also be borne in mind that only the capsule and two extensor tendons maintain dorsal stability with perhaps some input from the collaterals. The FPL and all the intrinsics exert a

flexor force, which in the absence of capsular integrity would cause the joint to subluxate or even dislocate volarly.

Clinical Features and Diagnosis

Pain over the dorsum of the MCPJ is the primary complaint with lack of full active extension of the proximal phalanx a close second [50]. The pain may be present for up to a year or more. In mild cases the volar subluxation may not be obvious clinically, although tenderness or a swelling over the dorsal aspect is an important sign to be elicited. X-rays will fail to show any fracture, but will demonstrate displacement (Fig. 15.27). Radial and ulnar laxity **must** always be assessed.

In severe cases, it should be obvious if there is a subluxation or dislocation. The head of the metacarpal buttonholes through the extensor hood displacing the EPL and APL tendons to either side, pulling on the distal phalanx causing IP hyperextension [49]. Thus there is a prominence of the head followed by a depression where the proximal phalanx has sunk volarly. Closed reduction may be achieved relatively simply by longitudinal traction. The joint is then stressed and is usually stable due to the intact (at least partially) collaterals and sesamoids. If it is found to be unstable, repair of the capsular structures and EPB should be undertaken.

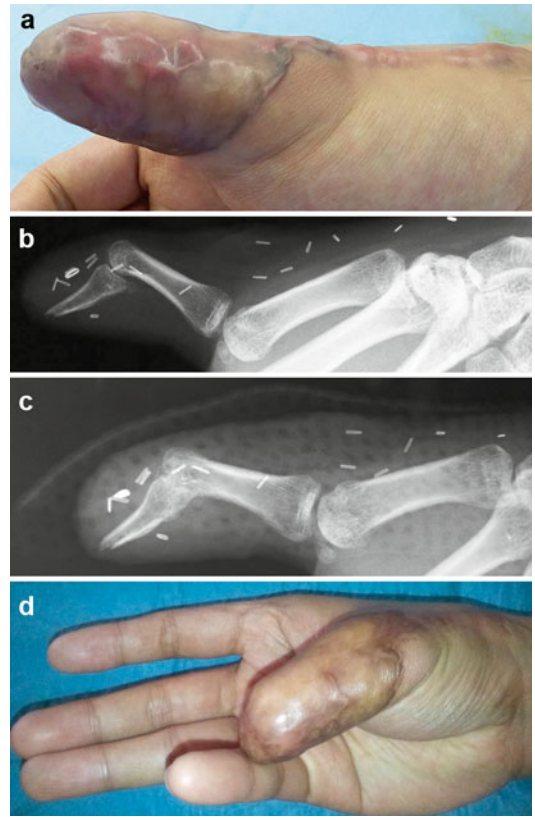


Fig. 15.27 Thirty four year old got his right (dominant thumb) caught in between rollers and sustained a degloving injury. (**a**) The plastic surgeon performed a reverse radial forearm flap. It was fine until he used a vacuum dressing to reduce swelling. (**b**) The whole soft tissue envelope shrunk and the MCP went into hyperextension and the DIP into flexion, the former dislocating. Not much movement was possible. (**c**) Splinting helped but not enough to reduce the joint. (**d**) Joints were reduced under anaesthesia and held with k-wires to overcome the forces and the issue resolved. He was able to flex and oppose his thumb

Treatment Options

The most critical point is stability of the MCPJ. Posner and Retaillaud advocated 'prolonged immobilisation in extension' with a thermoplastic splint [51]. We usually start gentle range of motion (ROM) exercises after 1 month. The splinting is continued for 6–8 weeks.

Surgical Treatment

In the presence of a subluxation, dislocation, or an extensor lag, surgery is indicated [50, 51]. Krause in their series of 11 cases succeeded in treating 4 patients with splinting alone but none of them had subluxation or extensor lag [50]. We would reduce the joint closed, assess the collaterals before holding it in extension with a fine 1.0 mm Kirschner wire. The reduction is checked with an image intensifier and the MCPJ capsule is then repaired using PDS sutures. The EPB tendon may have to be reinserted either with a mini bone anchor or using heavy sutures through part of the remaining capsule. The joint is then supported in extension for 6–8 weeks with a thermoplastic splint. The wire is removed at approximately 6 weeks and gentle ROM exercises initiated.

Outcome and Lit Review

There are a few points of interest. These injuries although uncommon alone, occur fairly

frequently with collateral ligament tears and are hence *'missed'*. The presentation therefore is usually delayed and subtle. The main features to look out for are the *subluxation and lack of full active extension,* which go hand in hand and signal a likely surgical treatment. Krause et al. reasoned that isolated capsular tears occurred in those with *more flexible thumbs*, in that the longer collateral ligaments allowed for more stretch and ultimate failure of the capsule [50]. They also postulated *dorso-radial tears were more frequent* because with the thumb in flexion (thumb in palm almost), an ulnar directed force was more likely since the radial directed force would be blocked by the 4 fingers. Hence the dorso-radial corner was most affected, it also being the weakest point [50]. They also postulated that attenuation and tearing of the capsule affected the EPL tendon hence active extension. Therefore a lack of full active extension meant surgical exploration and repair were essential [50].

Complications

Persistent pain, instability and lack of full active extension are the most obvious features that may appear due to a missed diagnosis or improper treatment. Failure to reattach the EPB may result in weakness of extension, although the EPL *may be able to* address that. It is essential to repair the capsular rent and realign the EPL to midline so that the line of pull is restored.

Persistent anterior dislocation may present as a boutonnière with fixed flexion of the MCP and hyperextension of the IPJ [49]. Osteoarthritis is not unusual in neglected cases.

Clinical Pearl

Although a relatively rare occurrence in isolation, it has serious consequences if left untreated. The thumb being an important digit, we must place emphasis on an alert eye and a good clinical examination assessing the **subluxation** and **extension lag**.

Volar Plate Injuries (Dorsal Dislocations)

Epidemiology

Dorsal MCP dislocations are far more common than volar [51] and occur more in the thumb than the fingers [9]. Farabeuf first described it in 1876 as resulting from a hyper-extension force most commonly due to a fall at work or play [52]. The phalanx is driven backwards over the metacarpal head along with the volar plate, sesamoids and intrinsics attached. In the initial stages, hyper-extensibility of the MCPJ may follow a tear in the volar plate resulting in a simple dislocation (Fig. 15.28). However, for a true complete dorsal dislocation to occur, with the proximal phalanx in a 'bayonet' position over the MC head, one of the collaterals *has to be* torn [53, 54]. Thus as the collaterals ultimately tear, the metacarpal head protrudes through the flimsy central portion of the volar plate dividing the FPL and Adductor to the medial side and the FPB to the lateral (Fig. 15.29). The FPL stays within its sheath and tends to form a 'noose' around the MC head which along with the FPB scissors and tightens when the phalanx is flexed, thus hindering reduction. Therefore specific steps must be followed for closed reduction otherwise as in the fingers, a simple complete dislocation may be converted into a complex irreducible dislocation (see Table 15.4 for the comparison).

Clinical Features and Diagnosis

In the acute stage of a simple dislocation, the thumb is in a grotesque Z deformity with the IPJ in flexion and the proximal phalanx pulled ulnarly and completely dorsiflexed over the herniated MC head at a 90° angle (Fig. 15.28). The thenar muscles are bruised and oedematous and movements worsen the deformity. Any attempt to flex the MCPJ causes excruciating pain and would also obviously tighten the flexor-adductor 'noose' around the MC head, preventing reduction. Usually there is no sensory deficit [9]. AP and lateral radiographs clearly demonstrate the dislocation and the absence of any fracture. It is crucial to note the *position of the sesamoids relative to the proximal phalanx (P1)* in the lateral radiograph

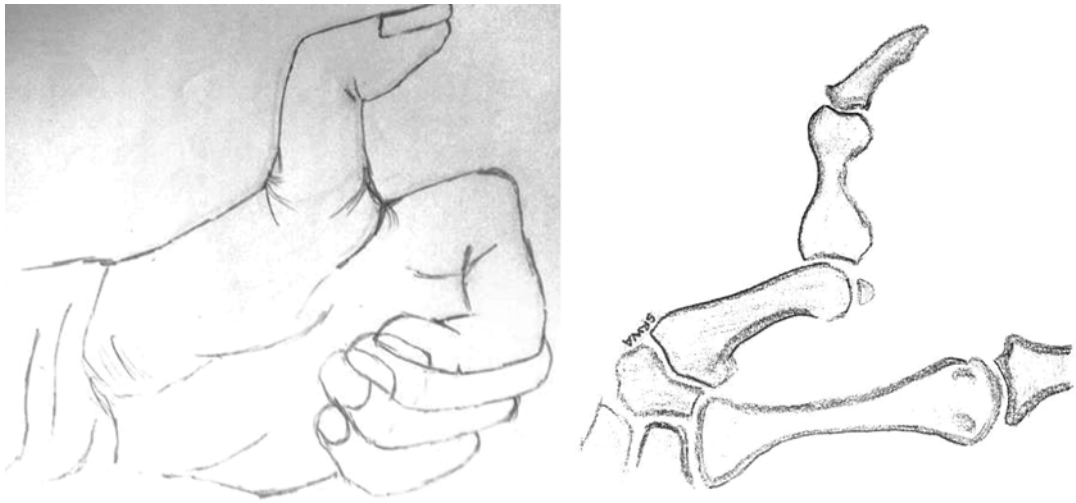


Fig. 15.28 The simple dorsal MCP Dislocation. Line drawing of clinical appearance of dorsal dislocation and the corresponding radiographic appearance: The position of sesamoids relative to P1 base is significant because it gives an indication of the location of the volar plate tear (proximal or distal). This affects management (see text)

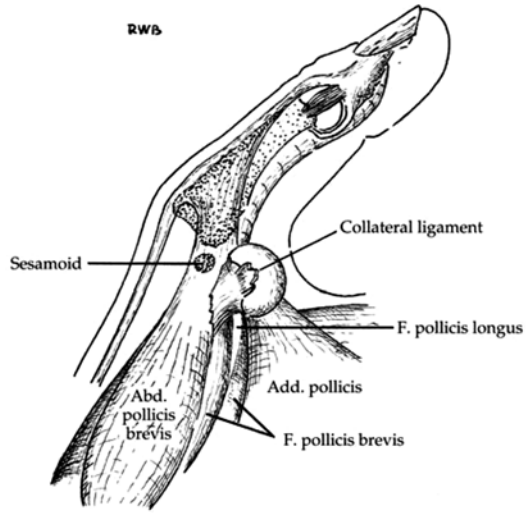


Fig. 15.29 Diagram of a Flexor-Adductor noose that forms around the 1st MC head in an irreducible dorsal dislocation. The FPB is lateral and FPL and adductor are medial, the latter winding around the MC neck. As the P1 flexes, it pulls on the APB, FPB and the FPL causing a 'strangulation' of the herniated MC neck (Taken with permission from Beasley [73])

Table 15.4 Comparison of a simple and complex dorsal thumb MCP dislocation

	Simple dorsal dislocation	Complex dorsal dislocation
Position of P1 in relation to MC	P1 at 90° to MC	P1 near parallel to MC shaft: "bayonet" position
Appearance	Grotesque	Near normal
Joint space	Maintained	Widened
Sesamoids	Usually follow P1	Intersposed in between P1 and MC
Reduction	Simple; closed	Complex; open

(Fig. 15.30): if as is usual, the VP is disrupted proximally, then the sesamoids would be attached to the distal end and close to the proximal phalanx base (Fig. 15.28) which is dorsal to the MC head; if the VP is avulsed from the P1 base (distal disruption), then the sesamoids would have retracted proximally pulling the intrinsic insertion off the volar base of P1. Sometimes, the tear is *through* the sesamoids and even rarer there is a fracture of the sesamoids which implies that the dislocation is simple [53, 54]. In contrast, interposition of the sesamoids (and hence volar plate) between the MC *head* and P1 strongly suggests a complex, irreducible dislocation [28] (Fig. 15.31).

Treatment Options

Closed reduction is usually successful if attempted correctly. Under adequate anaesthesia in theatre (we prefer TIVA – total intravenous

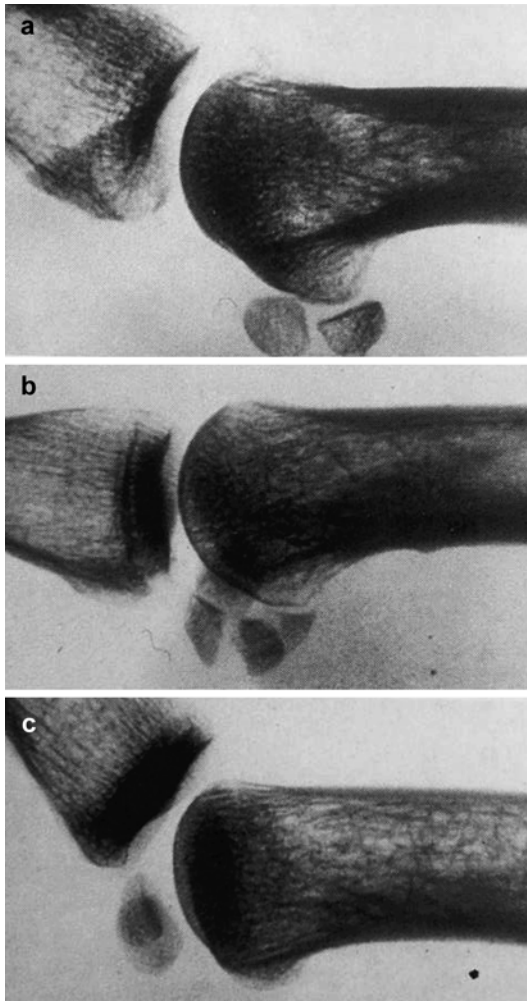


Fig. 15.30 How a lateral radiograph assists in determining the location of the volar plate (VP) tear. (**a**) Distal lesion: The VP has torn off from the proximal phalanx thus an extension view distances the sesamoids from the base of the proximal phalanx. (**b**) Both radial and ulnar sesamoids are fractured. (**c**) Proximal lesion: the sesamoids move with the proximal phalanx; the VP need not be attached here for it is flimsy (Taken with permission from Stener [54])

anaesthesia) the wrist and IPJ are flexed to relax a possibly tight FPL and the metacarpal is flexed and adducted to loosen the intrinsic 'noose' around the MC head. The P1 is then hyperextended and 'grounded' against the dorsal surface of the metacarpal 'rocking' it to release any trapped tissue in between. With dorsal pressure over its base it is then advanced distally pushing the sesamoids ahead of it. As it is flexed over the MC head curvature – a clunk may be heard upon reduction which is then confirmed with an image intensifier. Lateral and AP stability is tested in extension and flexion. If stable, the joint is splinted for 2 weeks in 20° of flexion. ROM exercises are cautiously started after that with continuation of splinting for another 2 weeks.

Surgical Treatment

If the joint is unstable after closed reduction (dislocates again or has 40° of medial or lateral laxity on stressing) open reduction is warranted [55]. A dorsal approach for reduction is easier, however a volar approach makes more sense because after reduction, the damaged structures are visualised more easily. Hyperextension instability can be reduced by repairing the rent in the proximal volar plate but more importantly reattaching the intrinsic tendons to their insertions using a mini bone anchor. Alternatively a pull out wire may be used (for proximal and distal avulsions). Occasionally, a Kirschner wire is needed for stabilising the joint.

Via a volar Bruner approach, the incision's apex is at the radial midaxial border. Care must be taken to identify the digital nerves especially the radial neurovascular bundle which crosses across the surgical field. Removal of interposed soft tissue which most often includes the FPL, sesamoids, volar plate and intrinsic tendons is done with care. These may need to be divided or retracted. Once the metacarpal head is reduced, stability is checked and repair carried out as necessary. Post-operative splinting is usually slightly longer at 4 weeks but flexion exercises may be started at 2 weeks.

Outcome and Lit Review

Coonrad reviewed 26 MP dislocations, all dorsal, none volar indicating the frequency of the former [46]. Of the 132 thumb dislocations McLaughlin described, he couldn't reduce 22 of them closed thus used a volar approach and after reduction started immediate mobilisation with good results [56]. Bertil Stener in his excellent descriptive articles emphasized the importance of the position of the sesamoids and their significance in management [54, 57]. In a distal rupture (at the P1 base or through the sesamoids), the active restraints to hyperextension (the FPB and the Adductor Pollicis which are attached to the sesamoids) 'pull' the

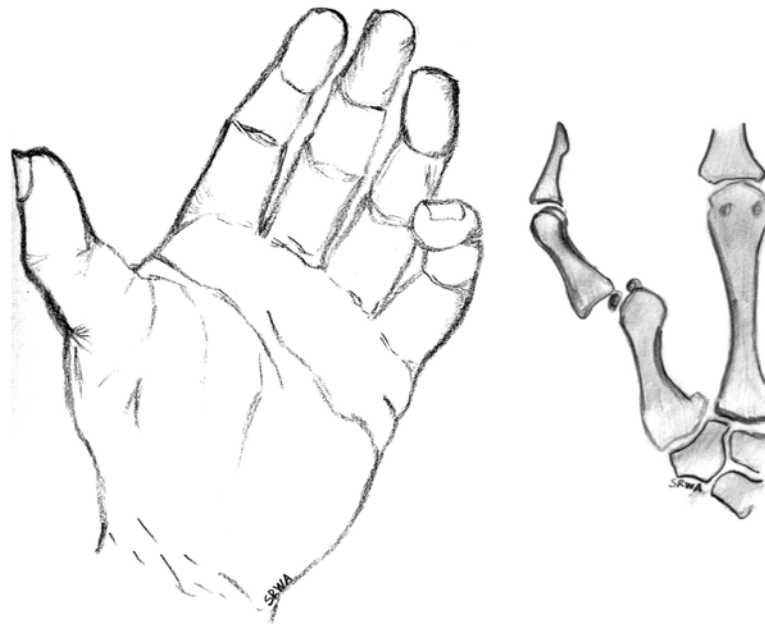


Fig. 15.31 The complex dorsal MCP Dislocation. The MP dislocation paradox – the clinical picture (on the *left*) is not as dramatic as the simple dorsal dislocation (Fig. 15.28) and may be easily missed. The corresponding radiograph (*right*) of a complex dorsal (irreducible) dislocation with sesamoids in the widened joint space. Note that P1 is near parallel to the MC in what is called a "bayonet" position

volar plate (passive restraint) proximally opening up the joint. This feature necessitates repair. In a proximal rupture both restraints are intact, the volar plate is membranous and therefore repair need not be carried out. There are no figures on the outcomes of treatment but generally if forceful extension is avoided in the first few weeks, they do well.

Complications

Most patients tend to do well but need to be warned about stiffness, which is not usually an issue with this joint. Failure to detect volar laxity however, would result in hyper-extension and volar instability of the joint which may become chronic, result in joint collapse with pain and weakness. In this case if arthritis has set in, fusion is a good option (Fig. 15.32).

Clinical Pearl

The thumb metacarpophalangeal joint is very forgiving but extremely important for function. Therefore, although the joint tends to do well on its own, care should be taken with diagnosis, particularly determining position and type of injury so that it can be adequately addressed in a timely fashion.

Ulnar Collateral Ligament Tear of the Thumb: Metacarpo-Phalangeal Joint

Epidemiology

In 1955 Campbell reported disruption of the UCL of the Thumb MCPJ in Scottish gamekeeper's as a result of chronic repetitive valgus strain and gave it the eponym 'gamekeeper's thumb' [58]. The corresponding *acute* injury is given the name Skier's thumb due to it comprising 32 % of skiing injuries, second only to medial collateral ligament injuries of the knee [59]. The **mechanism of injury** is an abruptly applied radially-directed force which may occur in falls, ball sports (rugby, soccer, volleyball, handball etc.) and daily activities. It is therefore no surprise that Moutet et al. in 1989 found that UCL tears made up 86 % of the 1,000 MCP joint injuries studied [60]. The important role of the UCL is to stabilise the MCPJ against valgus stress especially in 15° or more of flexion (when it is taut).

Pathology

The common denominator of ulnar sided injuries to the thumb MCP joint is the UCL. The basic pathology is a stretching of the medial side as the thumb is forced into radial deviation. Initially a partial tear may occur, but as the force continues,

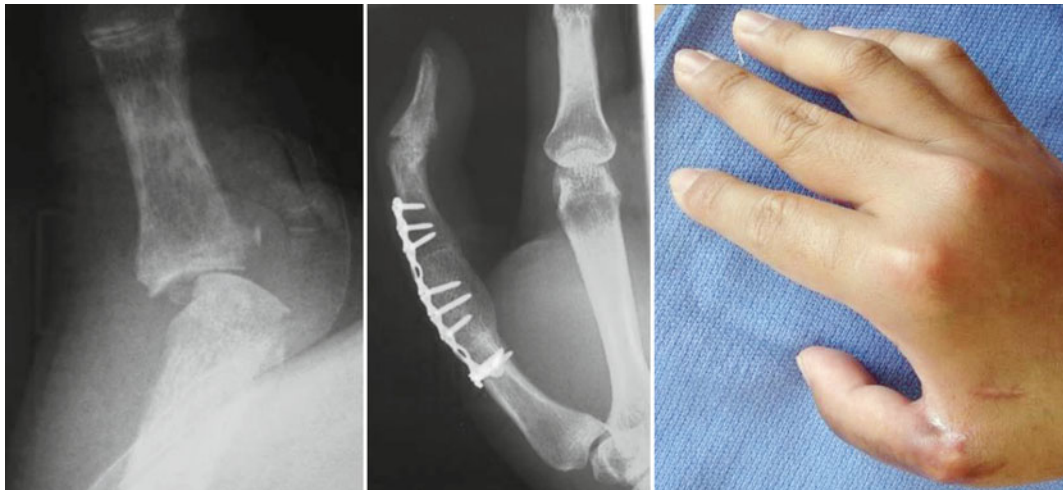


Fig. 15.32 MP dislocation occurred after osteomyelitis set in a patient getting bitten by a snake. Giving a grace period of 2 months for the infection to settle, fusion of the MCP joint was performed and the patient was satisfied

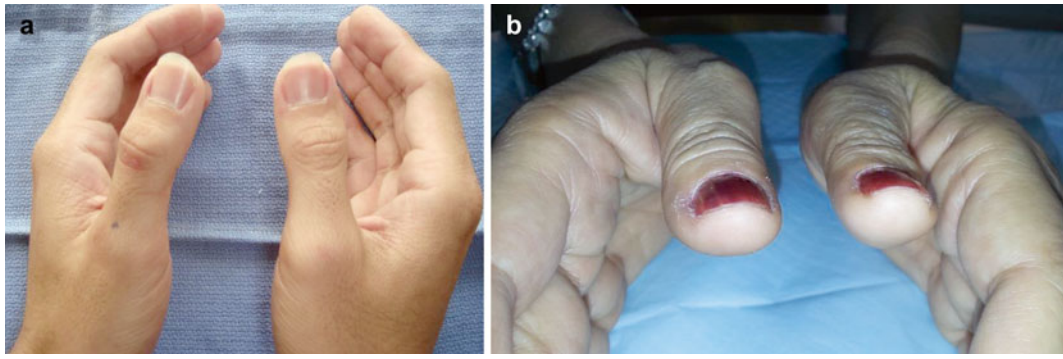


Fig. 15.33 (**a**) Right thumb is swollen at the MCPJ. (**b**) Left thumb is rotated in supination indicating a UCL rupture

both the main and accessory collaterals are torn, ultimately leading to a complete rupture. Three patterns of injury have been identified:

- The UCL is most commonly pulled from its insertion on the volar ulnar aspect of the proximal phalanx, with or without an avulsion fracture.
- Mid-substance and more proximal tears have been reported.
- On rare occasions, the UCL is intact and there is a volar ulnar base shear fracture.

As further radial deviation occurs, the proximal portion of the torn UCL slips out from underneath the adductor aponeurosis and becomes superficial. If no further deviation occurs, the thumb adducts back to its normal position and in so doing the leading (proximal) edge of the Adductor flips the torn UCL proximally over itself, away from the corresponding torn distal portion. This is what Bertil Stener described in his classic article in 1962 where he found that 25 out of 39 consecutive cases (64 %) had this lesion (Fig. 15.33) [61]. These lesions are important, as they often don't heal with conservative management.

Clinical Features and Diagnosis

The patient will often give a history of a fall on the outstretched hand with the thumb abducted. They make complaint of pain over the medial aspect of the MCPJ. On examination, the ulna side of the metacarpophalangeal joint is swollen

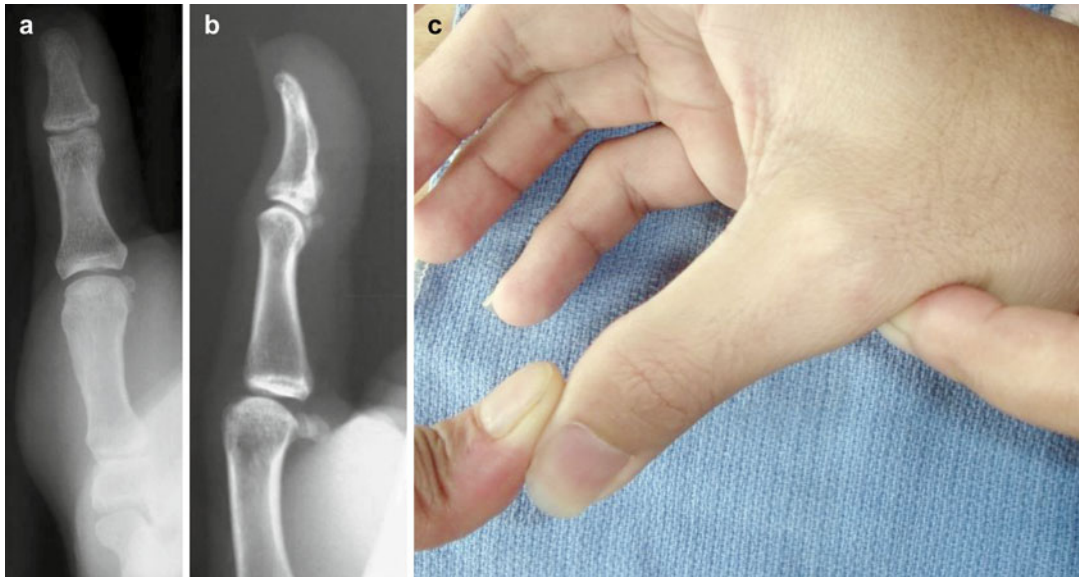


Fig. 15.34 Patient in Fig. 15.33a. (**a**) AP and (**b**) Lateral radiographs of a patient with a UCL rupture. Very minimal opening up of the ulnar side of the joint but significant volar subluxation can be seen. (**c**) Clinical testing shows >40° of opening in 30° flexion

and tender (Fig. 15.33a). Bruising and ecchymosis will be present in the acute setting. Occasionally, a tender swelling can be palpated corresponding to the displaced proximal torn ligament or called a Stener lesion [61]. If a mass is not felt, however, it does not mean that the lesion is not there [62]. Late presentation may include a reduced range of motion, grip strength and loss of function, particularly weakness in performing daily activities such as opening jars or doors or even turning the ignition.

Since the UCL also "pulls" up on the medial side of the proximal phalanx, a torn UCL will result in a "dropped" P1 proximal phalanx resulting in slight rotation (supination) with respect to the metacarpal head [63]. This can be visualised by assessing the rotation of the nail end on with both thumbs held side by side (Fig. 15.33b). The thumb may also be in a slight radial deviation.

Radiographs are essential prior to stress testing. An avulsion fracture may be seen over the volar ulnar base of the proximal phalanx or less commonly there is a shear fracture on the volar aspect of the radial condyle of the MC head. The phalanx is radially shifted and often there is volar subluxation seen on the lateral view (Fig. 15.34)

Table 15.5 Classification of collateral ligament tears

Grade 1	Tenderness over collateral ligament, no instability
Grade 2	Laxity compared to the contralateral digit with definite endpoint
Grade 3	Laxity without endpoint, joint instability

[63]. Fear of creating a Stener lesion or displacing a fracture is not a reason for avoiding stress-testing because the force required to cause the injury is much greater than that achieved in clinical testing [47, 50, 64, 65]. The most critical point is clinical evaluation of joint stability and to determine if the tear is partial (grade 1 or 2) or complete (grade 3) (Table 15.5). With the metacarpal held stable, a valgus force is applied against the MCPJ in two positions: 30° of flexion and then in full extension (Fig. 15.34c). Whilst Posner & Retaillaud advocated testing in extension due to the false positive that may be obtained in individuals with ligament laxity [51]. If there is laxity of >30° in flexion (or >15° compared to the contralateral side), the proper collateral ligament is completely torn and surgery is required. If however, it is equivocal and there is less than 30° of laxity in full extension, then the

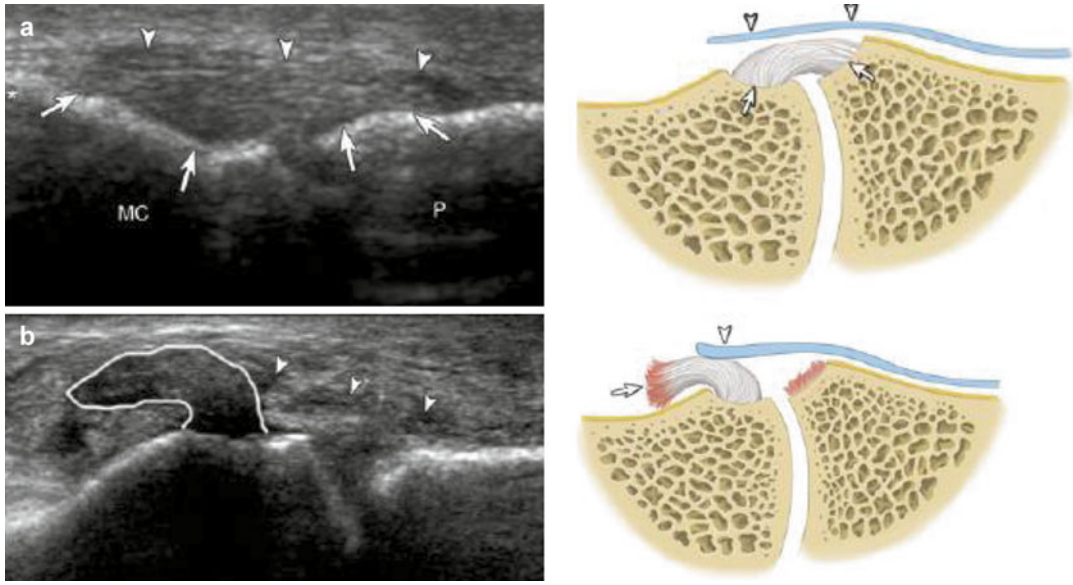


Fig. 15.35 (**a**) Image of an intact UCL with longitudinally oriented fibres which are hypoechogenic (*arrows*) (Taken with permission from Melville et al. [67]). (**b**) Image of a Stener lesion with the UCL retracted proximally by the Adductor edge (*arrow heads*) showing the lack of longitudinal fibres in the MCP area (Taken with permission from Melville et al. [67])

accessory collateral is likely to be intact and as such it is unlikely that the main collateral ligament is torn. If both are lax, however, a complete rupture has occurred and there is an 80 % chance of a Stener lesion being present [62]. Fracture of the proximal phalanx or MC shaft obviously precludes stress testing.

Ultrasound assessment has been growing in popularity and although initial studies found them not to be useful, later ones did. This has been due partly to the improved quality of the probes (10–17 MHz) which are now more sensitive and also a "tissue harmonic imaging" mode which enhances the image [66]. The points to look out for are an absence of normal longitudinal UCL fibres spanning the MCP joint and the presence of a heterogeneous mass-like abnormality (the torn UCL stump that has retracted or been flipped by the adductor aponeurosis) proximal to the MCPJ (Fig. 15.35) [67].

MRI may also be used to diagnose UCL ruptures and Stener lesions and is purportedly more accurate than US, however no recent studies have proven this. With the advent of super probes, US may eventually be used more in the emergency setting because it is cheap, non-invasive and dynamic. Clinical examination, however, should remain the mainstay of diagnosis.

Treatment Options

Grade 1 and 2 tears are treated with a custom made hand-based thermoplastic splint applied for a duration of 4–6 weeks. The IPJ is left free and gentle ROM exercises can be started immediately (with avoidance of stress to the UCL) and strengthening at 6 weeks depending on progress.

Surgical Treatment

Grade 3 tears require surgical intervention. Heyman found that 87 % of his complete tears diagnosed clinically were Stener lesions [62] Others have reported varying degrees of accuracy but generally a good clinical assessment suggestive of complete rupture demands surgical intervention.

An s-shaped skin incision is made centred over the thumb MCPJ (Fig. 15.36). Care is taken to avoid damage to the radial sensory nerve

Fig. 15.36 An S-shaped incision centred over the MCPJ. Applies to both UCL and RCL

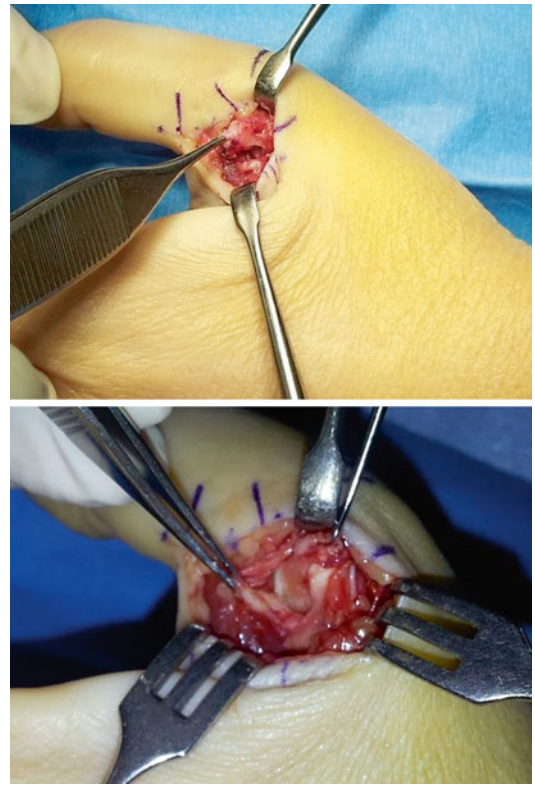


Fig. 15.37 The forceps is holding the torn portion of the UCL which has come off the MC head. The articular surface of the proximal phalanx can be seen

branches. The Adductor aponeurosis is divided and retracted to expose the torn ligament (Fig. 15.37). The UCL is then reattached to the proximal phalanx base (usual) with a bone anchor suture or if not available then with a transosseous bone tie. The former are easier to use and reduce surgical time (and complications!) [48]. Placement of the anchor on the proximal phalanx is usually at a point 3 mm distal to the articular surface and 3 mm dorsal to the volar cortex [48]. One limb of the suture is passed through the dorsal ligament then transversely to the volar side. The other suture limb is left free to allow it to slide and used to anchor the UCL once it is brought down to the bone and tied. The adductor is repaired over it with absorbable sutures. Stability is checked and a K-wire is inserted if there is concern or if initial joint displacement was significant (Fig. 15.38). Skin closure and immobilisation in a cast immediately after surgery is recommended. It may be changed to a heat molded plastic splint later in clinic. The wire can be removed in 4–6 weeks and ROM exercises started. Strength training is delayed to after 2 months.

Outcome and Lit Review

Studies on collateral ligament injuries are few and combine both RCL and UCL results, sometimes for both acute and chronic injuries with varying surgical techniques, thus giving a number of confounding conclusions. Despite that, outcomes are good especially for acute cases with good to excellent results in more than 90 % of cases as cited by Tang [48]. Problems arising are persistent pain, difficulty with activities and weakness. Chronic injuries fare worse and may warrant reconstruction or fusion.

Complications

Reduction in motion at the IP and MP joints is common, although this can be overcome with aggressive and regular therapy. Minor postoperative problems include numbness, superficial radial nerve injury and necrosis under the button pullout suture. More serious complications include persistent instability, decreased strength and reflex sympathetic dystrophy. Missed diagnosis in inexperienced medical practitioners may lead to chronic laxity and arthritis [59].

Fig. 15.38 (**a**) The MCPJ has been pinned with the ulnar side slightly tighter because the joint seemed a tad lax after repair. (**b**) The volar luxation seen in the earlier radiograph (Fig. 15.34b) has been corrected

Clinical Pearl

UCL injuries are well known and not often missed. Most clinicians are aware of the Stener lesion and look out for it. The key is an understanding of the clinical examination – inspection, palpation and movement (Table 15.6) -and its interpretation. Partial tears are not serious and have good outcomes. Complete tears need to be picked up and addressed surgically.

Radial Collateral Ligament Tear of the Thumb: Metacarpo-Phalangeal Joint

Epidemiology

Radial Collateral Ligament (RCL) injuries occur as a result of a fall on the outstretched hand with the thumb adducted rather than abducted. This will cause an abrupt adduction force to push the thumb ulnarly and volarly causing a dorso-radial tear. The frequency of these lesions comparing radial to ulnar according to Moutet 1:9 [60].

Table 15.6 Key points in management of Thumb UCL injuries

High index of suspicion and prompt diagnosis
Differentiate partial from complete tears and Stener lesions
Clinical examination:
Inspection – bruising, swelling and rotation/ subluxation
Palpation – site of tenderness and location if any, of lump
Movement – test the joint in 30° flexion and extension, compare
Radiography – look for volar subluxation, radial/ulnar deviation and avulsion fractures
Use US and MRI as adjuncts
Surgical approach: protect the nerves, identify the adductor aponeurosis, use bone anchors if available. Wire the joint if unstable
Protect with a thermoplastic splint

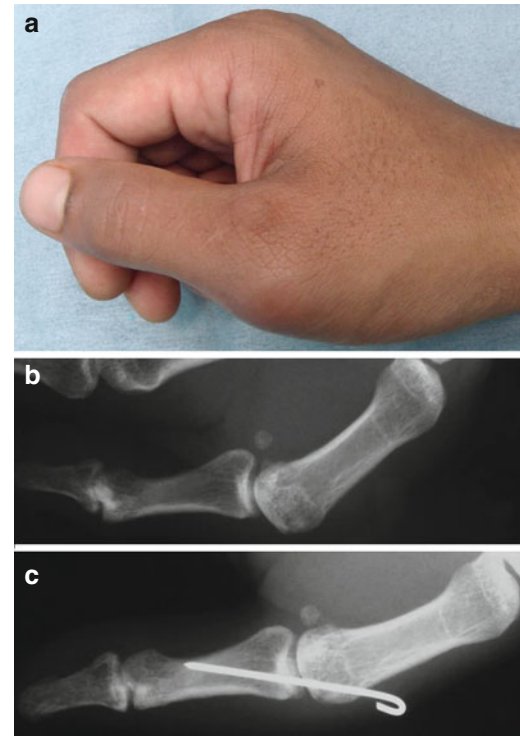


Fig. 15.39 (**a**) The thumb is in ulnar deviation, pronation and "dropped". There is a swelling on the radial side of the thumb MCPJ. (**b**) These features are confirmed on the radiograph below. No fracture is seen. There was a proximal tear of the RCL which was repaired and protected with the Kirschner wire to help reduce the joint (**c**)

Pathology

The site of avulsion is less predictable than UCL tears with a near equal distribution from the MC head and proximal phalanx [51, 54, 63]. This greater occurrence of proximal avulsion could be because the footprint of the RCL is relatively bigger on the phalanx [48]. A radial-sided Stener lesion rarely occurs because the Abductor aponeurosis is more dorsal covering the entire RCL [61].

Clinical Features and Diagnosis

The patient will present with pain, swelling (over the radial aspect of the MCP) and bruising. Ulnar deviation of the thumb is more commonly seen with RCL injury as is joint subluxation [48]. This is because the Adductor muscle inserts on the proximal phalanx at an angle of 48° and is more volar to the MP axis of rotation, exerting a significant force in the absence of the RCL [48]. The phalanx will go into pronation and will appear adducted (Fig. 15.39).

RCL injuries characteristically produce pain on ulnar deviation. An intra-articular anaesthetic may aid in the assessment. The classification based upon stress testing is given in Table 15.5. Testing with the MCP in flexion is assessing the main collaterals, whilst in extension it assesses the accessory collateral. An additional point, however, is that there is often significant volar subluxation (more so than in the UCL). This can be assessed using an anterior and posterior 'drawer' tests similar to that described in the knee. Specifically, both the phalanx and metacarpal are held firmly and moved in an anterior/posterior direction. Sometimes there may be up to 3 mm or more of translation.

Treatment Options

Grade 1 and 2 tears are usually treated with the application of a thermoplastic splint in extension for about a month [48]. Subsequently active and passive flexion-extension exercises are initiated with care (avoid ulnar stress). Grip and pinch strength can be started once the ligament heals at 6 weeks. Most incomplete tears will heal.

Surgical Treatment

Grade 3 tears require surgical intervention similar to the ulnar collateral ligament. In the absence

of a Stener-like lesion, this is justified particularly when there is significant joint displacement. Additional protection of the repair with a K-wire is warranted. Post-operatively a splint is applied for 4–6 weeks. This is followed by an appropriate rehabilitation programme, initially aimed at mobilizing the joint, but ultimately strengthening.

Outcome and Lit Review

Good to excellent results are seen with both conservative and surgical treatment in the acute setting. Coyle found 87 % of patients were symptom free, none had instability, 11 % had mild pain with heavy activity and only 8 % had a reduced grip strength [68].

Complications

Pain and continuing instability are the main concerns.

Clinical Pearl

The MCPJ of the Thumb is particularly prone to injury due to its exposed position. Forces in any direction may cause injury resulting in dorsal, volar, radial or ulnar displacement or dislocation. Pain and instability are the main disabling symptoms affecting pinch and grip, however repair and reconstructive options offer favourable results. If not, the thumb is particularly amenable to fusion with minimal loss of function (Fig. 15.32).

The Metacarpophalangeal Joint of the Fingers (MCPJ)

Introduction

The MCP joints of the hand are the fulcrum of finger motion. By being well embedded in the 'body of the hand' they have greater stability and less vulnerability to trauma (therefore less dislocations) as compared to the PIP and DIP joints. They can therefore support multiplanar motion allowing a wider degree of rotation and lateral motion to take place [6]. This mobility is provided by the design of the bony architecture and it's supporting soft tissues.

The volar side is well-padded and protected but the dorsal skin being thin exposes the knuckles (metacarpal heads) and extensor mechanism to injury especially when held in a grip. Human bites causing extensor tendon injuries, metacarpal damage and infection are commonly seen. Sagittal band disruption predisposes the extensor tendon to instability and a tendency to subluxate. Collateral ligaments here are also prone to injury especially the border digits (index and small fingers). However, contracture is an issue as well. Complete joint dislocation is uncommon.

Anatomy and Clinical Implications

The Metacarpophalangeal Joint (MCPJ) of the Fingers

Bony Architecture

The head of the metacarpal appears well-rounded on radiographs but it is quite asymmetrical. The MC head is narrow dorsally and widens palmarly to almost double the width, fitting neatly into the concave of the proximal phalanx. It is longer in the dorsal-volar than the proximal-distal plane, therefore as the finger flexes, the contact increases and the joint tightens to maximum in full flexion. Thus lateral motion and rotation is limited in flexion but is maximum in extension. The rotation of the MC head is different in each finger to allow convergence of all the digits towards the midline. Thus in the index, the head is rotated slightly ulnarly while in the ring and small fingers the rotation is slightly radial. Laterally, there are small depressions on the dorsal side of the head housing the origins of the collateral ligaments.

Joint Capsule

Similar to the DIP and PIP joints, **laterally** the *collateral ligaments* which may be up to 3 mm thick, run from the head of the metacarpal (MC) to the volar lateral base of the proximal phalanx, while the *accessory collateral ligaments* insert into the

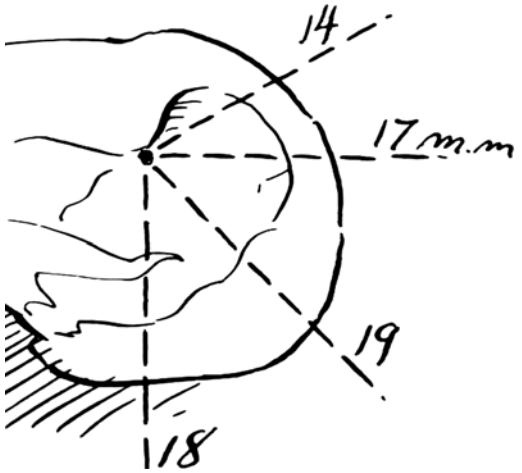


Fig. 15.40 The cam effect seen in the metacarpal head is important in splinting of the hand. As the finger moves more into flexion, the length of the collateral ligament increases, becoming maximum at approximately 50–60° of flexion (best to maintain collateral ligament length). If the finger is immobilised in extension, the collaterals will shorten and the joint will not be able to flex (Taken with permission from Glickel et al. [28])

volar plate [69]. In contrast to the PIPJ, the collaterals are lax in extension and tightened as they stretch over the wider volar base of the metacarpal head as the joint goes into flexion. This cam effect (Fig. 15.40) is an important point as a position of immobilisation after trauma or surgery must always be in the MCP joint in at least 50° of flexion so that the collaterals do not tighten in a shortened position. The latter will ultimately result in contracture and loss of flexion [28]. **Palmarly** the *volar plate (*VP) which sometimes houses a sesamoid bone is firmly attached distally to the P1 base with fibres from the accessory collaterals anchoring it laterally. Proximally there are no check-rein ligaments and attachment is at the neck, just proximal to the cartilaginous portion of the MC head and is membranous and flimsy allowing the 30° of hyperextension seen in most individuals. **Transversely**, the MC heads are further stabilised by ligaments that tether the adjacent volar plates together known as the deep transverse MC ligaments which insert together with the accessory collaterals into the PP and separate the lumbrical muscles (volar) from the interossei tendons (dorsal).

Extrinsic Joint Support

The intrinsic muscles of the hand provide support to the MCP joint especially in extension. The 4 dorsal interossei insert into the volar lateral tubercle of the P1 thereby acting as abductors of the fingers. In addition, the lumbricals and palmar interossei by inserting into the extensor expansion over the PIPJ, span from flexor to extensor surface and stabilise the MCPJs.

Extensor Retinacular System

A special mention of the Sagittal Bands (SB) must be made here for they are part of the retinacular system described in the PIPJ anatomy. The SB forms a cylindrical tube enclosing the MCPJ being composed of transverse and sagittal fibres around the MC head and oblique fibres more distally forming the triangular lamina. The extensor tendon itself is ensheathed in a superficial and deep layer of the SB, the latter being thinner over the two central fingers.

Dorsal Metacarpo-Phalangeal Dislocations

Epidemiology

Dorsal dislocations of the MCPJ occur primarily in the border digits (index more than small) as opposed to the protected central middle and ring fingers where only isolated cases have been reported [28]. Sedel in his study of posterior dislocations in 13 fingers found the distribution to be: index, five; middle, two; ring, one; and little finger, five, of which only two were open cases [49]. They are extremely rare as a comprehensive literature search by Kaplan revealed in 1957 [70]. The mechanism is usually a fall on the outstretched hand with the culprit finger being hyper-extended and displaced dorsally, the VP being avulsed from its proximal attachment. They are classified as simple when reduction is easily achieved; closed and complex if closed reduction fails, the latter being more common.

Simple MCP Subluxation

Subluxation differs from dislocation in that the volar plate is partially torn proximally and still draped over the metacarpal head. We prefer the *dislocation paradox* as described by Lattanza where the simple dislocation looks worse with the proximal phalanx in a vertical position over the 2nd MC and the IP joints in flexion [71]. The proximal phalanx is locked in 60–80° of hyperextension (Fig. 15.41) with some rotatory ulnar deviation towards the adjacent finger [70]. Radiographs show mild incongruity of the joint with normal joint space. Attempts at reduction by traction and hyperextension will convert the partial VP tear to a complete one with possible VP interposition and complex dislocation. Thus firstly the flexor tendons are relaxed by flexing the wrist, then the hyperextended P1 is compressed onto the MC head to prevent volar plate interposition [69]. Only then is reduction attempted by firmly applying dorsal pressure on P1 simultaneously flexing the joint into a reduced position. Extension block splinting for 3–4 weeks is sufficient to prevent redislocation [28].

Complex MCP Subluxation

Clinical Features and Diagnosis

The patient presents with an extended finger at the MCPJ and the distal joints are held in slight flexion (Fig. 15.42). Flexion of the MCP is painful and absent. There may be an obvious protrusion of the MC head producing a volar 'bump' and puckering of the adjacent skin [72]. Dorsally, just proximal to the P1 base, there is a hollow. The P1 is hyperextended and may be articulating with the dorsal part of the MC head. The index finger may displace radially and the little finger ulnarly [9] (Fig. 15.41). On radiographs, the joint space is markedly widened and the sesamoids may be seen within, indicating volar plate interposition pathognomonic of complex dislocations [73]. Lateral views may show osteochondral fractures of the MC head in up to 50 % of cases [28]. Brewerton's views are essential for they will show osteochondral fractures of the MC heads or P1 base. This is taken with the hand in the AP position i.e. palm facing upwards and the phalanges in contact with the film. The metacarpal-phalangeal joints are flexed to 45° with tube angled 20° (from an ulnar direction) aiming to the head of the third metacarpal (Fig. 15.43).

Fig. 15.41 Simple dorsal dislocation of the 5th MCPJ with the volar plate attached to the distal part (P1) as can be deduced by the sesamoid position. The finger is splayed ulnarly (Courtesy of a colleague, Dr. Ranjit Singh Gill)

Treatment Options

The pathogenesis is key to understanding treatment. During forceful hyperextension, the volar plate gets torn from the base of the MC head volarly and retracts intersposing between the volarly displaced MC head and the now dorsal P1. The MC head juts out through the torn VP and is trapped within a noose formed by the lumbrical muscle radially and the intact flexor tendons and their sheath still attached to the VP ulnarly (in the IF). Closed reduction is not possible because during longitudinal traction, the lumbricals and flexor tendons are pulled taut like a noose around the MC neck. A similar scenario occurs on the ulnar side but in this case, the MC head gets caught between the common insertion of abductor digiti minimi and flexor digiti minimi on the ulnar side and the flexor tendons and lumbrical muscle on the radial side. This button-hole effect again prevents closed reduction because traction tightens the 'noose' around the neck.

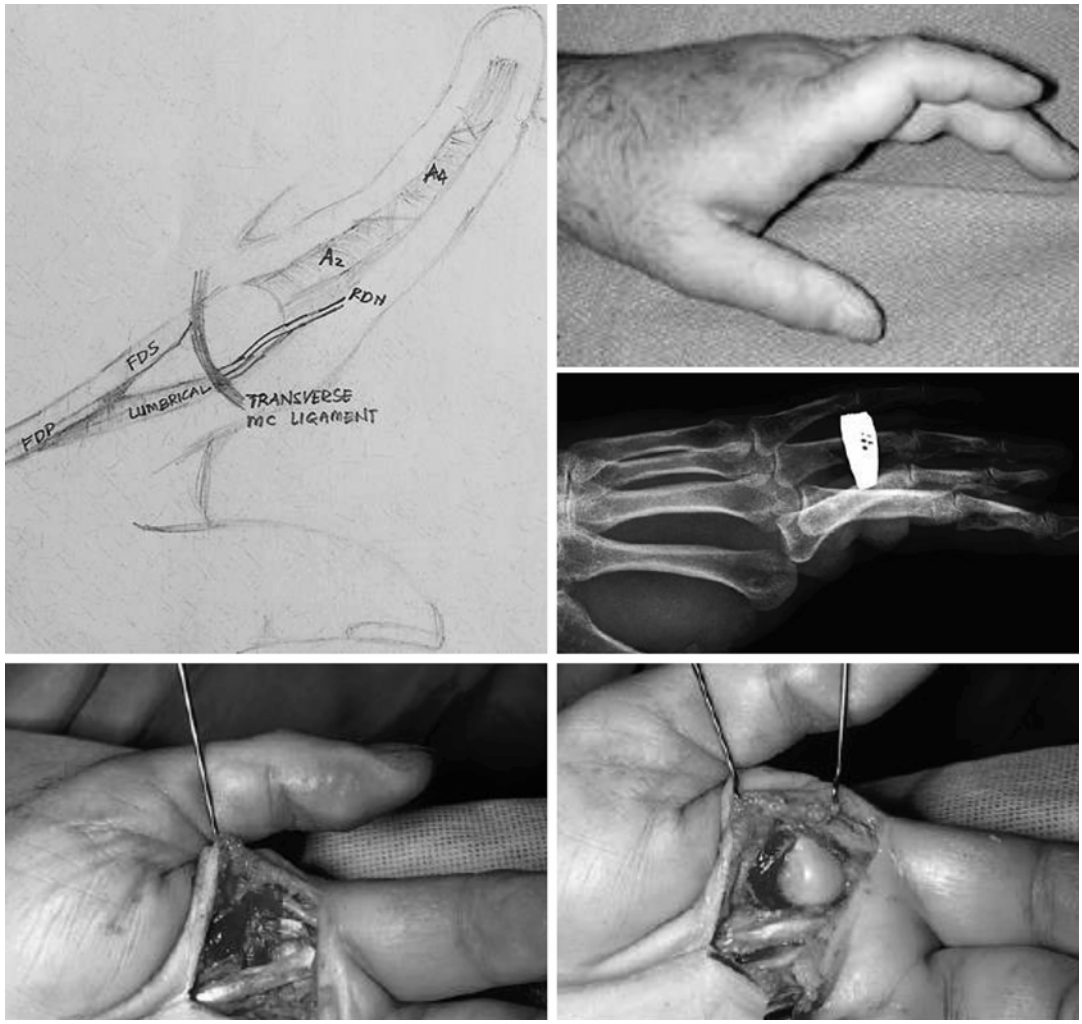


Fig. 15.42 **Clockwise**: *Top left* is a line drawing of a complex dorsal dislocation of the (R) index finger MCPJ showing the MC head in a 'noose' formed by the lumbricals radially, the FDP and FDS ulnarly and the transverse metacarpal ligament proximally at the neck. Applying traction on the finger would tighten the noose, preventing closed reduction. The radial neurovascular bundle may be cutting across the MC neck/head posing a risk during the volar approach. *Top right* is the clinical appearance. Below that is the radiological showing the proximal phalanx on top of the MC head in a bayonet position. Operative picture showing the MC head protruding through between the lumbricals radially and both flexor tendons medially. Dividing the flexor tendon sheath relaxes the flexor tendons enabling reduction which is usually stable (Taken from Beasley [73])

Surgical Treatment

There is ongoing debate over the most suitable approach (volar or dorsal) for this condition. In the **dorsal** approach described by Becton the dorsal capsule and extensor tendon are divided longitudinally exposing the volar plate dorsal to the head [74]. This is split into two halves and the proximal phalanx is then reduced onto the MC head. Any osteochondral fragment can be easily visualised and fixed. The other advantage of this approach is that it does not endanger the digital nerves, however volar structures are not easily accessed.

Caution must be exercised in the **volar** approach, for the digital nerves (especially the RDN) will be stretched over the volarly displaced MC head pushing it subcutaneously, making it

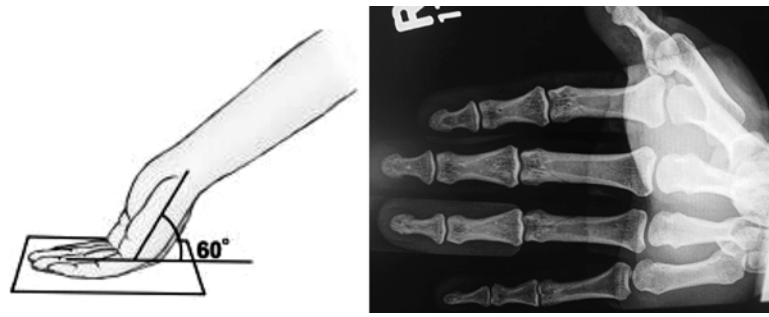


Fig. 15.43 The tube is angled at 20° from an ulnar direction and because it is focused on the third MC, the others are being viewed at a slight slant

vulnerable to injury. That is not the only concern, for although the volar plate is separating the MC head from P1, the actual culprit is the tight 'noose' formed by the flexor tendons and the lumbrical muscle around the MC neck. Traction only serves to tighten the noose. By dividing the A1 pulley as in a trigger finger release however, the flexors become lax and the volar plate can then be extricated and the joint reduced.

Once reduction is achieved, the joint is usually stable. Extension is avoided by immobilisation in a dorsal blocking splint with the MCPs held in 40–50° of flexion. Supervised gentle active ROM exercises may be started a week later, although a continuous passive motion machine (Fig. 15.44) is quite useful limiting ROM while allowing lubrication of the joint and remodelling of the supporting ligaments [69]. Table 15.7 summarises the key points to bear in mind.

Outcome and Lit Review

There are no series documenting outcomes but reports on 4 or less cases comparing the surgical approaches. Most say pre-injury ROM is achieved in approximately 2 months if prompt diagnosis and appropriate treatment is instituted. Complications arise as a result of delay.

Complications

The most common and disabling complication is joint stiffness which can be avoided by an aggressive therapy programme. Trauma to the articular surface during repeated attempts at closed reduction or during open reduction may lead to traumatic arthritis as would a delay in treatment or failure to diagnose an osteochondral fracture. Osteonecrosis of the MC head and in children premature closure of its physis has been reported. During the volar approach in open reduction, there is a possibility of damage to the radial (index) and ulnar (small finger) neurovascular bundles respectively due to their superficial position. Late reduction, prolonged immobilisation or excessive adjacent soft tissue damage may induce fibrosis resulting in a stiff joint.

Clinical Pearl

MCP dislocations are not common hence prompt diagnosis with timely intervention is of utmost importance in preventing stiffness, which is the most common and disabling complication. Although open reduction is the norm, care must be taken to identify the simple dislocation and not convert it to a complex one! Usual structures obstructing reduction are the volar plate, torn ligaments and the 'noose' around the metacarpal head. The dorsal approach is logical if there is an osteochondral fragment, otherwise the volar approach is preferable since all the relevant structures are visible and division of the volar plate or other vital structures is not required.

Volar Metacarpo-Phalangeal Dislocations

These are half as common and a reverse of the dorsal dislocation with interposition of the dorsal capsule into the joint [49]. Others structures reported have include the volar plate, the collaterals

Fig. 15.44 Hand, wrist and elbow CPM machines are useful adjunct to have in a hand unit. Our therapy centre has the full range up to the shoulder – which aids in preventing stiffness to the unaffected joints as well

Table 15.7 Key points in management of Finger MCP dorsal dislocations

High index of suspicion and prompt diagnosis
Differentiate simple (extended) from complex (bayonet) dislocations
Take care not to convert simple to complex – see text
Dorsal approach – if you suspect a fracture/ osteochondral fragment
Volar approach – identify and protect the neurovascular bundle
Range of motion exercises can be started within a week in a protected but aggressive therapy programme

and in border digits the juncturae tendinum. Closed reduction under anaesthesia is possible [75] otherwise a dorsal approach is advised.

Lateral Metacarpo-Phalangeal Dislocations

Epidemiology

Lateral subluxations (and their spontaneous or self-induced reduction) are more common than

the more severe dorsal dislocations, the mechanism of injury being forced radio/ulnar deviation with the MCP flexed. In flexion, the **collaterals** tighten and forced abduction-adduction or a twisting action with resulting strain may produce a **tear** or **avulsion** of the attachment, including injury to the accessory collaterals [69].

As athletics and contact sports become more competitive, radial collateral ligament (RCL) injuries are becoming increasingly more common, particularly when compared to the ulnar collateral ligament injuries of the finger MCPJs [28]. Usually, the central digits are 'protected', but suffer only partial tears with chronic pain being more of an issue than instability [73]. In a Belgian study, Delaere et al. published that 1 in 1,000 hand injuries involve the MCP collaterals. Of those requiring surgery, 39 % involved the finger as compared to 61 % the thumb [76]. Interestingly, all 7 RCL injuries were in the ulnar 3 fingers and all 5 UCL injuries were in the radial 2 fingers. They reported no index finger RCL injuries in contrast to more recent publications where 14 and 12 cases solely of Index RCL were reviewed [77, 78]. Classification of these tears is as described in Table 15.3 [51].

Clinical Features and Diagnosis

Patients may present late because the injury and resulting disability is mild, particularly with pain and swelling as opposed to instability. The collaterals are relaxed in MCP extension allowing a great deal of abduction-adduction. Therefore the joint is examined in flexion, starting with the normal contralateral digit to which tension is compared. Tenderness at the origin (MC head) or insertion (P1 volar base) of the ligament suggests an avulsion; there are conflicting reports as to which is more common. This must be differentiated from intrinsic tendinitis where pain is elicited by stretching the intrinsic [69]. Lateral views may show an avulsion fracture while AP and Brewerton's views may reveal intra-articular fractures of the metacarpal heads or phalangeal bases with joint incongruity.

Treatment Options

Grade 1 and 2 tears are usually treated with splinting – application of a thermoplastic splint in 45–60° of MCP flexion for 3 weeks [69]. Subsequently active flexion-extension exercises are initiated with the finger buddy taped for 'protection'. Most incomplete tears will heal. Chronic pain may still occur, especially when the joint is mobilised but local anaesthetic and steroid injection at the point of tenderness usually provides relief [73]. Casting has also been suggested but in tropical climes this may be uncomfortable and perhaps an overkill. When chronic pain does not resolve with the above methods, surgical release of scar tissue and repair of the collateral ligament may be required [69].

Surgical Treatment

When there is an articular fracture, joint incongruency, significant instability or laxity without an endpoint, surgical intervention is indicated. Prompt repair by a lateral or dorsal approach is advised. The injured collateral ligament is exposed by dividing along the lateral part of the extensor tendon and dorsal capsule. A mini suture anchor is embedded into the bone at the avulsion site and the two pre-loaded sutures are used to tie down the collateral ligament with the joint held in 45° of flexion [78]. The repair is then stressed through a range of motion and the wound closed in layers. The hand is splinted as described before. Motion is started in 3–4 weeks.

Outcome and Lit Review

Good results have been reported with Grade 1 and 2 tears, early presentation, prompt treatment and a good therapy regime. Delays in diagnosing or instituting treatment especially for Grade 3 tears carries a poor prognosis, resulting in permanent stiffness and loss of motion. Arthritis seems to develop in approximately 66 % of cases [77].

Complications

Undiagnosed or untreated lesions be they partial or complete will have pain, swelling and instability. In complete rupture (of the RCL for example) the finger may 'drop', pronate and even scissor the adjacent digit [77]. In some, there may be associated rupture of the dorsal interosseous attachment (similar to a Stener lesion) resulting in chronic pain and swelling. Gaston et al. in 14 patients, noted that 9 had grade 3 tears and of these 1 had mild degenerative change and 3 had

significant symptoms, two requiring fusion [77]. A good therapy regime and close follow-up is required to prevent stiffness and extension contracture of the MCP joint. Chronic regional pain syndrome has been reported [74].

Clinical Pearl
Collateral ligament disruptions although considered rare are not so. They may have an innocuous presentation but may be quite debilitating if left untreated. We would offer surgery to Grade 3 tears and conservative for the lesser ones.

Sagittal Band Disruptions

Epidemiology

The sagittal bands (SB) form part of the extensor retinacular system encircling the MCP head and volar plate [79]. They aid in stabilising the extensor digitorum tendon over the MC head. Due to its prominence, the heads are prone to injury especially when making a fist. The middle finger being the longest and most prominent has the highest incidence of injury. In an interesting study undertaken by Young and Rayan, they sectioned the SB to assess the effect on extensor tendon mobility. Sectioning the radial SB resulted in subluxation, particularly when the wrist and the MCP joints were flexed. The effect was least evident when the wrist was extended [80]. Conversely when the ulnar SB was sectioned this had little effect. This showed that the radial SB is particularly important in preventing ulnar subluxation of the extensor digitorum tendon and should be repaired primarily.

Clinical Features and Diagnosis

Damage to the extensor hood at the MCPJ results in instability of the extensor tendon and presents with snapping, pain and weakness. The radial sagittal band (SB) is usually affected because its fibres are thin and long as opposed to the ulnar SB fibres. The patient would complain of extensor tendon instability on making a fist. In acute cases, the injury is obvious, particularly when open (Fig. 15.45).

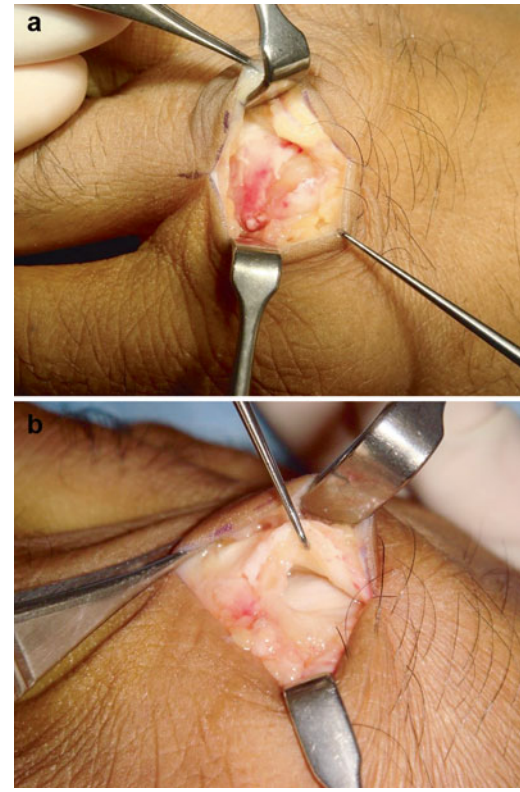


Fig. 15.45 (**a**) The right hand of an O&G surgeon showing a tear of the sagittal band between the Index and Middle metacarpal heads. (**b**) The skin hook is lifting up the ulnar component showing the capsule underneath. Occupational disaster for this will not heal if he continues to perform digital vaginal examinations or surgery. Post repair immobilization in a buddy splint for 6 weeks allowed healing and full ROM

Treatment Options

If the injury is acute, closed and presents within 2 weeks, a trial of extension splinting may be used. A word of caution though – the diagnosis must be made with certainty for if the extensor is cut, it may not heal.

Surgical Treatment

If the above conservative treatment fails, or there is an open wound, we prefer to debride and use an absorbable suture to repair the tendon burying the knots. The latter diminishes irritation and reduces the likelihood of stitch granuloma.

An extension splint is then used to rest the hand for 2 weeks. Thereafter, Buddy splinting to the adjacent ulnar digit is recommended. Active motion can be started at 3–4 weeks.

Outcome and Lit Review

Most literature describes results for reconstruction of sagittal bands and not primary repair.

Complications

In case of an open injury, infection remains a concern. Thorough debridement in the first instance is essential. Stiffness is something that can and should be avoided by starting therapy early.

Clinical Pearl

Repair of acute injuries to the sagittal bands is straight forward and usually depends if the injury is closed or open. The latter would obviously necessitate surgical repair.

The Carpometacarpal Joint of the Fingers (CMCJ)

Introduction

The distal carpal row and CMC joints of the fingers form **the base** of the (fixed) transverse metacarpal arch, while the two longitudinal arches – radial thumb and ulnar ring and small remain mobile. Therefore, although injuries to these joints are rare, as Bunnell remarked "reduction of CMC dislocation is necessary to restore muscle balance and proper mechanics" [81].

The index and middle fingers (2nd and 3rd MC) are relatively fixed at the base and allow grip and fine pinch, whilst the 4th and 5th metacarpals are mobile in the sagittal plane (5–10° and 20–30° respectively) allowing easier grasp by reducing the transverse diameter [82]. The metacarpal bases and CMC joints are held together by strong ligaments, hence force transmitted often tends to cause fractures rather than pure dislocations. The 4th and 5th rays are more commonly affected than the 2nd or 3rd. Usually, dorsal dislocations are more common than volar.

Anatomy and Clinical Implications

The Carpometacarpal Joint (CMCJ) of the Fingers

Bony Architecture

We know the distal carpal row forms a tightly-knit transverse base for the metacarpals to articulate with. The odd numbered metacarpals (one, three and five) articulate with one carpal bone only (trapezium, capitate and hamate respectively) while the 2nd MC articulates with the trapezium, trapezoid and capitate and the 4th MC articulates with both the capitate and hamate [83]. The 3rd and 4th metacarpals have medial and lateral collateral facets to articulate with their adjacent MCs while the 2nd (medial facet) and 5th (lateral facet) have only one facet each. It is these articulations that determine the shape of the base of the MCs (Fig. 15.46).

The 2nd MC base has a forked shaped design that locks into the trapezium and trapezoid. It has 3 superior facets and two lateral facets. The extensor carpi radialis longus (ECRL) attaches to its dorso-radial surface while the flexor carpi radialis (FCR) attaches on the volar side with a slip to the 3rd MC base [84]. The 3rd MC base

Fig. 15.46 The outline of the carpal bones (distal row) and their corresponding metacarpals drawn to show their articulations

has 2 lateral facets for the 2nd (lateral) and 4th (medial) metacarpals and one distal facet for the capitate. The extensor carpi radialis brevis attaches on its dorsal surface. The 4th MC base is quadrangular and superiorly it articulates with the capitate (radially) and sits flat on the hamate (ulnar); it has no tendinous attachments. The 5th MC base is also quadrangular articulating with the hamate's lateral facet obliquely. Dorsally there is a tubercle for the extensor carpi ulnaris (ECU) tendon and volarly the flexor carpi ulnaris (FCU) attaches via the piso-metacarpal ligament.

Joint Capsule

The 4 distal carpal bones form a tightly knit base for the medial metacarpals. The CMC joints are stabilised dorsally by six strong ligaments and volarly by six weaker ligaments. In addition, four inter-metacarpal ligaments bind the bases together forming a strong union thus making fracture more likely than pure dislocation.

Extrinsic Joint Support

All the wrist extensors (ECRL, ECRB and ECU) and flexors (FCR and FCU) attach to the bases of the metacarpals as described, providing dynamic support and strengthening the CMC joints.

Dorsal Carpometacarpal Dislocations

Epidemiology

CMC joints are involved in less than 1 % of all injuries to the hand and wrist [85]. Motor vehicle accidents with high impact to the hand can cause multiple fracture dislocations, although these can be overlooked, particularly if they are more severe life threatening injuries. As such, it is not uncommon that they are diagnosed late. Dorsal dislocations account for approximately 85 % of all CMC dislocations whilst the most frequently injured joint is the 5th CMC.

Pathology, Mechanism and Classification

A Karate blow can cause fracture dislocation of the 4th and 5th CMC joints. Rapid deceleration along with a high energy impact can cause all the CMC joints to dislocate. The mechanism includes stabilisation of the hand by a fixed object (handlebars on motorbike or steering wheel in a car) and a significant deceleration force pushing the metacarpals dorsally. Sedel classified dorsal CMC dislocations according to the metacarpals involved (Table 15.8). Treatment follows the classification.

Table 15.8 Dorsal dislocations of the CMC joints

Type 1: dislocation of the fixed MCs (2nd and 3rd)
Type 2: Dislocation of the mobile MCs (4th and 5th) in which mobility must be preserved.
Type 3: All MCs displaced. Are the interosseous and inter-metacarpal ligaments intact or not?

Clinical Features and Diagnosis

The patient is typically a young male (average age was 25 in one series) involved in a motor vehicle accident [82]. They present with extreme swelling and pain of the affected joints with significantly reduced motion. The 5th CMC joint is, however, closed to the motor branch of the ulnar nerve and the latter may by injured either by traction or compressed by swelling. Similarly, on the radial side of the hand, swelling around the 2nd to 4th CMC joint can compress the median nerve. As such, it is important that a thorough neurovascular examination is undertaken. Added to that, the wrist flexors and extensors should also be evaluated, as they insert close to the CMC joints (ECRL on the 2nd MC base, ECRB on the 3rd MC base and ECU and FCU on the 5th MC base). The mainstay of diagnosis, however, is radiological examination.

An antero-posterior film will show overlapping of the CMC joints or lateral displacement (Fig. 15.47). Careful observation of the outline of the CMC joints should be undertaken and any incongruity identified. A lateral view decides the type of dislocation – dorsal or volar. The second and third CMCs are best visualised in a 30° lateral supinated view while the 4th and 5th CMCs are best viewed in 30° lateral pronated views [28].

Associated fractures of the carpal bones, especially the hamate and the metacarpals – 4th and 5th are sometimes seen and should be identified (Fig. 15.48). Finally, a more detailed analysis requires a CT scan.

Fig. 15.47 Antero-posterior and lateral views of a patient with 2nd, 3rd and 4th CMC volar dislocations. Even the first is subluxed. There is a small chip fracture off the dorsal capitate (Pictures courtesy of Dr. Rajesh Singh)

Treatment Options

If the injury is acute, closed and presents within 2 weeks, a trial of closed reduction and external casting or splinting can be tried. It is, however, important to monitor this, particularly in the initial phase (first 24 h) as neurovascular complications can ensue. Further to this, follow-ups should be regular and detailed as re-displacement is not uncommon. The latter occurs more frequently in the 2nd and 3rd CMC joints, as a result of the dorsal pull of the ECRL and ECRB tendons. The 4th and 5th CMCs are usually stable post-reduction [82].

Surgical Treatment

The failure of conservative treatment or if there is an associated fracture necessitates open reduction and internal fixation, usually involving k-wires. Depending on the number of dislocations, a dorsal approach is recommended, with preservation of the sensory nerves. It is important to note that reduction may have been prevented by entrapment of a torn inter-metacarpal ligament, sometimes a tendon. These need to be removed to allow reduction to occur. Thereafter, this is held and fixed by k-wires, crossing from the metacarpal to the carpal bones. To that, any obvious torn ligament can also be repaired. Any large fracture fragments can also be separately fixed by either using a wire or a cannulated screw (Fig. 15.48). We prefer to use the latter as they provide a strong fixation, and are buried within the bone.

Outcome and Lit Review

Due to the paucity of reports, there are no clear outcome figures [82]. Generally, closed reduction, if it succeeds, yields good results. As stated previously, however, entrapped torn ligaments or tendons can prevent reduction and these need to be removed through an open technique [28]. The joint should also be fixed after reduction to ensure that the latter is maintained. If this is not

Fig. 15.48 This is what is known as the reverse Bennett's fracture [84]. There is a fracture base of the 5th MC with a proximal displacement of the 5th MC being pulled by the compressive forces of the ECU, FCU and also the abductor digiti minimi while a small radial fragment is held in place, still attached to the 4th MC. The shortening of 4 mm can be seen by looking at the MC heads. On the right, post-fixation, articular congruity has been restored; some length has been regained as well as a clear picture of the 5th CMC joint obtained

undertaken, the pull of wrist extensors can result in redislocation. Arthritis is a late complication of reduced or congruent CMC joints. This is treated either by interpositional fascial arthroplasty, or arthrodesis [28].

Complications

Apart from neurovascular complications, the principle cause of concern is redislocation. Initially, this is detected by clinical suspicion (a grossly swollen hand) and, thereafter, radiological examination. A CT scan would be particularly helpful. Long term complications include particularly arthritis and continuing pain affecting the joint. Here, either an interposition arthroplasty or arthrodesis are the main options. Ideally, the 2nd and 3rd metacarpals do better with fusion and the 4th and 5th with interpositional arthroplasty as some movement is maintained.

Clinical Pearl

As is the case with most injuries of the hand, early diagnosis and prompt treatment usually results in a good outcome. A thorough clinical examination is essential, supported by a plain x-ray or CT scan as necessary. Simple injuries can be treated by closed reduction, then mobilization. More complex injuries including fracture dislocations usually require open reduction and fixation. Post-operative monitoring is recommended.

The Thumb Carpometacarpal Joint: (CMCJ)

Introduction

The thumb's wide-ranging mobility is primarily due to the CMC joint which has a biconcave saddle design at which the first metacarpal base and the trapezium interlock. In a resting position, the joints are congruent, but with opposition there is rotation and twisting of the MC base upon the trapezium with resulting incongruency and potential instability. This is prevented by a multitude of strong ligaments, most notable of which is the anterior oblique (AOL) [86]. The purpose of this chapter is firstly to outline the relevant clinical anatomy, describe the clinical findings and finally to summarise the treatment options for the acute injury. Chronic instability which is probably more common will be dealt with elsewhere. Currently, the treatment algorithm is unclear, but ranges from closed reduction and casting to fixation with wires and bone anchors, through to reconstruction of the ligaments and dorsal capsulorraphy [87].

Anatomy and Clinical Implications

The Carpometacarpal Joint (CMCJ) of the Thumb

Bony Architecture

The trapezium is a quadrangular shaped bone directly in contact with the 1st MC base. The surface is convex from dorsal to palmar and concave from radial to ulnar. The base of the first metacarpal is a mirror image, although there are varying degrees of congruity. In women the surfaces are flatter and less congruent, hence less stable and more prone to injury.

Joint Capsule

The biconcave saddle joint of the 1st CMC has little inherent stability. The principle stabilizer is the joint capsule which is reinforced dorsally by the APL insertion. There are also four ligaments which assist in maintaining stability [88]:

- anterior oblique ligament (AOL – which passes from the tubercle of the trapezium to the volar beak of the 1st MC),
- posterior oblique ligament (POL – from a dorso-ulnar eminence on the trapezium to the MC base),
- dorsoradial cmc ligament – from the dorsoradial eminence on the trapezium to the opposing side of the MC base and
- inter-metacarpal ligament which supplements the dorsal CMCJ capsule.

These ligamentous-capsular structures allow motion in six degrees of freedom (flexion, extension, abduction, adduction, pronation, supination) whilst maintaining stability.

Extrinsic Joint Support

In addition to the ligaments, there are tendinous insertions and muscular structures which on contracture also enhance the stability of the CMC joint [88]. These include, abductor pollicus longus (APL), flexor carpi-radialis (FCR) and extensor carpi-radialis longus (ECRL) tendons.

First Carpometacarpal (Dorsal) Dislocations

Epidemiology

Where 1st CMCJ arthritis is common, joints dislocations are rare. Only one volar dislocation has been reported in the English literature otherwise (almost) all are dorsal [89]. The mechanism of injury is an axially directed force (dorsal) on the partially flexed thumb pushing the metacarpal base out of joint at its weakest point (dorso-radial). This occurs because the volar ligaments are much stronger than the thin dorsal capsule. It was earlier credited that the primary restraint to this dislocation is the stout AOL but later shown that the dorsal capsule and dorso-radial ligament are key to preventing dorsal dislocation [90]. Thus the conclusion is that the dorso-radial ligament has to be torn for a complete dislocation to occur [28].

Clinical Features and Diagnosis

CMC ligamentous injuries may be complete or partial. The reason that this injury is rarely reported in the literature is because partial tears are missed while complete tears with obvious dorsal dislocation are not common. Thus the more extensive the tear, the more obvious the

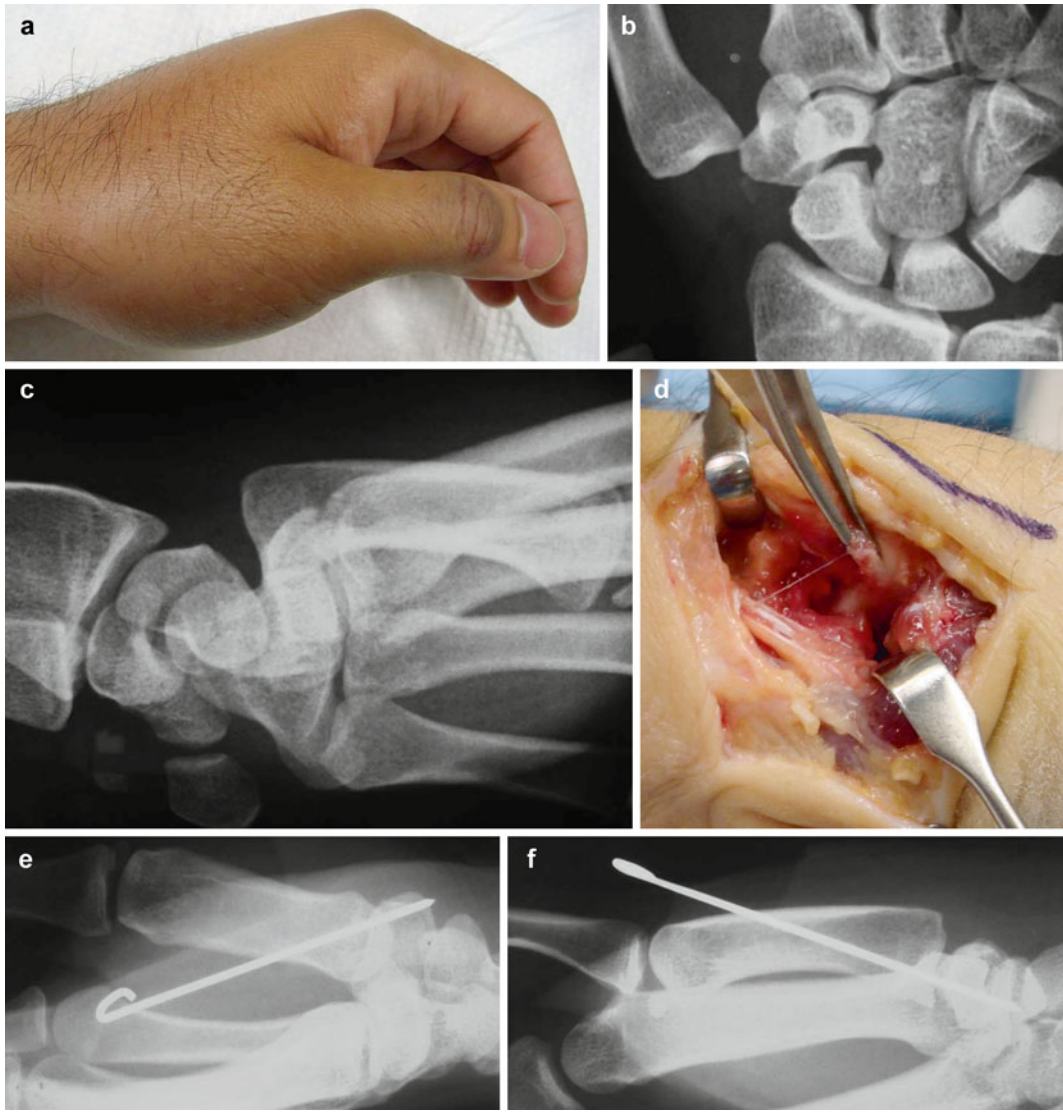


Fig. 15.49 (**a**) The base of the thumb ray is significantly swollen and as can be expected, the ROM reduced. (**b**) The thumb MC is obviously dislocated. (**c**) The dislocation is dorsal with a chip fracture off the trapezium. (**d**) Upon surgical exploration, the dorsal capsule and dorsoradial ligament was torn (held by the forceps) exposing the joint. (**e**, **f**) Repair of the structures with pinning to hold the joint stable resulted in a good outcome

dislocation and the more likely that it is treated (Fig. 15.49d). Patients present with extreme swelling and pain of the affected joints with significantly reduced motion. Less severe tears may present with mild swelling, although clinical examination, particularly stressing, will produce dorsal subluxation with pain. An x-ray should always be undertaken, as these injuries can be associated with fractures, particularly with Bennett's.

Treatment Options

If the injury is acute, with no obvious dislocation (mild subluxation of the joint) and presents within 2 weeks, a trial of closed reduction with external casting or splinting can be tried. The joint may also be fixed temporarily by a transarticular k-wire. If, however, there is some displacement and particularly if closed reduction fails, then surgery should be considered.

Surgical Treatment

Failure of conservative treatment or if there is significant initial displacement necessitates open reduction and repair of the dorso-radial ligament and capsule. We prefer to immobilize the joint with a Kirschner wire to maintain reduction at least initially (Fig. 15.49e, f).

Outcome and Lit Review

Due to the rarity of this injury, literature consists of anecdotal case reports only. In a comprehensive review by Fotiadis et al., there were conflicting views on the outcomes of conservatively treated patients; conversely early ligamentous reconstruction yielded good to excellent results with long-term stability [87]. Even so they cautioned that the surgical modality should not to be undertaken lightly for it is not a 'superfluous' option. They concluded that whilst initially non-operative treatment is appropriate, if a congruous reduction is not achieved then surgery is recommended.

Complications

If there is a basal 1st metacarpal fracture, this precludes a volar ulnar ligament injury. If not, undetected disruption of this ligament causes varying degrees of hypermobility with or without pain. Arthritis of the first CMC joint is a more common diagnosis. Particularly affecting postmenopausal women [86]. With this disease process, the base of the metacarpal can sublux dorso-radially, with associated adduction of the 1st metacarpal and hyperextension of the metacarpophalangeal joint [86].

Clinical Pearl

Partial tears we believe may be treated conservatively with closed reduction and splinting the thumb in an extended position. For a complete tear of the ligaments/dorsal capsule with significant joint displacement however, we feel that operative intervention with repair of the affected ligamento-capsular structures is essential to maintain long term stability. Any surgical intervention should focus on repair of the dorsal structures as opposed to reconstruction of the volar ligament which is more demanding and may not yield the desired outcome.

References

1. The McGraw-Hill Companies Incorporation. McGraw-Hill concise dictionary of modern medicine. The Free Dictionary, Farlex; 2002 [cited 4 Feb 2012]. Available from: http://medical-dictionary.thefreedictionary.com/instability.
2. Clayton RAE, Court-Brown CM. The epidemiology of musculoskeletal tendinous and ligamentous injuries. Injury. 2008;39:1338–44.
3. Mall NA, Carlisle JC, Matava MJ, Powell JW, Goldfarb CA. Upper extremity injuries in the national football league. Part I: hand and digital injuries. Am J Sports Med. 2008;36(10):1938–44.
4. Gigis PI, Kuczynski K. The distal interphalangeal joints of human fingers. J Hand Surg Am. 1982;7(2):176–82.
5. Schmidt HM, Lanz U. Chapter 9. Finger joints. In: Schmidt H-M, Lanz U, editors. Surgical anatomy of the hand, translation of 2nd German edition. Stuttgart: Thieme; 2004. p. 205–9.
6. Craig SM. Anatomy of the joints of fingers. Hand Clin. 1992;8(4):693–700.
7. Kapandji IA. Biomechanics of the interphalangeal joints of the thumb. In: Tubiana R, editor. The hand, vol. 1. Philadelphia: WB Saunders; 1981. p. 188–90.
8. Barmakian JT. Anatomy of the joints of the thumb. Hand Clin. 1992;8(4):683–91.
9. Abouna JM, Brown H. The treatment of mallet finger the results in a series of 148 consecutive cases and a review of the literature. Br J Surg. 1968;55(9):653–67.
10. Brzezienski MA, Schneider LH. Extensor tendon injuries at the distal interphalangeal joint. Hand Clin. 1995;11(3):373–86.
11. Stark HH, Boyes JH, Wilson JN. Mallet finger. J Bone Joint Surg Am. 1962;44A(6):1061–8.
12. Jones N, Peterson J. Epidemiologic study of the mallet finger deformity. J Hand Surg. 1988;13(3):334.
13. Warren R, Kay N, Norris S. The microvascular anatomy of the distal digital extensor tendon. J Hand Surg. 1988;13(2):161–3.
14. Kontor JA. Extensor tendon injuries and repairs in the hand. Can Fam Physician. 1982;28:1159–63.
15. Palmer RE. Joint injuries of the hand in athletes. Clin Sports Med. 1998;17(3):513–31.
16. Doyle JR. Extensor tendons – acute injuries. In: Green DP, Hotchkiss RN, Pederson WC, editors. Green's operative hand surgery. 4th ed. New York: Churchill Livingstone; 1999. p. 1950–87.
17. Lenzo SR. Distal joint injuries of the thumb and fingers. Hand Clin. 1992;8(4):769–75.
18. McFarlane RM, Hampole MK. Treatment of extensor tendon injuries of the hand. Can J Surg. 1973;16:366–75.
19. Garberman SF, Diao E, et al. Mallet finger: results of early vs delayed closed treatment. J Hand Surg. 1994;19(5):850–2.
20. Stern PJ, Kastrup BS. Complications and prognosis of treatment of mallet finger. J Hand Surg. 1988;13A:329–34.

21. Tubiana R. Surgical repair of the extensor apparatus of the fingers. Surg Clin North Am. 1968;48: 1015–31.
22. Bendre AA, Hartigan BJ, Kalainov DM. Mallet finger. J Am Acad Orthop Surg. 2005;13:336–44.
23. Din K, Meggitt B. Mallet thumb. J Bone Joint Surg Br Vol. 1983;65B(5):606–7.
24. Tabbal GN, Bastidas N, Sharma S. Closed mallet thumb injuries: a review of the literature and case study of the use of magnetic resonance imaging in deciding treatment. Plast Reconstr Surg. 2009;124(1):222–6.
25. Miura T, Nakamura R, Torii S. Conservative treatment for a ruptured extensor on the dorsum of the proximal phalanges of the thumb (mallet thumb). J Hand Surg Am. 1986;11(2):229–33.
26. Thayer DT. Distal interphalangeal joint injuries. Hand Clin. 1988;4(1):1–4.
27. Shah SR, Bindra R, Griffin JW. Irreducible dislocation of the thumb interphalangeal joint with digital nerve interposition: case report. J Hand Surg. 2010;35A(3):422–4.
28. Glickel SZ, Barron OA, Catalano III LW. Dislocations and ligament injuries in the digits. In: Green DP, editor. Green's operative hand surgery, vol. 1. 5th ed. Philadelphia: Elsevier/Churchill Livingstone; 2005. p. 343–88.
29. Benke GJ, Stableforth PG. Injuries of the proximal interphalangeal joint of the fingers. Hand. 1979;11(3): 263–8.
30. Dubousset JF. The digital joints. In: Tubiana R, editor. The hand, vol. 1. Philadelphia: WB Saunders; 1981. p. 197–201.
31. Leibovic SJ, Bowers WH. Anatomy of the proximal interphalangeal joint. Hand Clin. 1994;10(2):169–78.
32. Kuczynski K. The proximal interphalangeal joint. J Bone Joint Surg. 1968;50B(3):656–63.
33. Vicar AJ. Proximal interphalangeal joint dislocations without fractures. Hand Clin. 1988;4(1):5–13.
34. Allison DM. Anatomy of the collateral ligaments of the proximal interphalangeal joint. J Hand Surg Am. 2005;30A(5):1026–31.
35. http://eORIF.com/WristHand/PIP.html
36. Freiberg A, Pollard B, Macdonald MR, Duncan MJ. Management of proximal interphalangeal joint injuries. Hand Clin. 2006;22:235–42.
37. Liss FE, Green SM. Capsular injuries of the proximal interphalangeal joint. Hand Clin. 1992;8(4):755–68.
38. Freiberg A. Management of proximal interphalangeal joint injuries. Can J Plast Surg. 2007;15(4):199–203.
39. Elson RA. Rupture of the central slip of the extensor hood of the finger: a test for early diagnosis. J Bone Joint Surg Br. 1986;68:229–31.
40. Burton RI, Eaton RG. Common hand injuries in the athlete. Orthop Clin North Am. 1973;4(3):809–38.
41. Redler I, Williams JT. Rupture of a collateral ligament of the proximal interphalangeal joint of the finger. J Bone Joint Surg. 1967;49A:322–6.
42. Boyes JH. Special situations affecting tendons. In: Bunnell's surgery of the hand. 5th ed. Philadelphia: JB Lippincott Company; 1970. p. 436–48.
43. Aubriot JH. Chapter 14. The metacarpophalangeal joint of the thumb. In: Tubiana R, editor. The hand, vol. 1. 1st ed. Philadelphia: WB Saunders; 1981. p. 184–7.
44. Rondineli RD. American Medical Association; guide to the evaluation of permanent impairment. 6th ed. Chicago: American Medical Association Press; 2007.
45. Miller RJ. Dislocations and fracture dislocations of the metacarpophalangeal joint of the thumb. Hand Clin. 1988;4(1):45–65.
46. Coonrad RW, Goldner JL. A Study of the pathological findings and treatment in soft-tissue injury of the thumb metacarpophalangeal joint with a clinical study of the normal range of motion in one thousand thumbs and a study of post mortem findings of ligamentous structures in relation to function. J Bone Joint Surg Am. 1968;50(3):439–51.
47. Tang P. Collateral ligament injuries of the thumb metacarpophalangeal joint. J Am Acad Orthop Surg. 2011;19(5):287–96.
48. Patel S, Potty A, Taylor EJ, Sorene ED. Collateral ligament injuries of the metacarpophalangeal joint of the thumb: a treatment algorithm. Strategies Trauma Limb Reconstr. 2010;5(1):1–10.
49. Sedel L. Chapter 96. Dislocation of the metacarpophalangeal joint. In: Tubiana R, editor. The hand, vol. 2. 1st ed. Philadelphia: WB Saunders; 1985. p. 915–21.
50. Krause JO, Manske PR, Mirly HL, Szerzinski J. Isolated injuries to the dorsoradial capsule of the thumb metacarpophalangeal joint. J Hand Surg. 1996;21(3):428–33.
51. Posner MA, Retaillaud JL. Metacarpophalangeal joint injuries of the thumb. Hand Clin. 1992;8(4):713–32.
52. Farabeuf LH. De la luxation du pouce en arriere. Bull Acad Chir. 1876;2:21–62.
53. Stener B. Skeletal injuries associated with rupture of the ulnar collateral ligament of the metacarpophalangeal joint of the thumb. A clinical and anatomical study. Acta Chir Scand. 1963;125:583–6.
54. Stener B. Chapter 93. Acute injuries to the metacarpophalangeal joint of the thumb. In: Tubiana R, editor. The hand, vol. 2. 1st ed. Philadelphia: WB Saunders; 1985. p. 895–903.
55. Eaton RG, Dray GJ. Dislocations and ligament injuries in the digits. In: Green DP, editor. Operative hand surgery. New York: Churchill Livingstone; 1982. p. 657.
56. McLaughlin HL. Complex "locked" dislocation of the metacarpophalangeal joints. J Trauma. 1965;5(6):683–8.
57. Stener B. Hyperextension injuries of the metacarpophalangeal joint of the thumb –rupture of ligaments, fracture of sesamoid bones, rupture of flexor pollicis brevis. An anatomical and clinical study. Acta Chir Scand. 1963;125:265–93.
58. Campbell CS. Gamekeeper's thumb. J Bone Joint Surg Br. 1955;37B(1):148–9.
59. Baskies M, Lee S. Evaluation and treatment of injuries of the ulnar collateral ligament of the thumb metacarpophalangeal joint. Bull NYU Hosp Jt Dis. 2009;67(1):68.

60. Moutet F, Guinard D, Lebrun C, Bello-Champel P, Massart P. Metacarpo-phalangeal thumb sprains based on experience with more than 1,000 cases. Ann Chir Main. 1989;8(2):99–109. English, French.
61. Stener B. Displacement of the ruptured ulnar collateral ligament of the metacarpophalangeal joint of the thumb. A clinical and anatomical study. J Bone Joint Surg. 1962;44B(4):869–79.
62. Heyman P, Gelberman RH, Duncan K, et al. Injuries of the ulnar collateral ligament of the thumb metacarpophalangeal joint: biomechanical and prospective clinical studies on the usefulness of valgus stress testing. Clin Orthop Relat Res. 1993;292:165–71.
63. Smith RJ. Post-traumatic instability of the metacarpophalangeal joint of the thumb. J Bone Joint Surg. 1977;59A(1):14–21.
64. Heyman P. Injuries to the ulnar collateral ligament of the thumb metacarpophalangeal joint. J Am Acad Orthop Surg. 1997;5:224–9.
65. Tsiouri C, Hayton M, Baratz M. Injury to the ulnar collateral ligament of the thumb. Hand. 2009;4:12–8.
66. Gherissi A, Moussaoui A, Liverneaux P. Is the diagnosis of Stener's lesion echograph-dependent? A series of 25 gamekeeper's thumb. Chir Main. 2008;27(5):216–21.
67. Melville D, Jacobson JA, Haase S, Brandon C, Brigido MK, Fessell D. Ultrasound of displaced ulnar collateral ligament tears of the thumb: the Stener lesion revisited. Skeletal Radiol. 2013;42(5):667–73.
68. Coyle Jr MP. Grade III radial collateral ligament injuries of the thumb metacarpophalangeal joint: treatment by soft tissue advancement and bony reattachment. J Hand Surg Am. 2003;28A(1):14–20.
69. Hubbard LF. Metacarpophalangeal dislocations. Hand Clin. 1988;4(1):39–44.
70. Kaplan EB. Dorsal dislocation of the metacarpophalangeal joint of the index finger. J Bone Joint Surg Am. 1957;39-A(5):1081–6.
71. Lattanza LL, Choi PD. Intr-articular injuries of the metacarpophalangeal and carpometacarpal joints. In: Berger RA, Weiss APC, editors. Hand surgery, vol. 1. Philadelphia: Lippincott Williams & Wilkins; 2004. p. 175–94.
72. Calfee R, Sommerkamp T. Fracture-dislocation about the finger joints. J Hand Surg. 2009;34A(6):1140–7.
73. Beasley RW. Skeletal injuries of the thumb and fingers. In: Gumpert E, editor. Beasley's surgery of the hand. New York: Thieme; 2003. p. 200–25.
74. Becton JL, Christian JD, Goodwin HN, Jackson JG. A simplified technique for treating the complex dislocation of the index metacarpophalangeal joint. J Bone Joint Surg. 1975;57A:698–700.
75. Khouri SM, Fay JJ. Complete volar metacarpophalangeal joint dislocation of a finger. J Trauma. 1986;26:1058–60.
76. Delaere OP, Suttor PM, Degolla R, Leach R, Pieret PJ. Early surgical treatment for collateral ligament rupture of metacarpophalangeal joints of the fingers. J Hand Surg. 2003;28A:309–15.
77. Gaston GR, Lourie GM, Peljovich AE. Radial collateral ligament injury of the index metacarpophalngeal joint: an underreported but important injury. J Hand Surg. 2006;31A:1355–61.
78. Kang L, Rosen A, Potter HG, Weiland AJ. Rupture of the radial collateral ligament of the index metacarpophalngeal joint: diagnosis and surgical treatment. J Hand Surg. 2007;32A:789–94.
79. Kang L, Carlson MG. Extensor tendon centralization at the metacarpophalangeal joint: surgical technique. J Hand Surg. 2010;35A:1194–7.
80. Young CM, Rayan GM. The sagittal band: anatomic and biomechanical study. J Hand Surg. 2000;25A(6):1107–13.
81. Bunnell S. Fractures of metacarpals and phalanges. In: Boyes JH, editor. Bunnell's surgery of the hand. 5th ed. Philadelphia: J.B. Lippincott Company; 1970. p. 605.
82. Sedel L. Chapter 98. Dislocation of the carpopmetacarpal joint. In: Tubiana R, editor. The hand, vol. 2. 1st ed. Philadelphia: WB Saunders; 1985. p. 926–33.
83. El-Bacha A. Chapter 12. The Carpometacarpal joints (excluding the trapeziometacarpal). In: Tubiana R, editor. The hand, vol. 1. 1st ed. Philadelphia: WB Saunders; 1981. p. 158–68.
84. Gurland M. Carpometacarpal joint injuries of the fingers. Hand Clin. 1992;8(4):733–44.
85. Dobyns JH, Linscheid RL, Cooney III WP. Fractures and dislocations of the wrist and hand, then and now. J Hand Surg. 1983;8:687–90.
86. Eaton RG. Carpometacarpal joint injuries in acute and chronic ligamentous injuries of the fingers and thumb. In: Tubiana R, editor. The hand. Philadelphia: WB Saunders; 1985. p. 890–4.
87. Fotiadis E, Svarnas T, Lyrtzis C, Papadopoulos A, Akritopoulos P, Chalidis B. Isolated thumb carpometacarpal joint dislocation: a case report and review of the literature. J Orthop Surg Res. 2010;5:16–20.
88. Schmidt HM, Lanz U. Chapter 5. Thumb: joints. In: Schmidt H-M, Lanz U, editors. Surgical anatomy of the hand, translation of 2nd German edition. Stuttgart: Thieme; 2004. p. 106–12.
89. Farzan M, Siassi M, Espandar R. Thumb carpometacarpal joint volar dislocation: a case report. Acta Med Iran. 2002;40(1):52–4.
90. Shah J, Patel M. Dislocation of the carpometacarpal joint of the thumb. A report of four cases. Clin Orthop Relat Res. 1983;175:166–9.
91. Kuczynski K. Less-known aspects of the proximal interphalangeal joints of the human hand. Hand. 1975;7(1):31.

Acute Carpal Instability

16

Ian A. Trail

Keywords
Carpal fracture/dislocation • Early diagnosis/investigation • Complications • Early management • Rehabilitation • Salvage

Introduction

Instability of the carpus can range from predominantly simple inter-carpal ligament injuries e.g. scapholunate or lunotriquetral, to a complete fracture- dislocation, e.g. transcaphoid perilunar. As such, there is a spectrum of injury. The former often form part of a differential diagnosis of a scaphoid fracture, usually occurring as a result of a fall on an outstretched wrist, whilst the latter will require a more forceful injury which results in significant ligament dislocation, as well as a potential for fracture. In addition, predominantly isolated interosseous ligament injuries are often missed until symptoms become chronic and, as such, present late for definitive treatment. The pathogenesis and treatment of chronic instability will be covered in the chapter by Marc Garcia-Elias. It is, however, important to remember that these injuries almost always occur acutely and early treatment is ideal. The major disruptions are obviously easier to diagnose and should be treated as an emergency.

Fortunately, major ligament disruption within the wrist is uncommon, as most patients who fall sustain a fracture of the distal radius or the scaphoid. It is for this reason that carpal dislocations are seldom seen in older individuals. For children, the weakest link in the chain is the distal radial epiphysis, which will fracture. Generally, therefore, the most common age group is young individuals, particularly men in the second and third decade of life, who sustain an injury partaking in sport, or at work, or are involved in a road traffic accident. The most common major disruption is a transscaphoid perilunar dislocation. Other carpal bones, however, can be fractured, including the capitate.

Background and Aetiology

Whilst obviously trauma in varying degrees is a pre-requisite for injuries of this nature, there is still a considerable debate as to the exact mechanism of injury that produces the various types of instability.

I.A. Trail, MBCHB, MD, FRCS (Edin), FRCS (Lon), ECFMG
Upper Limb Research Department, Wrightington Hospital,
Hall Lane, Appley Bridge, Wigan, Lancashire WN6 9EP, UK
e-mail: upperlimb@wrightington.org.uk

I.A. Trail, A.N.M. Fleming (eds.), *Disorders of the Hand: Volume 1: Hand Injuries*,
DOI 10.1007/978-1-4471-6554-5_16, © Springer-Verlag London 2015

However, most researchers and experts accept that this injury is usually the result of a fall on the outstretched hand, with the wrist in hyperextension. A study on cadaveric wrists by Mayfield (1984) identified four stages of lunate instability [1]. These were:

1. Instability limited to the scapho-lunate joint
2. Added instability of the capito-lunate joint
3. Added damage to the triquetrolunate joint
4. Dorsal disruption of the radio-carpal ligament that leaves the lunate completely unstable and as such able to dislocate.

Using a cadaveric model, the forces to reproduce these types of injury were mimicked by having the wrist in extension, ulnar deviation and intercarpal supination. This supination occurs as a result of the thenar eminence striking the ground first. If, however, the hypothenar eminence strikes first, then the resulting pronation disrupts, predominantly, the dorsal ulna-triquetral complex, the triquetrolunate interosseous ligament and the anterior carpal capsule. The result of this is that the instability is predominantly on the ulnar side of the wrist. The so-called reverse perilunate instability [2].

Whilst in the majority of cases injuries follow a single trauma and from time to time it can follow a repeated or repetitive lesser trauma. Schroer et al. (1996) reported on the prevalence of carpal instability in a paraplegic population [3]. Of 162 paraplegic patients, 9 had a static carpal instability. As such, an acute on chronic presentation is possible. Similarly, patients who suffer with joint-laxity often have excessive mobility in the wrist joint [4]. These patients may be more at risk of developing ligament damage.

To understand what structures are damaged following trauma, it is important to have a thorough knowledge of the normal anatomy of the wrist and carpus.

The ligaments of the wrist are divided into intrinsic and extrinsic components. The two most important intrinsic (interosseous) ligaments, viz. the scapholunate and lunotriquetral ligaments, are divided into dorsal, proximal and palmar parts. The thickest and strongest part of the scapholunate ligament is located dorsally and that of the lunotriquetral ligament is located palmarly [5].

With regard to the extrinsic ligaments, there are several described on both the palmar and dorsal aspects of the wrist. It is generally accepted that those on the palmar aspect provide greater restraint to instability [6]. There are three strong palmar extrinsic radiocarpal ligaments, viz. the radioscaphocapitate, the long radiolunate and the short radiolunate ligaments. The radioscaphocapitate ligament, which extends from the radial styloid process through a groove on the waist of the scaphoid to the palmar aspect of the capitate, acts as a fulcrum around which the scaphoid rotates. The long radiolunate ligament, which lies parallel to the radioscaphocapitate ligament, extends from the palmar rim of the distal part of the radius to the radial margin of the palmar surface of the lunate. Located between the radioscaphocapitate and long radiolunate ligaments, at the level of the midcarpal joint, is an area of capsular weakness, known as the space of Poirier. The short radiolunate ligament, which is contiguous with palmar fibres of the triangular fibrocartilage complex, originates from the palmar margin of the distal part of the radius and inserts into the proximal part of the palmar surface of the lunate. The ulnolunate and ulnotriquetral ligaments arise from the volar edge of the triangular fibrocartilage and insert into the lunate and the triquetrum, respectively.

With regard to the radio-scapholunate ligament, Berger et al. (1991) felt that this structure could not be considered a true ligament, although this was disputed by Talesnik (1984) [7, 8].

Most authors agree that when the wrist and the carpus are injured, both the intrinsic and extrinsic ligaments are damaged. Generally, however, it is felt that for significant carpal instability to exist, the interosseus ligaments, specifically the scapholunate and luno-triquetral, have to be permanently damaged. On the ulnar side, Trumble et al. (1988) found that the pattern of volar intercalated segmental instability (VISI) required the rupture of both the triquetro-hamate and triquetro-lunate ligaments [9].

Returning to the radial side of the wrist, a number of authors believe that the principal area of ligament damage lies distally between the scaphoid and trapezium. Work by Short et al. (2005), however, confirmed that the scapho-lunate interosseous ligament is the primary stabiliser of the wrist and

that the radio-scapho capitate and scapho-trapezial ligaments act only in a secondary capacity [10].

Finally, we should also remember that the constitution of these ligaments changes with age. Weiss et al. (1994), in a cadaveric study, were able to identify scapho-lunate and lunar triquetral ligament defects in approximately 30 % of cases [11]. It should be noted, however, that most of these were in the central or weakest part of the ligament and did not represent true tears, lesions or defects. In addition, over half of the cadavers also had triangular fibro-cartilage tears.

Presentation, Investigation and Treatment Options

For the more severe cases, including dislocation, the diagnosis is often obvious. The wrist is grossly swollen, movements are markedly diminished and the patient is in severe pain. Movement of the fingers and indeed sensation at the finger tips is often also diminished. A plain x-ray will often indicate the severity of the injury. This can be further delineated by an urgent CT scan. On the p.a. view, overlapping of the distal carpal row at the mid carpal joint will be seen. Added to this, Gilula's arch's is broken [12]. On the lateral view, the distal concavity of the lunate no longer contains the head of the capitate. The latter appearing dorsally displaced (Figs. 16.1 and 16.2).

For the more subtle types of carpal instability, a careful history and physical examination is essential. Attention must be paid to the position of the wrist at the time of injury and the location of pain. Swelling and local tenderness are noted and the ranges of motion and grip strengths of the injured and uninjured sides are measured and compared. The most important differential diagnosis for pain on the radial side of the wrist is a fracture of the scaphoid. The problems of early diagnosis of this injury are well known, but, after 2–3 weeks, the standard tests for carpal instability can be performed. The pseudoinstability test, described by Kelly and Stanley in 1990, in which there is a loss of the normal antero-posterior translation of the carpus is useful [13]. Lack of this motion, due to protective spasm, is akin to the positive apprehension sign of shoulder instability. Other tests include that of Watson et al. (1986), which stresses the scapholunate interosseous ligament (Fig. 16.3) [14]. In a normal wrist, radial deviation causes scaphoid flexion that is able to overcome the resistance of an examining thumb on the scaphoid tubercle. In a scapholunate dissociation with radial deviation, the scaphoid flexes, but is unable to overcome the volar resistance of the thumb. The scaphoid has to escape and is forced dorsally, with the proximal pole clunking dorsally. Scapholunate or lunotriquetral ballottement may reveal specific joint instability and Lichtman et al. (1981) described a

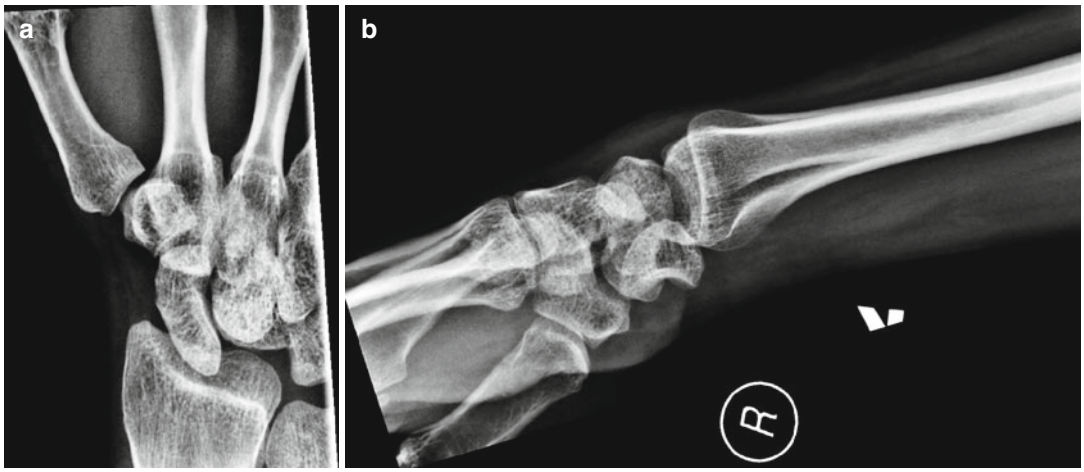


Fig. 16.1 AP and lateral image of a perilunar dislocation

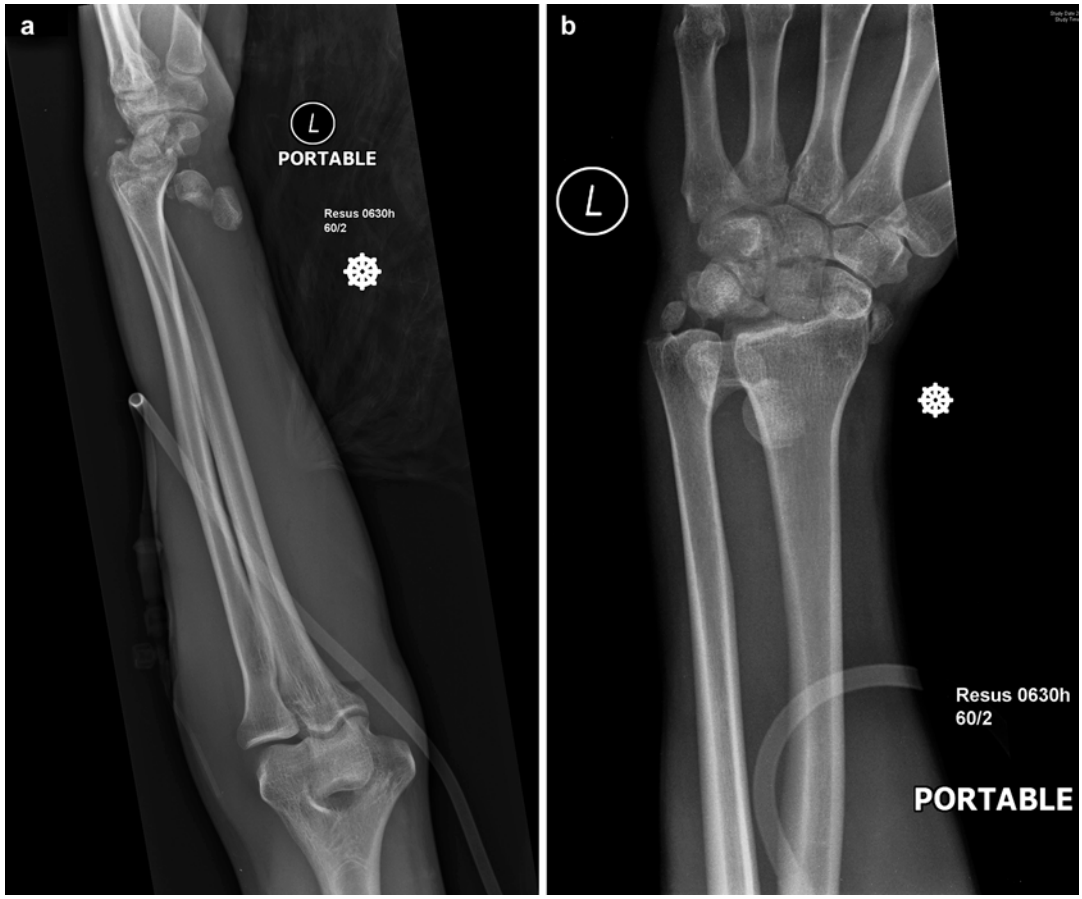


Fig. 16.2 AP and lateral image of a transcaphoid perilunar dislocation

pivot shift test for midcarpal instability (Fig. 16.3) [15]. All of these tests are specific for a particular ligamentous lesion, but overlap is not uncommon and, perhaps, the most specific and reliable test is point tenderness over the affected ligament.

With regard to investigations, the work of Schernberg (1990), on the radiological examination of the normal wrist, has shown the importance of the quality and reproducibility of the images obtained [16]. On the posterior/anterior views, the width of the scapholunate gap should be no greater than that of the triquetrolunate gap, that is no more than 3 mm (Fig. 16.4). Gilula and Weeks (1978) found that a scapholunate angle greater than 80° was indicative of DISI [12]. Schernberg also found that stress views were needed to diagnose 18 out of 27 cases of wrist injury. In addition, Degreif et al. (1990) advocated comparison of both wrists because of the considerable variations in normal anatomy [17]. Other authors have found that examination under an image intensifier can be particularly useful for dynamic instabilities [15, 18].

More sophisticated investigations, including arthrography and scintigraphy, may be of value. Arthrography can, undoubtedly, demonstrate leakage of contrast between the various intercarpal joints. Herbert et al. (1990), however, showed that an arthrogram is of little diagnostic value, unless it can be compared with that of the opposite, undamaged wrist [19].

CT and MRI scans are increasingly being used in the investigation of chronic wrist pain. Certainly, the CT scan has replaced other forms of tomography for the investigation of complex injuries of the carpus [20]. MRI, however, appears to have the greatest potential, although, like CT, it only gives static images [21]. Initially,

MRI scans often failed to reveal small tears of the interosseous ligaments [22]. Work by Schädel-Höpfner et al. (2001) from Germany revealed that the addition of intravenous contrast did not improve the accuracy of MRI scanning significantly [23]. Certainly, it did not compare with wrist arthroscopy. At our hospital, however, with the additional use of surface coils and improved software, we believe that the diagnostic value of MRI arthrograms have improved considerably.

Fig. 16.3 Watson's manoeuvre. Stressing the scapholunate interosseous ligament

Arthroscopy of the radiocarpal and midcarpal joints remains the gold standard in the diagnosis of carpal instability and, undoubtedly, also has a place in the acute case. Roth and Haddad (1986) and Cooney (1993) have all advocated its use and there is no doubt that it can provide much information about the altered mechanics and pathology of the wrist at all levels [24, 25]. Kelly and Stanley (1990) and Dautel et al. (1993) examined groups of patients with symptoms suggestive of a scapholunate interosseous ligament tear, but with normal radiographs and established the diagnosis by dynamic manoeuvres undertaken during radiocarpal and midcarpal arthroscopy [13, 26]. Fischer and Sennwald (1993) detected ligament tears by arthroscopy in every wrist in 20 cases of carpal instability [27]. Other, and more recent,

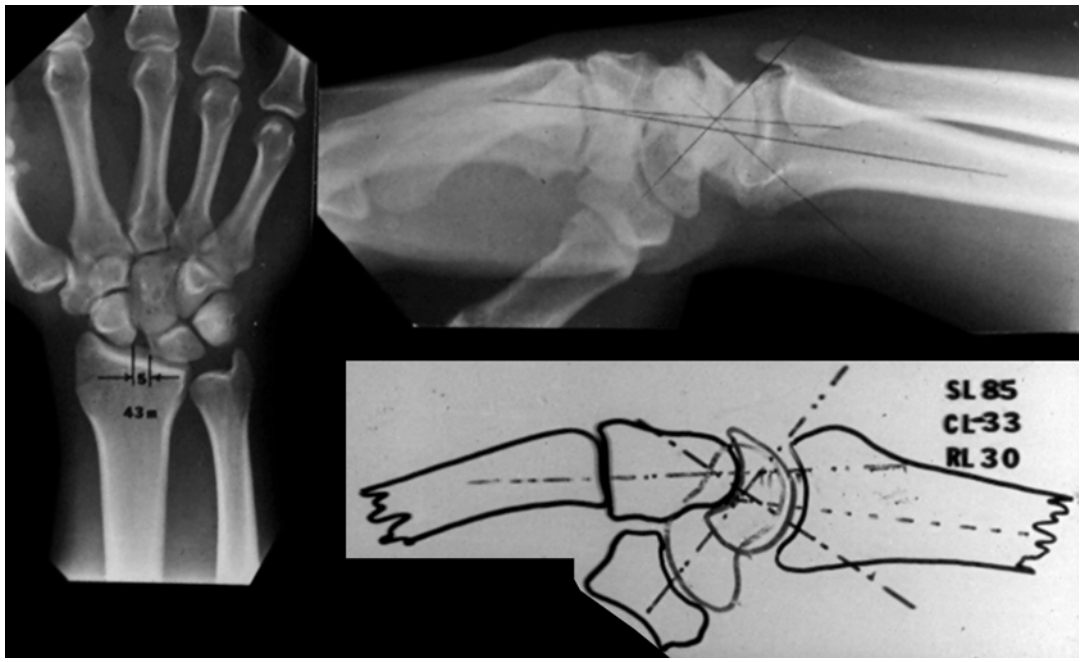


Fig. 16.4 AP radiological image of the wrist showing widened scapholunate interval. Lateral view showing scapholunate angle greater than 80°

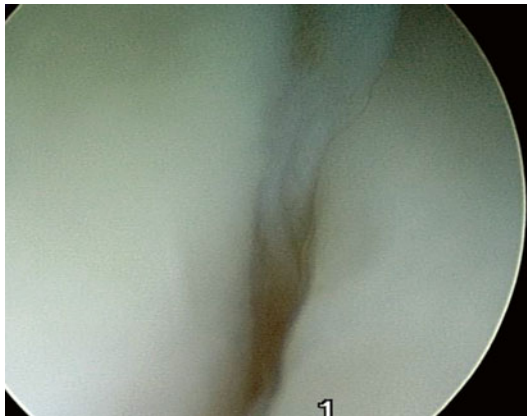


Fig. 16.5 Arthroscopic view of the midcarpal joint showing widening of the scapholunate interval

reports have indicated that arthroscopic evaluation of the wrist is more accurate and specific than arthrography in detecting the site and extent of ligament injury [28, 29].

At the radio-carpal level, the volar carpal ligaments are assessed in a radial to ulnar direction to determine whether an extrinsic ligament injury has occurred. Midcarpal arthroscopy, with the use of a triangulation probe, is also performed routinely. The space between the scaphoid and lunate bones is assessed for evidence of ligamentous laxity. A diagnosis of partial, or complete, carpal ligament injury is established on the basis of the ease of separation of the scaphoid from the lunate (Fig. 16.5) and of the lunate from the triquetrum. This is facilitated by unloading the wrist and stressing the various joints. Geissler initially classified the arthroscopic findings of a scapholunate ligament injury. These were, if the probe can be rotated within the space, a tear of the scapholunate or lunotriquetral interosseous ligament is probable. If either the probe or the arthroscope can be passed from the midcarpal to the radiocarpal joint, rotatory subluxation of the scaphoid (a complete scapholunate ligament tear with extrinsic ligament laxity) is confirmed. This has been further classified by Kozin (1999) (Table 16.1) [30].

Returning to the radiocarpal joint, a triangulation probe assists in the assessment of the size, location and extent of tears of the triangular fibrocartilage complex. Associated osseous injuries (fractures of the proximal pole of the scaphoid or dorsal triquetral chip fractures) can also be visualised.

Table 16.1 Arthroscopic classification of scapholunate ligament injuries

Grade	Description
Grade 1	Attenuation or haemorrhage, no incongruency
Grade 2	Incongruency or step-off of carpal space, slight gap less than width of probe
Grade 3	Incongruency or step-off of carpal space, probe passed between scaphoid and lunate
Grade 4	Incongruency or step-off of carpal space, scope (2.7 mm) passed through gap between scaphoid and lunate

Table 16.2 Classification of carpal dislocations

I.	Dorsal perilunate/volar lunate dislocation[a]
II.	Dorsal transscaphoid perilunate dislocation[a]
III.	Volar perilunate/dorsal lunate dislocation
IV.	Variants
	A. Transradial styloid perilunate dislocation[a]
	B. Naviculocapitate syndrome
	C. Transtriquetral fracture-dislocation
	D. Miscellaneous
V.	Isolated rotary scaphoid subluxation
	A. Acute subluxation
	B. Recurrent subluxation
VI.	Total dislocation of the scaphoid

[a]The most common patterns of injury

Classification of Carpal Instability

For the major dislocations, generally the classification relates to the various types of perilunar dislocation. This would focus on the direction of dislocation that is displacement of the lunate, together with the bones and any fractures involved (Table 16.2) [31].

Johnson (1980) further classified ligamentus injuries around the lunate as either a lesser arc perilunar dislocation which is purely ligamentus, or a greater arc which involves a fracture on one or more of the surrounding bones [32]. The terms used include the prefix trans, which indicates a fracture through a bone, or peri which would indicate a dislocation (Figs. 16.6 and 16.7).

Fig. 16.6 CT reconstruction of transradial perilunar dislocation

Fig. 16.7 X-ray image of total radiocarpal dislocation

For the isolated interosseous ligament injuries, the most frequently used terms are those introduced by Linscheid et al. (1972) and Dobyns et al. (1975), who identified four groups of carpal instability, viz. Dorsal Intercalated Segment Instability (DISI); Volar Intercalated Segment Instability (VISI), Ulnar Translocation and Dorsal Subluxation [33, 34].

The radiological appearances of the dorsiflexion and volar-flexion instability patterns have already been discussed. Ulnar translocation describes ulnar shift of the carpus on the radius and is commonly seen in patients with rheumatoid arthritis. Dorsal subluxation describes dorsal shift of the carpus, often seen after malunion of distal radial fractures [35].

Taleisnik in 1984 introduced the concepts of static and dynamic instability [8]. Static instability is an end state, with marked scapholunate dissociation, fixed flexion of the scaphoid and fixed extension of the lunate. Dynamic instability exists in the presence of partial ligament injuries resulting in pain, but with minimal, or even no changes on the plain radiographs. The diagnosis is made by dynamic radiology or arthroscopy.

The terms carpal instability dissociative (CID), carpal instability non-dissociative (CIND) and carpal instability complex (CIC) were introduced by Dobyns and Gabel, in 1990 [36]. CID describes instability due to loss of linkage between the individual bones of either row; CIND means that there is no dissociation between individual carpal bones, but instability at the radiocarpal or midcarpal joints. CIC includes instabilities which were not otherwise classifiable.

Surgical Techniques and Rehabilitation Including Results

For the major dislocations there is no doubt that treatment is required as an emergency. That is, any dislocation should be reduced as soon as possible. Thereafter, the wrist should be either immobilized in a plaster of Paris cast or splint, reductions stabilised by a percutaneous pinning or by the application of an external fixator or finally, by open reduction, ligamentus repair or fracture fixation as appropriate. Treatment by simply immobilizing the wrist in a cast has fallen

into disrepute. Specifically, closed reduction is often difficult and, if attempted, would require traction and complete muscle relaxation. Secondly and more importantly, post reduction x-rays often show marked signs of continuing instability. It is unusual for the scaphoid and lunate to assume normal positions following an injury of this magnitude.

Clinical Pearl
For major dislocation, reduction should be undertaken as an urgency. At the same time, fracture fixation or ligamentous repair should be undertaken as appropriate. Any reduction or repair will have to be protected by temporary fixation.

If, however, closed reduction can be obtained, then stabilization using trans articular k wire is an option. The wire should be inserted transversely across the proximal carpal row in such a fashion that any instability is reduced. Pins are usually removed between 6 and 8 weeks, although a further period of immobilization may well be required.

More recently, however, a number of long term studies have demonstrated the superiority of open reduction with ligament repair and percutaneous k-wire fixation [37–39]. There is no doubt that open techniques do allow more accurate reduction and repair of the appropriate ligaments. This will of course require both dorsal and palmar approaches to the wrist and, as such, quite a wide dissection. Poor prognostic factors according to Garcia-Elias et al. (1986) included the time interval between accident, diagnosis and treatment and the accuracy of reduction [40].

Technique of Open Reduction and Repair

The operation is undertaken with the patient supine and under a full general anaesthetic, or brachial plexus block with sedation. Finally, a tourniquet is applied. The approach to the scapholunate ligament is through a dorsal incision, with the rationale being that the most important and strongest part of the ligament is posterior. Better exposure is achieved through a longitudinal skin incision, although if correctly sited a transverse incision can give adequate exposure and is more cosmetically acceptable. The extensor retinaculum is then opened in the fourth compartment, to allow retraction of the extensor tendons to the fingers. The extensor pollicis longus is also protected. The posterior capsule of the wrist can then be opened, either transversely or using the ligament splitting capsulotomy as recommended by Berger, 1998 [41]. The scapholunate ligament will lie just distal to Lister's tubercle. Repair of the ligament will very much depend on findings and could include either a direct suture or, if the ligament avulsed from bone, reattachment, with either transosseous fixation or small bone anchors. The latter is undoubtedly simpler. An additional and useful technique involves inserting anchors into either or both the scaphoid and lunate and passing sutures through the ligament or its attachment and, if necessary, tying anchor to anchor [42]. This technique is certainly useful and relatively easy to perform. Thereafter, the repair is protected by k-wire fixation. It is very tempting to pass wires to the radius into the lunate and scaphoid and certainly this is an effective technique for stabilization. However, wires used in this situation can break, leaving prominent wires within the wrist joint, unless extremely thick wires are used (1.9 mm). A better technique is to pass the wires transversely from scaphoid to lunate.

Clinical Pearl
For acute scapholunate ligament injury, early open repair and temporary fixation is currently regarded as the optimum treatment.

For the lunotriquetral ligament, the approach would be from the volar aspect. Here a longitudinal incision is used. The long flexor tendons and the median nerve are identified and retracted

radially, with the ulna nerve and artery being retracted ulnarwards. This would then expose the volar capsule. Often this capsule could be damaged and any tear can be extended. Otherwise, a transverse incision is probably the best. This allows exposure of the lunate and triquetrum bones and the torn ligament. The rationale for a palmar approach to a lunar triquetral ligament is that it is again the volar section that is the strongest. Its repair, like the scapholunate, will depend on the findings. Similar techniques can be used.

Traditionally, rehabilitation will involve removal of the k-wires, usually at 6 weeks, followed by immobilization of the wrist in the neutral position in a cast for a further 6 weeks. If, however, the surgeon is confident with the strength of his repair, then gentle supervised mobilization could begin at 6 weeks. Initially, in our unit, this would take the form of "dart throwing". That is movements from flexion/ulnar deviation through extension and radial deviation. Between episodes of mobilization, the patient has a removable splint. More general mobilization techniques are introduced and by 12 weeks the patient can concentrate on strengthening.

For greater arc injuries, that is dislocations that include fractures of adjacent bones, particularly of the scaphoid, but also the capitate and triquetrum, the recommended treatment would be open reduction and internal fixation of the specific fractures. Indeed, fixation of a scaphoid fracture would often stabilize the radial side of the wrist. For surgical technique, the reader is referred to Tim Davis' chapter.

Whilst this is almost always done as an open procedure, more recently it has been described as an arthroscopically assisted percutaneous fixation [43]. For results Moneim et al. (1984), reported healing of the scaphoid fracture in 15 of the 17 wrists in their series [44]. Inoue et al. (1990), (with a mean follow-up of 18 months), reported good to excellent results in over 50 % of cases [45]. Finally, Herzberg et al. (1993), in a large series, reported that an open injury and a delay to treatment had an adverse affect on clinical results, whereas an anatomical type of injury had less influence [46]. In cases treated early, the clinical results were satisfactory, although the incidence of post-traumatic arthritis was high (56 %). In the dorsal perilunar dislocation group with pure ligamentous injuries and in the dorsal transscaphoid group, the best radiological results were observed after open reduction and internal fixation. In the latter group, however, the fixation of the scaphoid alone was not always sufficient. There was occasional scapholunate dissociation, lunotriquetral dissociation, ulnar translation of the carpus or other carpal collapse patterns.

Forli et al. in 2010, in a series with greater than 10 year follow up, reported radiological evidence of arthritis and static carpal instability. However, these findings did not cause reduced function [47]. Finally, and despite the severity of these, avascular necrosis of the lunate is rare. The only definite reported cases were by White and Omer (1984), who reported an incidence of 12.5 %, that is 3 of 24 cases [48]. However, the clinical course was transient, with a resolution of the radiodensity. None of the three cases progressed to classic avascular necrosis.

For fractures of the capitate, these can often be displaced or rotated to 180° [49]. The mechanism of injury being the direct impact of the capitate bone against the dorsal edge of the radius. Thereafter, the capitate head rotates as the distal fragment realigns itself. The nature of this injury is better seen on a CT scan. Given the displacement, however, recommended treatment again would be open reduction and internal fixation. It is also important to remember, with this displacement, the median nerve is often compressed and, as such, an acute carpal tunnel release is required.

Surprisingly, perilunar dislocations are sometimes missed and, as such, cases do present late with ongoing wrist pain, particularly swelling, but also loss of movement. In these cases open reduction should be attempted, although this may require both a volar and dorsal approach. Siegert et al. (1988), reported 16 cases that had been untreated for a minimum of 6 weeks [50]. Of the six patients who underwent open reduction and internal fixation, all had a satisfactory outcome and none required additional surgery. For the other, four had isolated carpal bone excision, two

had wrist arthrodesis and two had proximal row carpectomy. Many of these required subsequent operations.

Weir (1992), in a small series of cases of late reduction, reported poor results, particularly in terms of range of movement and x-ray appearance [51]. Despite that, function was surprisingly good, in that all patients returned to normal activities, including heavy manual work.

An external fixator across the wrist has also been recommended [52]. If, however, there is significant cartilage damage then a salvage procedure, either fusion or proximal row carpectomy, may be more appropriate.

Isolated Scapholunate Dissociation

For the isolated interosseous ligament injury, whilst there is now undoubtedly a trend towards surgical intervention, if the disability is minor, that is if more than 80 % of the normal range of motion and grip strength has been retained, Dobyns et al. 1975 suggested that no treatment is required [34]. Certainly, in our practice, a number of patients choose not to undergo reconstructive surgery and, with some modifications of life style, seem to cope extremely well with a chronic scapho-lunate ligament injury.

A study from Wrightington Hospital summarised the outcome of 11 patients with arthroscopically proven interosseous scapho-lunate ligament injury, but without any radiological signs of either DISI deformity or scapho-lunate gapping [53]. The average follow up of these patients was 7 years. At review, all patients were still experiencing pain, with a mean measurement on the visual analogue pain scale of 3.2 at rest, worsening to 6.5 after activity. This was compared to a pain score measured at diagnosis of 7.6 at rest and 8.2 after activity. All patients took oral analgesia from time to time and, occasionally, wore a splint. Wrist movement in all of the patients was diminished compared to the contralateral side, with grip strength 60 % of normal. All but one of the patients had altered their occupation and all were generally performing lighter work. Radiologically, there had been no deterioration in X-ray appearances, although one patient had developed some very early radio-carpal osteoarthritis, seen at the tip of the radial styloid.

When this condition is diagnosed early however, and treatment is indicated, an attempt should be made to bring about healing of the torn interosseous ligament. Palmer et al. (1978) reported good results from immobilisation in plaster for 8 weeks, if treatment started within 4 weeks of injury and if an anatomical reduction was maintained [54]. This often required the use of supplementary closed pinning with a Kirschner wire under radiographic control. Cases that cannot be reduced and held by this technique, and those diagnosed later, do poorly with immobilisation and often require surgery. Ligament reconstruction, whether undertaken through a volar or a dorsal approach (Taleisnik, 1984) also often need supplementary Kirschner wire fixation and the results range from good to fair, depending on the quality of the tissue available for repair [8]. At this time and as stated previously, it is normal practice to repair the scapholunate ligament through a dorsal approach and the triquetro-lunate through a palmar approach when possible, as this addresses the stronger parts of those ligaments. The results of direct repair were reported by Wyrick et al. in 1998 [55]. They had 17 patients available for follow up for an average of 30 months. In this series it should be noted that the scapho-lunate ligament repair had been augmented by a dorsal capsulodesis. At follow up, no patient was pain free, average wrist motion was 60 % of normal and grip strength 70 %. Radiologically, the scapho-lunate angle had improved from 78° pre-operatively to 47° at surgery. Unfortunately, at long term follow up, the scapho-lunate angle had deteriorated back to 72°. Similar changes were noted for the scapho-lunate interval. Overall, only two patients had an excellent or good result, with 6 of the 17 having good or excellent results on X-ray analysis. As a consequence, these authors advocated a cautious approach when describing the outcome of this form of surgery.

In 2000, Bickert et al. from Heidelberg, Germany, reported a retrospective analysis of acute scapholunate ligament repair using Mini bone anchors [56]. At follow-up, at a mean of 19 months after surgery, results were found to be good or excellent in eight patients, satisfactory in two and poor in a further two. Assessments were

by pain score, grip strength and DASH. Radiological assessment revealed stable scapholunate ligaments in ten patients: by this, it was meant that the mean scapholunate angle was 55°, with a scapholunate gap of no more than 3.2 mm.

Isolated Lunotriquetral Dissociation

This condition is less common and, again, if diagnosed early can be treated by either simple immobilisation or acute ligament repair. Reagan et al. (1984) found that simple immobilisation was only useful for acute injuries with capsulodesis, tenodesis and arthrodesis being reserved for chronic cases [2].

Conclusion

Acute carpal instability is a spectrum of injuries, ranging from a simple inter-carpal ligament injury through to fracture and dislocation. CT scans are undoubtedly helpful in the fracture/dislocation cases, whilst single interosseous ligament injuries are often best diagnosed by MRI arthrograms or an arthroscopy. For the major disruptions, treatment would take the form of immediate reduction and fixation of any fracture and if necessary ligament repair. Delayed presentation is more difficult and will often require a salvage procedure, either fusion or proximal row carpectomy. For a single interosseous ligament injury an open direct repair and temporary fixation would currently be regarded as the optimum treatment. Appropriate rehabilitation is also crucial.

References

1. Mayfield JK. Wrist ligamentous anatomy and pathogenesis of carpal instability. Orthop Clin North Am. 1984;15:209–16.
2. Reagan DS, Linscheid RL, Dobyns JH. Lunotriquetral sprains. J Hand Surg Am. 1984;9:502–14.
3. Schroer W, Lacey S, Frost FS, Keith MW. Carpal instability in the weight-bearing upper extremity. J Bone Joint Surg Am. 1996;78:1838–43.
4. Garcia-Elias M, Ribe M, Rodriguez J, Cots M, Casas J. Influence of joint laxity on scaphoid kinematics. J Hand Surg Br. 1995;20:379–82.
5. Berger RA, Imeada T, Berglund L, An KN. Constraint and material properties of the subregions of the scapholunate interosseous ligament. J Hand Surg Am. 1999;24:953–62.
6. Katz DA, Green JK, Werner FW, Loftus JB. Capsuloligamentous restraints to dorsal and palmar carpal translation. J Hand Surg Am. 2003;28:610–3.
7. Berger RA, Kauer JM, Landsmeer JM. Radioscapholunate ligament: a gross anatomic and histologic study of fetal and adults wrists. J Hand Surg Am. 1991;16:350–5.
8. Taleisnik J. Classification of carpal instability. Bull Hosp Jt Dis Orthop Inst. 1984;44:511–31.
9. Trumble T, Bour CJ, Smith RJ, Edwards GS. Intercarpal arthrodesis for static and dynamic volar intercalated segment instability. J Hand Surg Am. 1988;13:384–90.
10. Short WH, Werner FW, Green JK, Masaoka S. Biomechanical evaluation of the ligamentous stabilizers of the scaphoid and lunate: part II. J Hand Surg Am. 2005;30:24–34.
11. Weiss AP, Sachar K, Gendreau M. Conservative management of carpal tunnel syndrome: a reexamination of steroid injection and splinting. J Hand Surg Am. 1994;19:410–5.
12. Gilula LA, Weeks PM. Post-traumatic ligamentous instabilities of the wrist. Radiology. 1978;129:641–51.
13. Kelly EP, Stanley JK. Arthroscopy of the wrist. J Hand Surg Br. 1990;15:236–42.
14. Watson HK, Rye J, Akelman E. Limited triscaphoid intercarpal arthrodesis for rotatory subluxation of the scaphoid. J Bone Joint Surg Am. 1986;68:345–9.
15. Lichtman DM, Schneider JR, Swafford AR, Mack GR. Ulnar midcarpal instability-clinical and laboratory analysis. J Hand Surg Am. 1981;6:515–23.
16. Schernberg F. Roentgenographic examination of the wrist: a systematic study of the normal, lax and injured wrist. Part 1: the standard and positional views. J Hand Surg Br. 1990;15:210–9.
17. Degreif J, Benning R, Rudigier J, Ritter G. Scapholunar dissociatio--hen an accident sequela, when a normal congenital variant? Langenbecks Arch Chir Suppl II Verh Dtsch Ges Chir. 1990;731–34.
18. Stanley D, Herbert TJ. The Swanson ulnar head prosthesis for post- traumatic disorders of the distal radioulnar joint. J Hand Surg Br. 1992;17:682–8.
19. Herbert TJ, Faithfull RG, McCann DJ, Ireland J. Bilateral arthrography of the wrist. J Hand Surg Br. 1990;15:233–5.
20. Stewart NR, Gilula LA. CT of the wrist: a tailored approach. Radiology. 1992;183:13–20.
21. Zlatkin MB, Greenan T. Magnetic resonance imaging of the wrist. Magn Reson Q. 1992;8:65–96.
22. Munk PL, Vellet AD, Levin MF, Steinbach LS, Helms CA. Current status of magnetic resonance imaging of the wrist. Can Assoc Radiol J. 1992;43:8–18.
23. Schädel-Höpfner M, Iwinska-Zelder J, Braus T, Böhringer G, Klose KJ, Gotzen L. MRI versus arthroscopy in the diagnosis of scapholunate ligament injury. J Hand Surg Br. 2001;26:17–21.
24. Roth JH, Haddad RG. Radiocarpal arthroscopy and arthrography in the diagnosis of ulnar wrist pain. Arthroscopy. 1986;2:234–43.

25. Cooney WP. Evaluation of chronic wrist pain by arthrography, arthroscopy, and arthrotomy. J Hand Surg Am. 1993;18:815–22.
26. Dautel G, Goudot B, Merle M. Arthroscopic diagnosis of scapho-lunate instability in the absence of X-ray abnormalities. J Hand Surg Br. 1993;18:213–8.
27. Fischer M, Sennwald G. Arthroscopy in diagnosis of carpal instability. Helv Chir Acta. 1993;59:693–6.
28. Cooney WP, Dobyns JH, Linscheid RL. Arthroscopy of the wrist: anatomy and classification of carpal instability. Arthroscopy. 1990;6:133–40.
29. Weiss AP, Akelman E. Diagnostic imaging and arthroscopy for chronic wrist pain. Orthop Clin North Am. 1995;26:759–67.
30. Kozin SH. The role of arthroscopy in scapholunate instability. Hand Clin. 1999;15:435–44.
31. Green DP, O'Brien ET. Classification and management of carpal dislocations. Clin Orthop Relat Res. 1980;149:55–72.
32. Johnson RP. The acutely injured wrist and its residuals. Clin Orthop. 1980;149:33–44.
33. Linscheid RL, Dobyns JH, Beabout JW, Bryan RS. Traumatic instability of the wrist. Diagnosis, classification, and pathomechanics. J Bone Joint Surg Am. 1972;54:1612–32.
34. Dobyns JH, Linscheid RL, Chao EYS, Weber ER, Swanson GE. Traumatic instability of the wrist. AAOS Instr Course Lect. 1975;24:182–99.
35. Dias JJ, McMohan A. Effect of Colles' fracture malunion on carpal alignment. J R Coll Surg Edinb. 1988; 33:303–5.
36. Dobyns JH, Gabel GT. Gymnast's wrist. Hand Clin. 1990;6:493–505.
37. Green DP, O'Brien ET. Open reduction of carpal dislocations: indications and operative techniques. J Hand Surg Am. 1978;3:250–65.
38. Minami A, Kaneda K. Repair and/or reconstruction of scapholunate interosseous ligament in lunte and perilunate dislocations. J Hand Surg Am. 1993;18:1099–106.
39. Sotereanos DG, Mitsionis GJ, Giannakopoulos PN, Tomaino MM, Herndon JH. Perilunate dislocation and fracture dislocation: a critical analysis of the volar-dorsal approach. J Hand Surg Am. 1997;22:49–56.
40. Garcia-Elias M, Irisarri C, Henriquez A, Abanco J, Fores J, Lluch A, Gilabert A. Perilunar dislocation of the carpus. Ann Chir Main. 1986;5(4):281–7.
41. Berger RA. Ligament anatomy. In: Cooney WP, Linscheid RL, Dobyns JH, editors. The wrist. Diagnosis and operative treatment. St. Louis: Mosby; 1998. p. 73–105.
42. Rosati M, Parchi P, Cacianti M, Poggetti A, Lisanti M. Treatment of acute scapholunate ligament injuries with bone anchor. Musculoskelet Surg. 2010;94:25–32.
43. Jeon L-H, Kim H-J, Min W-K, Cho H-S, Kim P-T. Arthroscopically assisted percutaneous fixation for trans-scaphoid perilunate fracture dislocation. J Hand Surg Br. 2010;35(8):884–8.
44. Moneim MS, Hofammann KE, Omer GE. Transscaphoid perilunate fracture-dislocation. Clin Orthop Relat Res. 1984;190:227–35.
45. Inoue G, Tanaka Y, Nakamura R. Treatment of transscaphoid perilunate dislocations by internal fixation with the Herbert screw. J Hand Surg Br. 1990;15:449–54.
46. Herzberg G, Comtet JJ, Linscheid RL, Amadio PC, Cooney WP, Stalder J. Perilunate dislocations and fracture dislocations: a multicentre study. J Hand Surg Am. 1993;18:768–79.
47. Forli A, Courvoisier A, Wimsey S, Corcella D, Moutet F. Perilunate dislocations and transscaphoid perilunate fracture-dislocations: a retrospective study with minimum ten-year follow-up. J Hand Surg Am. 2010;35:62–8.
48. White RE, Omer GE. Transient vascular compromise of the lunate after fracture-dislocation or dislocation of the carpus. J Hand Surg Am. 1984;9:181–4.
49. Monahan PR, Galasko CS. The scapho-capitate fracture syndrome: a mechanism of injury. J Bone Joint Surg Br. 1972;54:122–4.
50. Siegert JJ, Frassica FJ, Amadio PC. Treatment of chronic perilunate dislocations. J Hand Surg Am. 1988;13:206–12.
51. Weir IGC. The late reduction of carpal dislocations. J Hand Surg Br. 1992;17:137–9.
52. Fernandez DL, Ghillani R. External fixation of complex carpal dislocations: a preliminary report. J Hand Surg Am. 1987;12:335–47.
53. O'Meeghan CJ, Stuart W, Mamo V, Stanley JK, Trail IA. The natural history of an untreated isolated scapholunate interosseous ligament injury. J Hand Surg Br. 2003;28:307–10.
54. Palmer AK, Dobyns JH, Linscheid RL. Management of post-traumatic instability of the wrist secondary to ligament rupture. J Hand Surg Am. 1978;3:507–32.
55. Wyrick JD, Youse BD, Kiefhaber TR. Scapholunate ligament repair and capsulodesis for the treatment of static scapholunate dissociation. J Hand Surg Br. 1998;23:776–80.
56. Bickert B, Sauerbier M, Germann G. Scapholunate ligament repair using the Mitek bone anchor. J Hand Surg Br. 2000;25:188–92.

Acute DRUJ Instability

17

Brian D. Adams

Keywords
Acute distal radioulnar joint instability • Distal radioulnar joint instability • Wrist instability

Introduction

The distal radioulnar joint (DRUJ) is a diarthrodial, synovial articulation that provides the distal link between the radius and ulna and a pivot for pronation-supination. Because its articulation is incongruent, the soft tissues play a substantial role in guiding and restraining the joint. During forearm motion, the DRUJ moves synchronously with the proximal radioulnar joint and thus any injury or deformity involving the radius or ulna can alter the function of both joints. The distal radioulnar and ulnocarpal joints are also anatomically and functionally integrated, so that both are affected by traumatic and arthritic conditions. Because of these interdependences, evaluation and treatment of the DRUJ is challenging. In the treatment of DRUJ instability, proper skeletal alignment is paramount and must be corrected before any soft tissue repair. In addition, the sigmoid notch and ulnar head must be sufficiently competent to allow the ligament restraints to function under normal tension and length. This chapter will discuss the relevant anatomy and pathophysiology of DRUJ instability, along with the rationale and technique for different treatment options.

Anatomy

The DRUJ is a trochoid articulation that allows both rotation and translation during normal forearm motion. The overall dimensions of the sigmoid notch average 15 mm in the transverse plane and 10 mm in the coronal plane. The shape of the notch varies considerably in both planes, ranging from relatively flat to hemicylindrical. Its dorsal bony rim is typically acutely angled while the volar rim is more rounded but frequently augmented by a fibrocartilaginous lip (Fig. 17.1). Acquired deficiency of the sigmoid notch, such as a fracture of the rim, has been shown clinically and in biomechanical investigations to cause instability [1–4]. In addition, individuals with a developmental deficiency of the notch are more prone to joint instability following ligament injury. The ulnar head serves as the seat for the

B.D. Adams, MD
Department of Orthopaedics, University of Iowa Hospitals and Clinics, Iowa, IA, USA
e-mail: brian-d-adams@uiowa.edu

I.A. Trail, A.N.M. Fleming (eds.), *Disorders of the Hand: Volume 1: Hand Injuries*,
DOI 10.1007/978-1-4471-6554-5_17, © Springer-Verlag London 2015

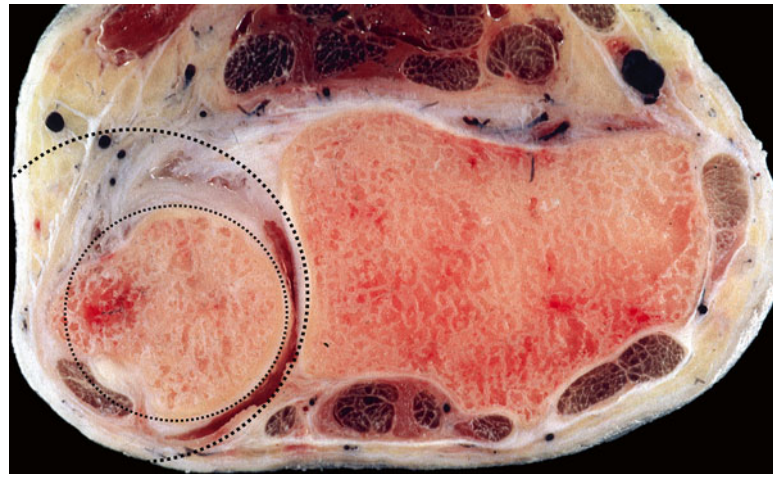


Fig. 17.1 Cross section through the DRUJ in a cadaver. The sigmoid notch is shallow with a radius of curvature substantially larger than that of the ulnar head. The rims of the notch are augmented by fibrocartilaginous lips

sigmoid notch, around which the radius rotates. It is asymmetrical in the sagittal plane, forming a cylinder of about a 130° arc with articular cartilage covering as little as 90º of this arc [5].

The ulnar styloid is a continuation of the subcutaneous ridge of the ulna, projecting 2–6 mm distally. It provides increased area for soft-tissue attachments and is frequently fractured resulting in ligament destabilization. At the base of the styloid lies a shallow concavity termed the fovea that is replete with vascular foramina and is an attachment site for ligaments. Identification of this site is essential for repair and reconstructive procedures because the axis of forearm motion passes through it.

The soft-tissue structures that contribute to DRUJ stability are the pronator quadratus, extensor carpi ulnaris (ECU), interosseous membrane (IOM), DRUJ capsule and components of the triangular fibrocartilage complex (TFCC) [6]. The relative contributions of these structures to joint stability are controversial however there is common agreement that severe instability requires disruption of multiple structures [2, 6]. The pronator quadratus and ECU musculotendinous unit provide some level of dynamic stability. The pronator quadratus produces indirect DRUJ stabilization by coaptating the joint during active pronation and passive supination. ECU contraction elevates the ulnar carpus dorsally and depresses the ulnar head palmarly. The interosseous membrane is important in maintaining the radius and ulna as a single functioning forearm unit, especially when the radial head or proximal radioulnar joint is disrupted. Complete radioulnar dissociation at the DRUJ does not occur until the IOM is also incompetent.

The TFCC, named by Palmer and Werner, is the most commonly used term for the interconnected soft tissues that span and support the DRUJ and ulnocarpal articulations [7]. The primary functions of the TFCC are to (1) extend the smooth articular surface of the distal radius to cover the ulna head, (2) transmit axial force across the ulnocarpal joint while partially absorbing the load, (3) provide a strong but flexible connection between the distal radius and ulna that allows forearm rotation, and (4) support the ulnar portion of the carpus through connections to both the ulna and radius. Its anatomic complexity and multiple functions place it at substantial risk for injury and degeneration.

The TFCC is generally accepted to be the major static, soft tissue stabilizer, and the palmar and dorsal radioulnar ligaments are the prime components of the TFCC. These ligaments appear as thickenings at the combined junctures of the triangular fibrocartilage, DRUJ capsule and ulnocarpal capsule. They are comprised of longitudinally oriented lamellar collagen and thus suited to bear tensile loading. As each radioulnar ligament extends ulnarly, it divides in the coronal plane into

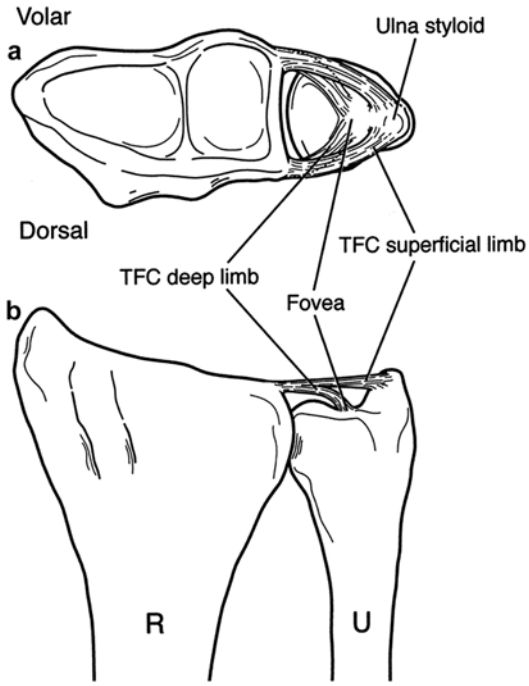


Fig. 17.2 Normal division of dorsal and palmar radioulnar ligaments into superficial (distal) limbs that attach the ulnar styloid and deep (proximal) limbs that attach at the fovea of the ulnar head. *TFC* triangular fibrocartilage. Distal view (**a**) and dorsal view (**b**) of wrist (From Adams [31])

two limbs. The deep or proximal limb attaches to the fovea, and the superficial or distal limb attaches to the base and midportion of the ulnar styloid (Fig. 17.2). The space between the limbs near the ulnar styloid contains richly vascularized loose connective tissue. This tissue is sometimes called the ligamentum subcruetum, which is a confusing term because it has neither the histologic nor mechanical features of a ligament. This diverging fiber arrangement has clinical implications for ulnar styloid fractures. A basilar styloid fracture imparts mechanical discontinuity of the superficial limb with the ulna and signifies potential disruption of the deep limb because the energy of injury passes close to this site. Conversely, the proximal fibers attaching to the fovea may remain intact preserving DRUJ stability.

The vascular supply to the articular disk is variable and plays a central role in its healing potential and the treatment options. Its vascular supply is primarily via the anterior interosseous artery and ulnar artery [8]. The anterior interosseous artery provides palmar and dorsal branches to the DRUJ. The dorsal branch supplies most of the dorsal periphery, and the palmar branch supplies the volar periphery near the radius. Dorsal and palmar branches of the ulnar artery supply the styloid area and ulnar part of the volar periphery. Vascular penetration into the disk extends only to its outer 15 %, leaving the central portion essentially avascular [8, 9]. With aging, the vascular supply of the peripheral disk decreases [10]. Based on these findings, the central disk has little or no possibility to heal while the periphery of the disk has good potential. Similar to its vascular distribution, the neural supply of the TFCC excludes the central portion of the disk. The volar and ulnar portions of the TFCC receive innervation from the ulnar nerve, and the dorsal portion from the posterior interosseous nerve. The dorsal sensory branch has a variable supply to all portions of the TFCC.

Biomechanics

The normal arc of pronation and supination ranges among individuals from 150° to 180°. Additional rotation, up to 30°, occurs through the radiocarpal joint. The axis of forearm motion varies during rotation, especially under load, but generally passes near the cross-sectional centers of the radial head proximally and ulnar head distally. During forearm rotation, DRUJ translation occurs because the sigmoid notch is shallow, subtending a sector of only 47° to 80°, and its radius of curvature is 50 % greater than that of the ulnar head. Total dorsopalmar translation with the forearm in neutral rotation was measured at 8 to 9 mm in normal cadaveric joints subjected to externally applied forces, although in vivo studies suggest that the actual amount of translation may be considerably less [11]. Nonetheless, when the unloaded forearm is in the neutral position, articular contact is maximal, reaching 60 % of the available surface area. At the extremes of pronation and supination there may be as little as 2 mm of articular contact at the rims of the notch (less than 10 % of the articular surface area) [5].

Opposing views exist regarding which of the two radioulnar ligaments are the major restraints to dorsal or palmar joint displacement, however two clinically relevant themes have emerged. First, the ligaments act in concert with the rims of the sigmoid notch to constrain the joint. Second, both ligaments are necessary for normal stability in either direction. Thus, in patients with bidirectional or severe unidirectional instability, both ligaments should be suspected of injury. From a clinical perspective, it is likely that the foveal attachments are the most critical stabilizers of the DRUJ.

Although distal radius fractures are known to frequently involve the sigmoid notch, especially the dorsal rim, the extent of involvement is probably underestimated on standard radiographs and better shown by CT [12]. The clinical implication of residual incongruity of the sigmoid notch has not been well studied. Anatomic and biomechanical studies and case reports suggest that the rims of the sigmoid notch play important stabilizing roles for the joint [4].

In Galeazzi fracture-dislocations of the forearm, a class 1B TFCC injury is almost inevitably present, although there is a spectrum of DRUJ instability that may be associated. In one study, a more distal fracture of the radial shaft was associated with a higher risk of DRUJ instability than a midshaft fracture [13].

Aetiology of Acute Instability

The most common cause for DRUJ instability is a distal radius fracture. Dorsal angulation of the radius induces palmar instability of the ulna. Angulation greater than 20–30° creates marked incongruity of the DRUJ, distorts the TFCC, and alters joint kinematics [14, 15]. The radioulnar ligaments can tolerate no more than 5–7 mm of radial shortening before one or both ligaments tear [16]. The TFCC typically tears at its ulnar attachments [17]. As the severity of injury increases, the secondary stabilizers of the DRUJ and other structures on the ulnar side of the wrist sustain injury, including the IOM, ECU sheath, ulnocarpal ligaments, and lunotriquetral interosseous ligament.

The majority of ulnar styloid fractures do not cause DRUJ instability. The styloid shaft provides attachments for portions of the ulnocarpal ligaments, ECU tendon sheath and superficial limbs of the radioulnar ligaments while the styloid tip is devoid of soft-tissue attachments. Thus, a fracture through the base of the styloid is more predictive of a TFCC tear than the more common fracture through the tip [18]. Despite the shaft providing attachments for several stabilizing ligaments of the DRUJ, the radioulnar ligaments also have substantial attachments at the fovea of the ulnar head. A basilar fracture may thus occur without complete disruption of these ligaments. Conversely, a complete avulsion of the radioulnar ligaments and gross instability can occur without any type of styloid fracture [16, 17]. Occasionally, a small fleck of bone is avulsed from the fovea indicating disruption of the deep limbs of the radioulnar ligaments. These variations of injury must be recognized to avoid pitfalls in treating instability. For example, fixation of an ulnar styloid fracture will not be sufficient if the integrity of the radioulnar ligaments is not restored.

Presentation

Although the radius together with the carpus comprise the mobile unit of the DRUJ, by convention DRUJ dislocation or instability is described by the position of the ulnar head relative to the distal radius. Traumatic TFCC injuries usually result from combined rotational and axial forces to the forearm. The majority of isolated, irreducible or "locked," DRUJ dislocations are dorsal and caused by hyperpronation and wrist extension, usually in a fall on the outstretched hand. Conversely, volar dislocations are more subtle and occur in the supinated forearm or from a direct blow to the ulnar aspect of the forearm. Although most acute, complete dislocations are dorsal, chronic volar instability is probably more common but often missed because the diagnosis is more difficult to make.

Persistent symptoms following wrist injuries, especially after a distal radius fracture, are frequently caused by residual dysfunction of the

DRUJ and often misdiagnosed. Pain, reduced motion, weakness and mechanical symptoms may result. Once chronic instability develops it rarely improves spontaneously but symptoms may lessen and become tolerable in mild cases. It is unknown if instability predisposes to arthritis.

Physical Examination

An acute dislocation usually produces an obvious deformity with the ulnar head locked over a rim of the sigmoid notch. Because the DRUJ, ulnocarpal joint, lunotriquetral joint, and proximal radioulnar joint are closely linked anatomically and functionally, an examination of all joints and structures is essential to ensure that symptoms are being attributed to the correct source. Increased anteroposterior translation of the ulna on the radius during passive manipulation is evidence of DRUJ instability. Because joint translation varies with forearm position and among individuals, the test must be done in all forearm positions and compared with the opposite side. The examiner should inspect the DRUJ, wrist, and forearm both volarly and dorsally for swelling and for differences with the other side. A single palpating fingertip is used to elicit tenderness to best identify potential sites of pathology. Tenderness in the soft depression between the flexor carpi ulnaris (FCU) tendon, ulnar styloid, and triquetrum, is suggestive of a TFCC injury.

Local tenderness, swelling, and limited motion are the characteristic findings on presentation. Deep tenderness along the IOM and swelling or pain at the proximal radioulnar joint may indicate a concomitant Essex-Lopresti injury. Instability following reduction is marked by increased translation of the ulnar head in neutral forearm rotation and may also be present in supination or pronation depending on the injured soft tissue stabilizers. Accurate assessment of a DRUJ injury associated with a shaft fracture of the radius or ulna is much more difficult and is usually not possible until the fracture is reduced and stabilized.

Active and passive motion of the wrist and DRUJ are measured and compared with the opposite side. Decreased motion and crepitus during pronation/supination are signs of DRUJ arthritis, which may be accentuated by manually compressing the joint. ECU tendonitis and lunotriquetral ligament tears can mimic DRUJ symptoms. ECU subluxation is most apparent in supination and ulnar deviation. The lunotriquetral joint is assessed with the shear or ballottement test. In this test, the examiner stabilizes the lunate between the thumb and index finger of one hand while manually shearing the triquetrum against the lunate articular surface in a dorsopalmar direction with the thumb and index finger of the other hand. Pressing and manipulating the pisiform will elicit pain and crepitus in pisotriquetral arthritis.

Imaging

Plain radiographs of the wrist are initially reviewed for bony deformities from acute or old fractures, erosions or depositions from arthropathies and joint degeneration from posttraumatic or osteoarthritis. Two orthogonal views of the forearm should be obtained when forearm deformity is suspected. Positioning is key to obtaining good plain wrist films. A zero-rotation PA view is achieved by placing the humerus at 90º of abduction and the elbow at 90º of flexion on a flat surface. Signs of DRUJ instability in this view are: an ulnar styloid base fracture, a fleck fracture from the fovea, widening of the DRUJ, and greater than 5 mm of ulnar minus variance compared to the opposite [19]. This view should also be inspected for evidence of carpal instability and ulnar impaction syndrome.

A true lateral radiograph is obtained with the arm at the patient's side and the elbow flexed 90º. An accurate view is marked by the pisiform palmar surface visualized midway between the palmar surfaces of the distal pole of the scaphoid and the capitate or the midpoint of the hook of the hamate. Other evidence of correct alignment include superimpositions of the lesser four metacarpals, the proximal pole of the scaphoid on the lunate and the radial styloid in the center of the lunate. However, the lateral view is imprecise for diagnosing DRUJ subluxation [20–22].

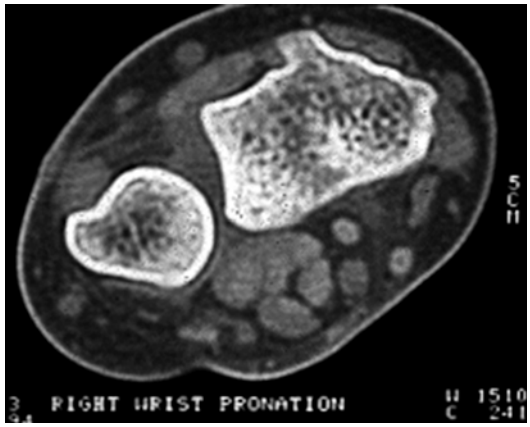


Fig. 17.3 CT scan showing volar subluxation

Fig. 17.4 MRI of peripheral TFCC tear. *Arrow* marking site of TFCC avulsion injury from its attachment at the fovea

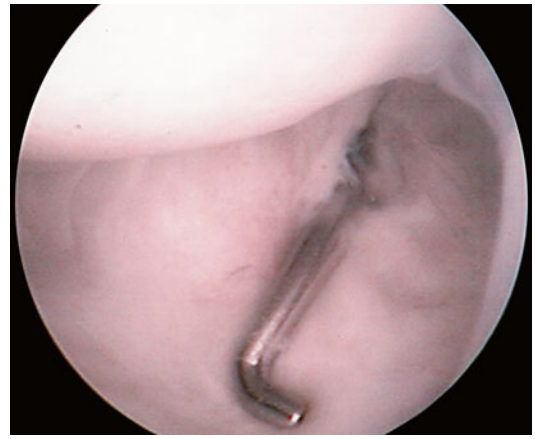


Fig. 17.5 Trampoline sign showing hypermobility of TFCC

Computed tomography(CT) has become the standard method for imaging DRUJ instability. To be a valuable study, both wrists must be evaluated on the same images and in identical forearm positions to make accurate comparisons between the normal and affected sides. It is important to align the forearms in the axis of the gantry and to image in neutral, supination and pronation. Because CT accurately delineates the cross-sectional anatomy of the DRUJ, it is also useful for assessing sigmoid notch competency, ulnar head deformity, and DRUJ arthritis and direction of instability, which are important in selecting proper surgical treatment (Fig. 17.3).

Magnetic resonance imaging (MRI) has been used to diagnose TFCC tears but its sensitivity, specificity, and accuracy vary among reports. Gadolinium or saline injection improves the detection of TFCC communicating defects. To be valuable in the management of DRUJ instability, MRI is used to detect peripheral lesions of the TFCC (Fig. 17.4). Similar to CT, MRI can be used to make anatomic measurements to assess subtle instability but it is more costly.

Other imaging modalities such as arthrography and scintigraphy have limited roles in assessing DRUJ instability but are useful when the diagnosis of instability is in question or other concurrent problems are suspected, such as ulnar impaction syndrome interosseous carpal ligament tears or a non-destabilizing TFCC tear.

Arthroscopy is sensitive for identifying TFCC tears in the central portion of the disk but incomplete peripheral tears are more difficult to detect and to judge severity. Injury to the TFCC periphery and tears of the lunotriquetral interosseous ligament or ECU sheath are all common with instability. A positive trampoline sign is indicative of a lax or hypermobile TFCC but does not establish the diagnosis of DRUJ instability (Fig. 17.5). In the assessment of DRUJ instability, arthroscopy

is most useful to evaluate symptoms that are inconsistent with instability or if other injuries are suspected of contributing to the complaints, especially if these can be treated by debridement alone. The value of DRUJ arthroscopy is limited because visualization is often poor.

Treatment Options

Isolated dorsal DRUJ dislocation is more common than palmar dislocation. When a DRUJ dislocation is recognized acutely, reduction is easily accomplished unless there is interposed soft tissue, such as the ECU tendon. Under appropriate anesthesia, gentle pressure is applied over the ulnar head while the radius is rotated toward the prominent ulna. After reduction, the joint should be tested over the full range of forearm rotation to determine the stable arc. Typically, a dorsal dislocation is most stable in supination, and a palmar dislocation in pronation. If the joint is only stable in extreme pronation or supination, additional treatment should be considered, such as radial-ulnar pinning in the position of greatest stability or TFCC repair. The TFCC is nearly always ruptured from the ulna [17, 23]. If the joint is stable in an acceptable position of forearm rotation, it is treated by an above elbow cast in this position for 3–4 weeks followed by use of a well-molded short-arm cast for 2–3 weeks. Interval evaluation with radiographs is recommended to assure a stable reduction in the cast.

If instability persists after initial treatment, one option is to pin the ulna to the radius proximal to the DRUJ. Another option, particularly when treating open radius fractures with concomitant DRUJ instability, is external fixation of the wrist with additional outrigger fixation of the distal third of the ulna in the position of maximum stability. When severe or bidirectional instability exists, ulnar styloid fixation or open repair of the TFCC, combined with radial-ulnar pinning should be considered.

Ulnar head fractures and sigmoid notch fractures with or without an extensive distal radius fracture pose additional challenges.

TFCC Repair (Class 1B Injury)

Peripheral TFCC tears can be diagnosed and sutured to the capsule using arthroscopic-assisted techniques; however, the indications for this technique in the treatment of DRUJ instability are not defined [24]. Because the arthroscopic repair does not reattach the TFCC to its anatomic attachments on the fovea or the ulnar styloid, it may not be as effective as an open repair in this situation. My preference is to perform an open repair of the TFCC for DRUJ instability. Some authors have advocated an ulnar-shortening osteotomy in conjunction with either an open or arthroscopic TFCC repair to reduce the loads on the TFCC, especially in patients with positive ulnar variance [24].

A 5-cm skin incision is made between the fifth and sixth extensor compartments and centered over the ulnar head. The extensor digiti quinti (EDQ) sheath is opened and the tendon is retracted. The DRUJ is exposed through an "L"-shaped capsulotomy. The longitudinal limb begins at the ulnar neck and extends to the distal edge of the sigmoid notch. Care is taken to preserve the origin of the dorsal radioulnar ligament from the sigmoid notch. The transverse limb is made along the proximal edge of the dorsal radioulnar ligament and extends to the radial margin of the ECU sheath. The capsule is elevated and retracted proximally to expose the ulnar head and neck. The proximal surface of the TFCC is inspected for injury, especially at its attachment to the fovea. Fibrosis is typically present. If the TFCC is suitable for repair, its distal surface is exposed through a transverse ulnocarpal capsulotomy made along the distal edge of the dorsal radioulnar ligament. With the use of a 0.045-in. Kirschner wire, two or three holes are created in the distal ulna extending from the dorsal aspect of the ulnar neck to the fovea (Fig. 17.6). This site for the holes reduces the irritation from the suture knots compared with the subcutaneous ulnar border. Two horizontal mattress sutures (2–0 absorbable monofilament) are passed from distal to proximal through the ulnar periphery of the TFCC, which lies near the fovea, by entering through the ulnocarpal capsulotomy

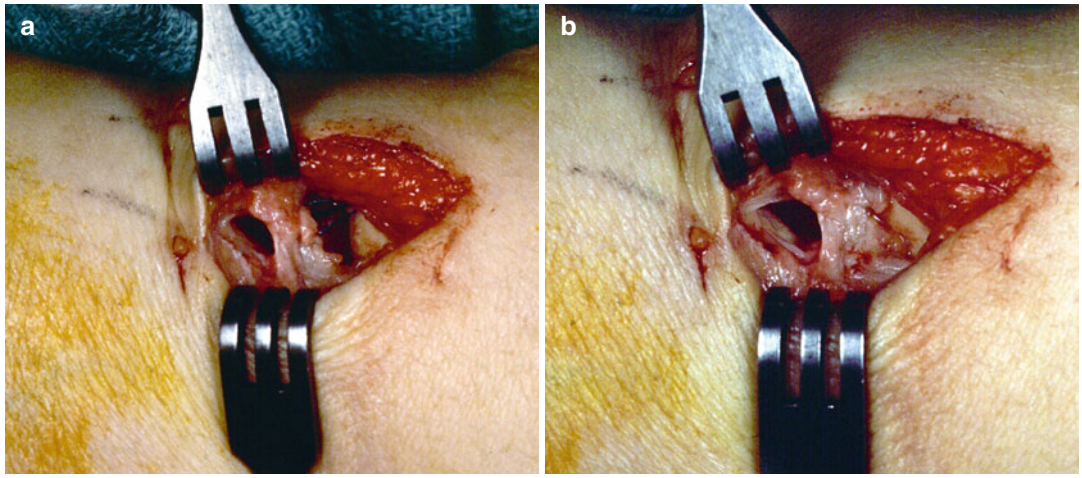


Fig. 17.6 (**a**) Open peripheral TFCC repair to fovea via transosseous sutures, with sutures in place but without the applied tension to the sutures. (**b**) Tension applied to the sutures to draw the TFCC down to its anatomic attachment at the fovea

and exiting through the DRUJ capsulotomy. With the use of a straight needle or a small suture passer, the sutures are passed through the bone holes. The sutures are tied over the ulnar neck with the joint reduced and the forearm in neutral rotation. The dorsal DRUJ capsule and retinaculum are closed together as a single layer but should not be imbricated, because loss of pronation may result. The extensor digiti minimi is left superficial to the retinaculum. A longarm splint is applied with the forearm rotated 45° toward the most stable joint position (e.g., supination for dorsal instability). At 2 weeks, the splint is converted to a long-arm cast for 4 weeks, followed by a well-molded shortarm cast for an additional 2–3 weeks. A removable splint is then used for 4 weeks while motion is regained. Strengthening and resumption of activities is delayed until near-painless motion is recovered.

Ulnar Styloid Fracture Repair

In his classic article, Frykman reported that ulnar styloid fractures occurred in approximately 61 % of distal radius fractures. Most of these fractures are not associated with DRUJ instability or long term symptoms. Fractures through the tip of the styloid do not require intervention because they do not cause DRUJ instability and are associated with a good prognosis [25]. Fractures through the styloid base, especially when displaced, are associated with a higher risk of DRUJ instability [18], although recent studies have cast doubt on the need to routinely fix even displaced styloid fractures [26–29]. Fixation of the styloid will restore DRUJ stability provided the TFCC is not otherwise damaged. A variety of methods have been described to fix fractures of the ulnar styloid, including Kirschner wires, tension band wiring, compression screw, variable-pitch headless screws, mini-fragment plates, and suture anchors. The size of the fragment often dictates the fixation options. Hardware irritation with need for hardware removal is not uncommon owing to its subcutaneous location on the ulnar border of the wrist.

The styloid can be exposed through a dorsal incision if necessary for treatment of other injuries, but the preferred approach to the styloid is located just palmar and parallel to the ECU tendon. In the surgical approach, the sensory branch of the ulnar nerve is protected and the ECU sheath is preserved. When using a tension band technique, one or two oblique Kirschner wire(s) are passed through the tip of the ulnar styloid. A 24-gauge tension band is passed around the tip(s) of the wire(s) and through a hole in the

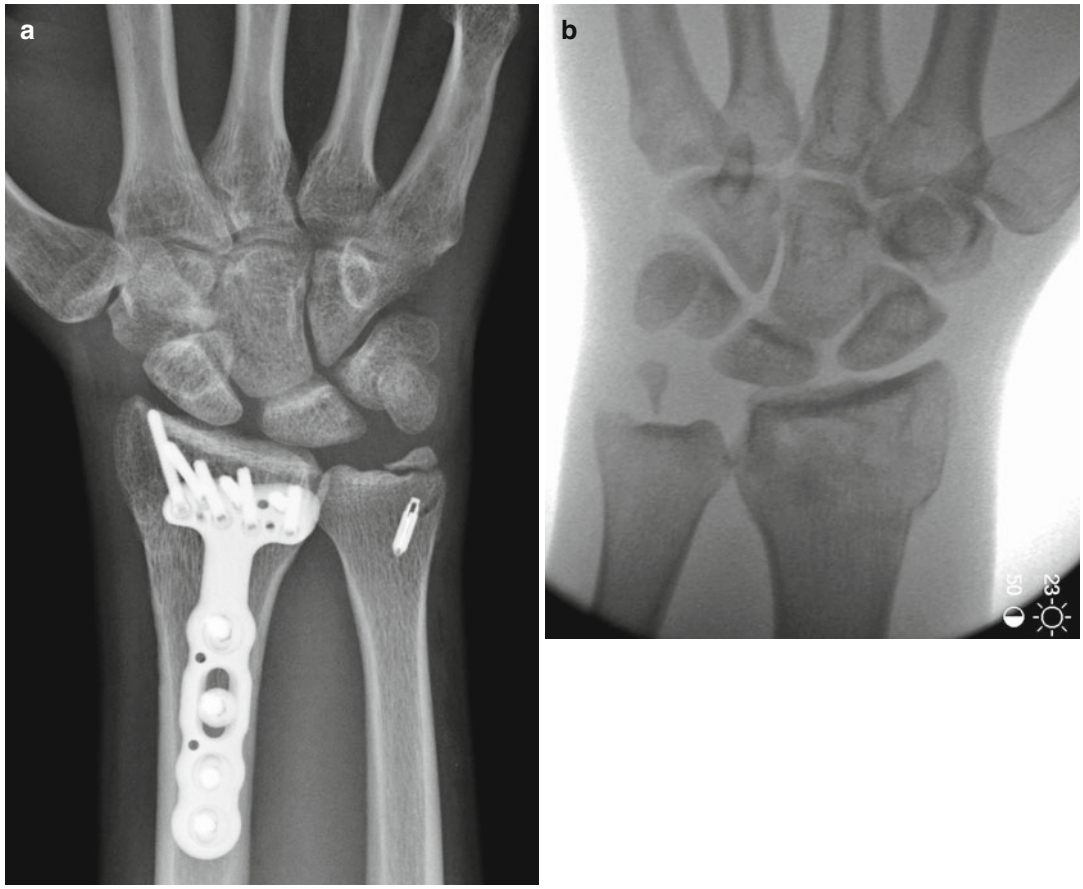


Fig. 17.7 (**a**) Suture anchor repair technique for an ulnar styloid fracture showing the displaced ulnar styloid. (**b**) Suture anchor inserted and styloid drawn to a near anatomic position

ulnar neck in a figure-of-eight fashion. Multiple Kirschner wires or a screw can be used for larger fragments.

I prefer to use suture anchor fixation to avoid irritating hardware. A longitudinal incision is created over the styloid, palmar and parallel to the ECU tendon. Care is taken to protect the sensory branch of the ulnar nerve, and the ECU sheath is preserved. A bone anchor is inserted through the fracture site and seated below the fracture line into the ulnar neck. The attached sutures are passed through drill holes in the styloid fragment if it is very large or passed around the fragment. The sutures are then crossed over the subcutaneous surface of the ulna and one end is passed through a transverse drill hole made near the ulnar neck to create a figure-of-eight. When the suture ends are tied it creates combined interosseous compression and a tension band. Stability is adequate to allow gentle early motion when allowed by the other injuries (Fig. 17.7).

The size and degree of displacement of the ulnar styloid fracture fragment are usually good predictors of DRUJ stability [30]. Although radiographic union is not consistently achieved with any technique, sound fibrous healing in a good position is generally compatible with resolution of symptoms and DRUJ stability. In my experience, ulnar styloid nonunion is rarely symptomatic unless the fragment is displaced or associated with DRUJ instability.

Conclusion

Restoration of stability and a full, painless arc of rotation are the goals of treatment for the unstable DRUJ. Acute DRUJ instability is commonly associated with distal radius fractures but usually respond to fracture fixation alone, however if an intra-operative clinical and fluoroscopic exams shows persistent instability then further treatment may be indicated. Similarly, simple dislocations nearly always respond to early closed treatment but open repair should be considered if the reduction is not stable. Good outcomes can be expected with a repair of the TFCC or ulnar styloid fracture. Complex injuries associated with ulnar head fractures or interosseous ligament tears require more comprehensive treatment. By recognizing and treating acute DRUJ instability the long-term complications of chronic instability can be avoided.

References

1. Adams BD, Berger RA. An anatomic reconstruction of the distal radioulnar ligaments for posttraumatic distal radioulnar joint instability. J Hand Surg. 2002;27(2):243–51.
2. Stuart PR, et al. The dorsopalmar stability of the distal radioulnar joint. J Hand Surg. 2000;25(4):689–99.
3. Tolat AR, Stanley JK, Trail IA. A cadaveric study of the anatomy and stability of the distal radioulnar joint in the coronal and transverse planes. J Hand Surg. 1996;21(5):587–94.
4. Wallwork NA, Bain GI. Sigmoid notch osteoplasty for chronic volar instability of the distal radioulnar joint: a case report. J Hand Surg. 2001;26(3):454–9.
5. af Ekenstam F, Hagert CG. Anatomical studies on the geometry and stability of the distal radio ulnar joint. Scand J Plast Reconstr Surg. 1985;19(1):17–25.
6. Kihara H, et al. The stabilizing mechanism of the distal radioulnar joint during pronation and supination. J Hand Surg. 1995;20(6):930–6.
7. Palmer AK, Werner FW. The triangular fibrocartilage complex of the wrist – anatomy and function. J Hand Surg. 1981;6(2):153–62.
8. Bednar MS, Arnoczky SP, Weiland AJ. The microvasculature of the triangular fibrocartilage complex: its clinical significance. J Hand Surg. 1991;16(6):1101–5.
9. Chidgey LK, et al. Histologic anatomy of the triangular fibrocartilage. J Hand Surg. 1991;16(6):1084–100.
10. Mikic ZD. Age changes in the triangular fibrocartilage of the wrist joint. J Anat. 1978;126(Pt 2):367–84.
11. Pirela-Cruz MA, et al. Stress computed tomography analysis of the distal radioulnar joint: a diagnostic tool for determining translational motion. J Hand Surg. 1991;16(1):75–82.
12. Ruby LK, Ferenz CC, Dell PC. The pronator quadratus interposition transfer: an adjunct to resection arthroplasty of the distal radioulnar joint. J Hand Surg. 1996;21(1):60–5.
13. Rettig ME, Raskin KB. Galeazzi fracture-dislocation: a new treatment-oriented classification. J Hand Surg. 2001;26(2):228–35.
14. Adams BD. Effects of radial deformity on distal radioulnar joint mechanics. J Hand Surg. 1993;18(3):492–8.
15. Kihara H, et al. The effect of dorsally angulated distal radius fractures on distal radioulnar joint congruency and forearm rotation. J Hand Surg. 1996;21(1):40–7.
16. Adams BD, Samani JE, Holley KA. Triangular fibrocartilage injury: a laboratory model. J Hand Surg. 1996;21(2):189–93.
17. Melone CP Jr, Nathan R. Traumatic disruption of the triangular fibrocartilage complex. Pathoanatomy. Clin Orthop Relat Res. 1992;(275):65–73.
18. Hauck RM, Skahen 3rd J, Palmer AK. Classification and treatment of ulnar styloid nonunion. J Hand Surg. 1996;21(3):418–22.
19. Moore TM, et al. Results of compression-plating of closed Galeazzi fractures. J Bone Joint Surg Am. 1985;67(7):1015–21.
20. Mino DE, Palmer AK, Levinsohn EM. The role of radiography and computerized tomography in the diagnosis of subluxation and dislocation of the distal radioulnar joint. J Hand Surg. 1983;8(1):23–31.
21. Mino DE, Palmer AK, Levinsohn EM. Radiography and computerized tomography in the diagnosis of incongruity of the distal radio-ulnar joint. A prospective study. J Bone Joint Surg Am. 1985;67(2):247–52.
22. Nakamura R, et al. Distal radioulnar joint subluxation and dislocation diagnosed by standard roentgenography. Skeletal Radiol. 1995;24(2):91–4.
23. Hermansdorfer JD, Kleinman WB. Management of chronic peripheral tears of the triangular fibrocartilage complex. J Hand Surg. 1991;16(2):340–6.
24. Trumble TE, Gilbert M, Vedder N. Ulnar shortening combined with arthroscopic repairs in the delayed management of triangular fibrocartilage complex tears. J Hand Surg. 1997;22(5):807–13.
25. Geissler WB, Fernandez DL, Lamey DM. Distal radioulnar joint injuries associated with fractures of the distal radius. Clin Orthop Relat Res. 1996;327:135–46.
26. Kim JK, Koh YD, Do NH. Should an ulnar styloid fracture be fixed following volar plate fixation of a distal radial fracture? J Bone Joint Surg Am. 2010;92(1):1–6.
27. Sammer DM, et al. The effect of ulnar styloid fractures on patient-rated outcomes after volar locking plating of distal radius fractures. J Hand Surg. 2009;34(9):1595–602.

28. Souer JS, et al. Effect of an unrepaired fracture of the ulnar styloid base on outcome after plate-and-screw fixation of a distal radial fracture. J Bone Joint Surg Am. 2009;91(4):830–8.
29. Zenke Y, et al. The effect of an associated ulnar styloid fracture on the outcome after fixation of a fracture of the distal radius. J Bone Joint Surg Br. 2009;91(1): 102–7.
30. May MM, Lawton JN, Blazar PE. Ulnar styloid fractures associated with distal radius fractures: incidence and implications for distal radioulnar joint instability. J Hand Surg. 2002;27(6):965–71.
31. Adams B. Distal radioulnar joint. In: Trumble TE, editor. Hand surgery update 3: hand, elbow, and shoulder. Rosemont: American Society for Surgery of the Hand; 2003. p. 147–57.

Massive Upper Limb Trauma

18

S. Raja Sabapathy and Hari Venkatramani

Keywords
Complex Upper Limb Trauma • Crush Injury Upper Limb • Revascularisation • Open Fractures • Flap Cover • Radical Debridement • Primary reconstruction • Staged Reconstruction

Introduction

The term, 'Massive Upper limb trauma', is difficult to define, though the terms massive injury, complex injury and severely mutilated upper limb are used interchangeably. Generally, the upper extremity could be considered to be massively injured when there is loss of or extensive injury to two or more functional components of the limb. Complex skeletal injury and significant soft tissue loss are common components in such injuries. These injuries, unless aggressively and appropriately treated will end up in amputation or in a severely compromised extremity. In the absence of a specific scoring system to relate the injury of individual functional components to disability, this definition would be good to identify the patients who would come under this group.

Massive upper limb injuries are sustained in road traffic accidents, in industries and in war. Use of body armour and head gear has reduced the mortality in the theatre of war, but the problem of the severely injured extremity remains. Better understanding of the pathophysiological responses to major trauma and rapid transport facilities to specialized treatment centres has increased the survival rate in polytrauma with the ultimate morbidity being closely related to the functional status of the upper limbs and the quality of vision. Aggressive management of the upper limb injury on day 1, advances in microsurgical techniques and managing it by a protocol based plan has made salvage of many of these injuries possible (Fig. 18.1). Surgeons involved in managing these injuries should have a trauma expert in their team who would look into the overall management of these patients because these patients are at great risk of severe systemic complications like prolonged hemorrhagic shock, systemic inflammatory response syndrome, and multiple organ dysfunction syndrome [1]. In this

S.R. Sabapathy, MS, MCh, DNB, FRCS (Edin), MAMS (✉)
Department of Plastic Surgery, Hand and Reconstructive Microsurgery and Burns, Ganga Hospital, 313- Mettupalayam Road, Coimbatore 641043, India
e-mail: rajahand@vsnl.com

H. Venkatramani, MS, MCh, DNB
Trauma Reconstructive Surgery, Ganga Hospital, Coimbatore, India
e-mail: drhariv@gmail.com

I.A. Trail, A.N.M. Fleming (eds.), *Disorders of the Hand: Volume 1: Hand Injuries*,
DOI 10.1007/978-1-4471-6554-5_18, © Springer-Verlag London 2015

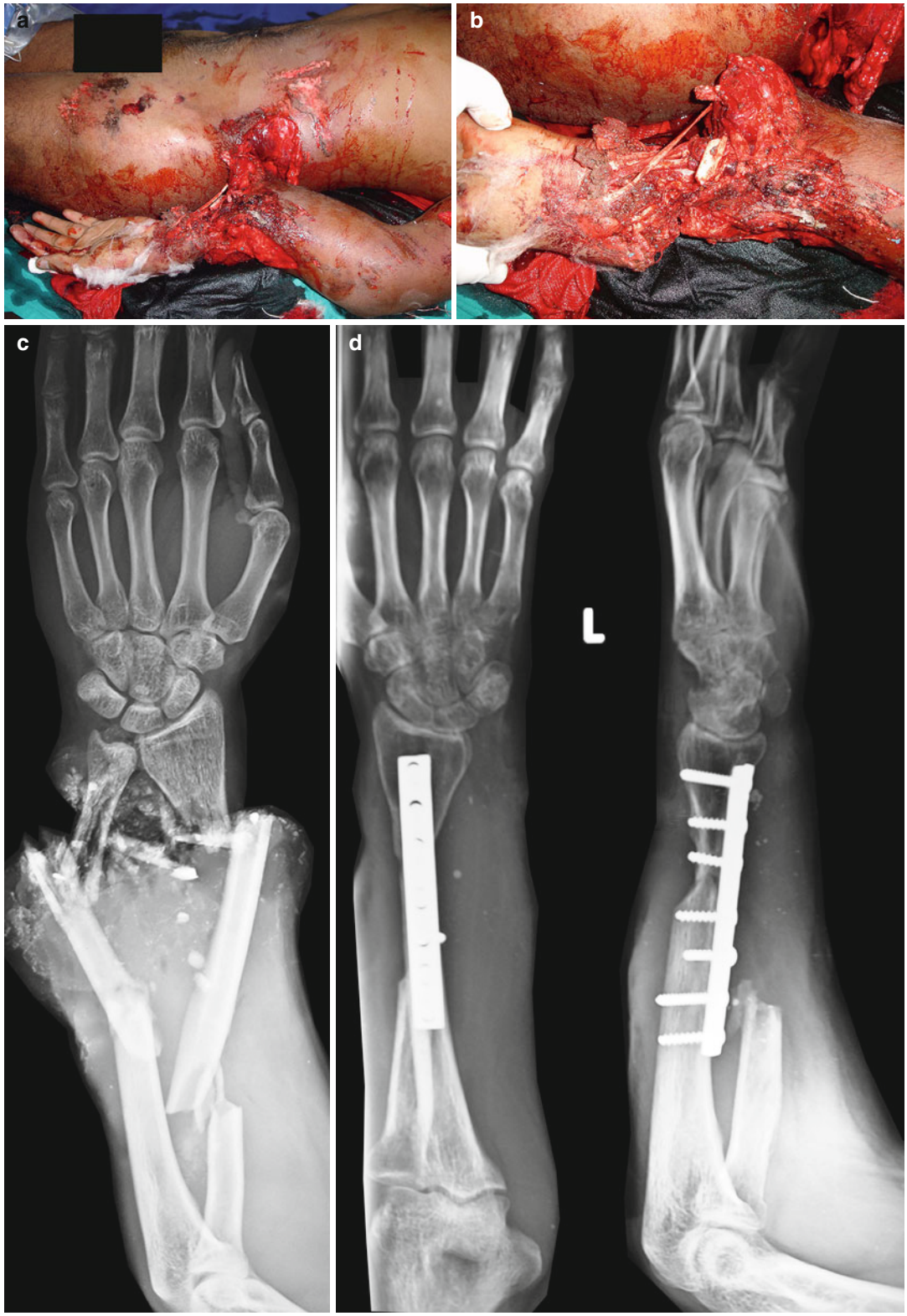

a
b
c
d
L

Fig. 18.1 (continued)

chapter the protocol and technical considerations that we have found effective to obtain a good functional outcome in the management of a massively injured upper extremity are discussed.

Assessment of the Patient with the Massively Injured Upper Extremity

A massively injured upper extremity draws so much of attention that it might lead an examiner to miss a critical associated injury to a vital organ. Hence it is mandatory to follow the standard ATLS protocols at the time of initial evaluation. Under no circumstances should this protocol be breached.

A few minutes must also be spent on eliciting relevant history. Even if the injury is severe, a few moments spent talking to the persons who accompanied the patient or who were at the scene of the accident could yield valuable information. The time duration since the accident will be a deciding factor regarding revascularization in a patient with proximal vessel injury. The time taken to extricate a limb in an industrial crush injury, the temperature of the injuring machinery and such similar details, all have a direct bearing on the treatment considerations and outcome. Presence of co- morbid diseases like diabetes, hypertension and chronic lung disease and drug history must be noted. More problems happen by not following this protocol than any other worthwhile procedure done in the first few minutes after the arrival of the patient. Unless the injured part is actively bleeding, this protocol should never be breached. If there is significant bleeding from the wound, a compression bandage and elevation are the first aid measures to be followed. This is the only situation that permits general examination to be done as the second step. Tetanus prophylaxis, antibiotic administration and pain relief are further steps in the line of management. Patzakis and Wilkins [2] have shown that early administration of antibiotics is essential. In their study patients receiving antibiotics within the first 3 h of injury had a significantly lower incidence of infection.

Pain Relief: The Concept of 'On Arrival Block'

Patients with a massively injured upper extremity are usually in severe pain. Early pain relief must be provided not only as a humanitarian duty, but also to improve the post therapeutic or post-surgical outcome. Ensuring adequate analgesia helps prevent or reverse some or all of the undesired

Fig. 18.1 (**a**, **b**) A 22 year old sustained a run over injury with fracture pelvis and crush injury left forearm. The wound was severely contaminated and the distal part was avascular. There was crushing of the tendons with segmental fracture of radius and ulna (**c**). After resuscitation stabilization of the fracture pelvis was done by an external fixator. It was followed by radical debridement, shortening of the bones and creation of one bone forearm (**d**) and revascularization. The wounds healed uneventfully and he went on to have a useful function of the hand (**e**, **f**)

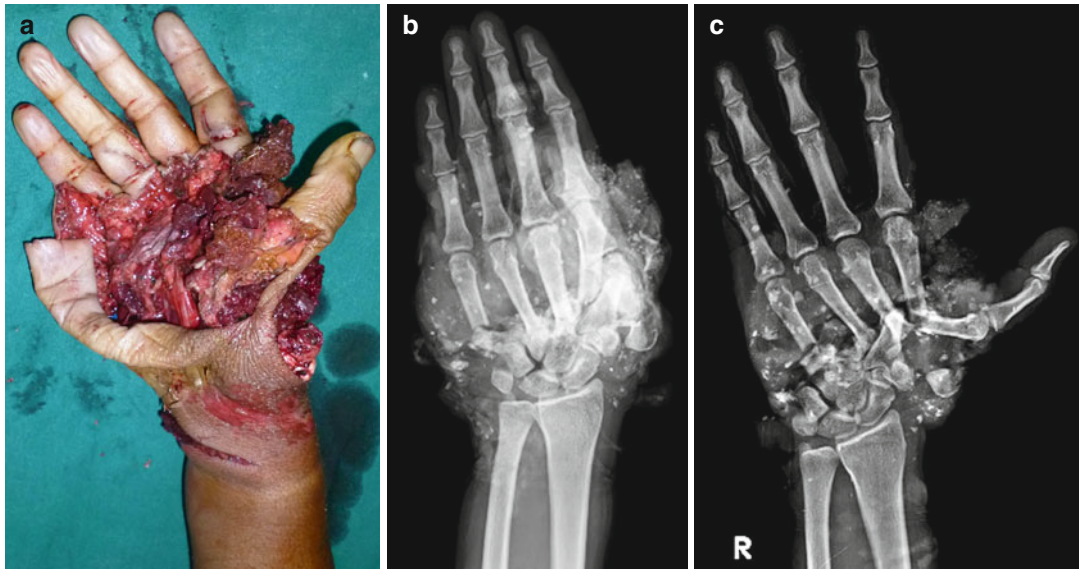


Fig. 18.2 (**a**) A machine crush injury of the hand. (**b**) Radiograph taken on arrival with the bandage. (**c**) Radiograph taken after brachial block on arrival which shows the fractures more clearly

cardiovascular, respiratory, gastrointestinal and psychological effects that accompanies major trauma [3]. In addition it provides a good deal of confidence to the patient in the system of care.

At Ganga Hospital Hand Injury Centre, all major upper limb injuries are received in the ante room of the Operation Theatre. This facilitates the presence of the senior surgeon and the anaesthesiologist on duty to be present to do the primary evaluation. Vascular status and the sensation in the hand are noted and a supraclavicular block is given to the injured extremity by the senior anaesthesiologist. 0.5 mg Bupivacaine, 2 mg per kg mixed with 2 % Lidocaine with Adrenaline (1:200,000), 7 mg/kg in 2:1 ratio is used for the block. The regional anaesthetic block provides immediate pain relief. We have found it to be a significant patient satisfaction outcome determinant. If the injured part is bleeding, it allows the use of a tourniquet. Radiographs are done after the block allowing proper positioning of the limb to obtain good quality radiographs. With a painless limb, manipulation of the limb is possible to obtain various views. It avoids overlapping of the bones at fracture sites, which would otherwise occur when radiographs are done with the compression dressing that the patients have when they arrive to the hospital (Fig. 18.2). The pain free status also allows the surgeon to better assess the injury and discuss the plans with the patient and the family. The massively injured upper extremity may need staged reconstructive surgical procedures and many months of rehabilitation. Gaining the confidence of the patient and the family during the stage of initial assessment is of paramount importance. Obtaining early pain relief is one of the most important measures to achieve this.

The only contraindication for the 'on arrival block', is the presence or suspicion of a brachial plexus injury. Supra clavicular blocks in the presence of a pre ganglionic injury of the brachial plexus can result in severe hypotension due to the anesthetic fluid seeping through the open dural sleeves and reaching the cord [4].

Assessment of the Massively Injured Upper Extremity

To Salvage or Not to Salvage?

Salvage of the upper limb still remains important because upper limb prosthesis usage and development does not compare favorably with that seen in

the lower limb. Wright et al. from the Mayo clinic reported an overall rejection rate of 38 % in upper limb prosthesis users [5]. In a recent review, the mean rejection rates of 45 and 35 % were observed for body powered and electric prosthesis in the paediatric population and it was 26 and 23 % respectively in the adult population [6]. As per the available evidence today, hand transplantation though feasible, has not found wide acceptance due to the cost of care and the effects of long term immunosuppression [7]. Current opinion seems to favor hand transplant only for bilateral amputees. Most of the massively injured upper limbs are unilateral injuries and salvage is preferable to later transplantation. Hence for the foreseeable future, upper limb salvage following massive trauma will remain important. In the developing world where the affordability of sophisticated prosthesis is less, salvage and reconstruction of the massively injured upper extremity takes priority. Even if functionally compromised, over a period of time the patients with the reconstructed upper extremities learn to use the available movements very well. Experience of the past two decades has convinced us that if the hand is structurally intact and is capable of function, efforts at reconstruction of proximal tissue losses can be made to obtain useful and satisfactory function in the hand (Fig. 18.3).

Unfortunately there are no scores which would accurately guide the surgeon to decide on salvage or amputation of a massive upper limb injury. Hence it

Fig. 18.3 (**a**) An avulsion injury at the wrist level. Removal of the dressing revealed an avascular hand with comminuted fractures of the distal radius and ulna with disruption of the carpus. (**b**) The hand at the end of debridement shows the distal part connected by long flexor the fingers and the median nerve. The hand salvaged by shortening of the forearm, arthrodesis of the wrist and revascularization of the radial artery and repair of the dorsal veins. (**c**) The critical structures in the raw area were covered by gracilis free flap attached to the ulnar artery which achieved primary wound healing. (**d**) Though the hand was shorter by 10 cm he went on to have useful function in the reconstructed hand holding weights up to 2 kg

Fig. 18.3 (continued)

Fig. 18.3 (continued)

is imperative to go through the protocol of salvage and decide on amputation of the upper limb only after assessing the patient under anaesthesia and better still the decision is made after debridement. At the end of debridement, a clearer picture emerges and a plan could be made with realistic expectations. The Mangled Extremity Severity Score (MESS), which is extensively used to predict lower limb salvage (a score greater than 7 for probable amputation), was not found to be an accurate predictor for salvage in the upper limb. While a score of less than 7 can predictably guide a team for salvage, a score of more than 7 must not be used to decide on amputation. The decision to amputate must be based on clinical judgment and individualized to every patient based on the findings of irreversible muscle ischaemia or impossibility of reconstruction [8, 9]. The experience, infrastructure and the skill levels of the surgical team also influence the decision and the ultimate outcome.

Debridement of the Massively Injured Upper Extremity

Debridement is the first and the critical step towards the salvage of the upper limb. If an 'on arrival block' has been given this anaesthetic can be used for the debridement. Efforts must be made to get the patient with a massively injured upper extremity onto the operating table as early as possible. If the upper limb injury is part of multiple injuries then these factors become more significant. In polytrauma situations, the duration of hypovolemia, hypoxia and hypothermia must be kept as low as possible. If infrastructure is developed to permit the initial assessment of the massively injured extremities in the ante room of the Operation Theatre, the time to incision will be much reduced. Steele et al. found this to be a significant factor in the outcome of polytrauma patients [10].

Debridement must be done under tourniquet, with good lighting and by an experienced surgeon. If the wound is badly contaminated, preliminary irrigation of the wound can be done to wash away the major contaminants. Irrigation alone cannot be relied on to remove all contaminants. It has to be done by surgery.

Realization of the value of primary reconstruction and its attendant benefits led to the concept of radical debridement [11]. Conventionally debridement was meant to remove all obvious contaminants and non-viable tissue, followed by a relook under anaesthesia 24 h later. If the wound bed is still not looking satisfactory, the procedure could be repeated every 24–48 h until the surgeon is satisfied of the adequacy of the debridement. When primary reconstruction came into practice, to avoid infection, debridement was done in a radical fashion making sure that all that was left was viable. Emphasis shifted from what was removed to what was left in the wound. This was termed 'wound excision' [12]. Since this is the crucial part of management, the technique is described in detail.

Technique

A thin rim of ragged skin margin is first excised. With sharp dissection a plane is found beneath the contaminants on the wound surface and the whole layer is excised. All intermuscular planes are inspected for contaminants. Under the tourniquet viable muscle looks homogenous, red and contains no hematomas. Non viable muscle is crushed and does not have a uniform appearance. If the tendons are avulsed from the musculotendinous junction, the muscles attached to the tendons are excised. Muscles attached to the avulsed tendons cannot be vascularized. Failure to remove these non-viable muscle bellies is one of the commonest cause of infection. The tendons could be retained for use in secondary reconstruction. Abraded bone surfaces are nibbled. Open joint cavities are irrigated with copious amount of fluid. Free bone fragments without soft tissue attachment are removed. Cortical bone without soft tissue attachment does not act as bone graft. Critical longitudinal structures like blood vessels and nerves are safe guarded. Even if their surface is contaminated, it is possible to find a tissue plane over their surface which could be excised to leave them free from contaminants. We call this 'skeletonization of longitudinal structures'. Time spent on doing this part of the procedure prevents infection which would eventually lead to secondary loss of vital tissues. In this way radical debridement differs from 'en bloc' excision practiced in oncological resections, the main difference being the painstaking efforts to retain the longitudinal functional tissues by carefully removing the contaminants.

We recommend the use of tourniquet during debridement. Otherwise even when the first incision is made the bleeding that occurs covers the whole area and may make one miss debriding the wound fully. Secondly in a massively injured upper extremity, contaminants in the intermuscular planes are easier to identify under tourniquet than with blood all around. Thirdly, if the raw area is large, the blood loss during debridement could be quite significant if the tourniquet is not used. These patients are usually haemodynamically compromised and preventing further loss of blood is good for them. During wound excision all open vessels encountered are coagulated with bipolar diathermy. At the end of surgical debridement the wound is irrigated well with fluid and the tourniquet released. Bleeding from the skin edges and the wound surface is inspected. Viable skin demonstrates bright red bleeding in the sub dermal plane. If certain tissues appear non-viable, the tourniquet can be reinflated and the wound further debrided. This procedure can be repeated until the surgeon is sure that the wound is free of contaminants and contains only viable tissue. The wound must look like a surgically created defect (Fig. 18.4).

Special attention is needed to assess degloved skin edges. As mentioned earlier, the indicator of skin viability is bright red bleeding in the subdermal plane. Dark colour bleeding in the subdermal area or in the subcutaneous plane is suspicious. The plane of degloving is always superficial to the deep fascia. So the degloved skin is bereft of the blood supply from the vessels that are in the

Fig. 18.4 (**a**) A dorsal combined loss in a road traffic accident which is extensively contaminated by road dust. (**b**) The status after copious irrigation and (**c**), after surgical debridement. Such radical debridement ensures primary healing of the wound when covered with a flap (**d**, **e**)

fascial plexus. Hence the extent of survival of the degloved skin depends upon the vessels that are in the base of the flap. Survival of the degloved skin is higher in the upper limb than in the lower limb because the number of perforators arising from the major vessels is greater in the upper limb than in the lower limb. If doubtfully viable degloved skin is retained, a second look must be planned in 24 or 48 h later. Non viable skin must be excised and no further delay is advisable. Avulsed skin flaps which are excised and amputated parts could be a source of skin, nerve or tendon grafts and consideration of this 'spare part' concept must be entertained before they are discarded [13].

Serial or staged debridement is advisable under certain circumstances when some patients may not be stable enough to withstand a long surgical procedure or in cases of major degloving, crush and electrical injuries when one cannot be

sure of the extent of damage. There is also the possibility that serial debridement can provide the opportunity to excise compromised tissue not obvious at initial debridement, although as Scheker and Ahmed comment, the development of edema and granulation tissue actually makes the visualization less likely on subsequent procedures [11].

There are a number of studies on the value of different types of solutions ranging from antibiotic containing solutions to tap water for wound irrigation purposes during debridement. Although wound irrigation with antibiotic solutions has been effective in experimental studies [14, 15], there is still a lack of convincing clinical data [16]. In our personal experience spanning two decades we have come to realize that precise surgical debridement and large volume irrigation is adequate. We use plain autoclaved water for irrigation.

During debridement, tight compartments must be released. If the injury needs revascularization it is better to do fasciotomy before revascularization. Performing fasciotomy immediately after revascularization causes profuse bleeding from the skin margins and the extent of fasciotomy may not then be complete. Fasciotomy and carpal tunnel release when indicated are better done under tourniquet during the stage of debridement.

Debridement – Clinical Pearls

Tourniquet up from beginning
Copious (sterile water) lavage of obvious contaminants
Fasciotomy, as necessary, before revascularization or tourniquet down
Wound edge excision
Avulsed muscle(s) debrided but retain tendons
En-bloc resection of dead muscle/contaminants
Nibble contaminated bone, remove loose fragments, wash joints out
Skeletonisation of longitudinal structures (nerve, vessels)
Plan second looks as needed (especially for degloved skin)

Reconstruction of the Massively Injured Upper Extremity

Revascularization Considerations

Two things have to be ascertained as part of early assessment of the injured extremity. One is viability of the limb and the other is the presence or absence of nerve injury. While a viable limb is obvious, one must have a high index of suspicion to recognise a limb which has vascular insufficiency. The finger tips are paler and blanching on pressure is slower. A period of less than 3 s is conventionally thought to indicate good perfusion, but the best way is to compare it with the opposite hand. Pulse oximetry should show a pulsatile flow. When a combination of absent pulses, delayed capillary refill and absent pulse oximeter tracing exists in an injured limb a diagnosis of major vessel injury must be made and never attribute this to spasm of the vessels. In an open injury the site of injury would be identified during debridement and angiograms are not needed. An attempt must be made to revascularise the limb as early as possible (Fig. 18.5).

The more proximal the level of injury, the greater is the urgency to revascularise the limb. Delayed revascularization of a proximal limb injury has as much a risk of reperfusion injury as replantation. Reperfusion injury is directly related to the ischemia time and the extent of the ischemic muscle mass. In replantation, initial vascular shunting could be done. The arterial repair can be done first and clamps released for a few minutes to wash away the metabolites before opening up the venous repair. In a partially attached limb, isolating the limb circulation after arterial repair is not possible and blood immediately enters the systemic circulation after clamp release. Hence caution must be exercised in revascularization of proximal injuries when the ischemia period goes beyond the recommended period It is difficult to give an accurate safe limits for revascularization of proximal injuries, because it will also depend upon the extent of collateral circulation that is present. Generally the same guidelines for replantation are followed [17]. A clinical guideline that we use is to check for the stiffness of the thenar

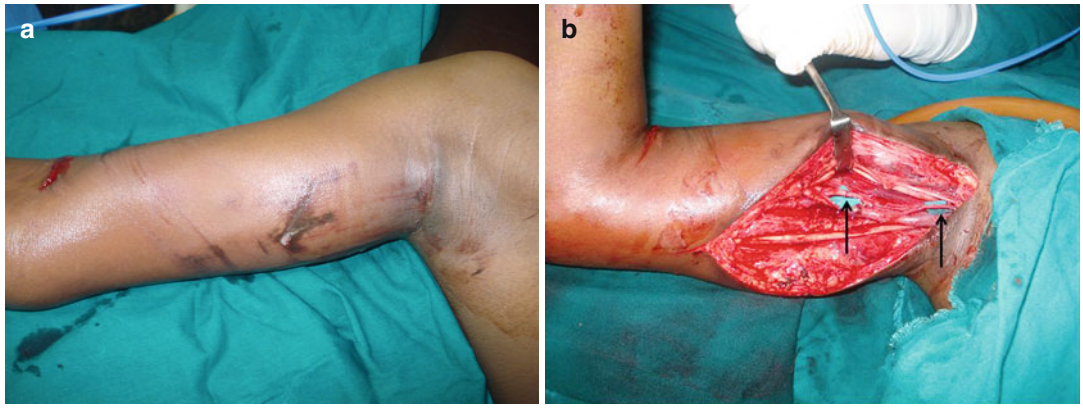


Fig. 18.5 (**a**) A closed crush injury of the arm. There was absent radial pulse, no pulse oximeter reading and Doppler probe showed abrupt stoppage of signals at the site of injury. Exploration revealed a segmental thrombosis of the brachial artery which was repaired with a vein graft (between *arrows*) (**b**)

muscles. If the thumb is stiffer than usual on passive movement, then it is unsafe to revascularise a major crush injury.

Although collateral circulation is better in the upper limb than in the lower limb, repair of major vessels still must be undertaken as augmenting blood supply has significant advantages. It ensures better survival of tissues in the area of injury and local flaps, if needed, survive better in a limb with a pulsatile blood flow (Fig. 18.6). Vein grafts are often needed for vascular repair. If the vascular injury is associated with comminuted fractures, bone shortening may obviate the need for vein grafting, but there should be a low threshold to use vein grafts. In continuity vascular lesions raise the possibility of performing an embolectomy. In our experience in massively injured extremities there is no role for embolectomy. The thrombus in the vessel is mostly due to damage to the vessel caused by fractured bone or due to external compression. Usually there is a significant degree of intimal damage in the injured segment of the vessel which promotes thrombosis. It is safer to excise the injured segment and repair it than relying on an embolectomy (Fig. 18.7).

The quality of debridement has to be good whenever vascular repair is done. Infection around the site of vascular repair or a vein graft is a disaster. It will lead to thrombosis and may even result in a blowout. The site of vascular repair must be on a good vascularized bed and must have adequate soft tissue cover. Although skin grafts have successfully been applied on vessel repairs and we have also had some successes, we would consider it as a risk not worth taking. In the event of loss or poor graft take due to infection, the risk of thrombosis or a blowout is high. It may also occur a few of weeks after injury when the salvage options involve major efforts. Hence, we recommend that all vascular repair sites be provided with good soft tissue cover.

Skeletal Fixation Considerations

In the reconstruction of a massively injured upper extremity, skeletal fixation follows debridement and precedes the step of revascularization if this is needed. Stable skeletal fixation is a primary requisite for soft tissue reconstruction. In a massively injured extremity skeletal fixation poses some challenges. If the limb is avascular the bone fixation has to be done very quickly. Ischaemia time has to be short and speed of skeletal fixation is paramount to success in limb salvage surgery.

Internal fixation is preferred over external fixation in the upper limb and hence the debridement has to be adequate, lest infection ruins the entire reconstructive effort. Restoration of skeletal stability prevents the kinking of repaired vessels and facilitates tendon repair. A stable skeleton also relieves the pain and makes the patient comfortable

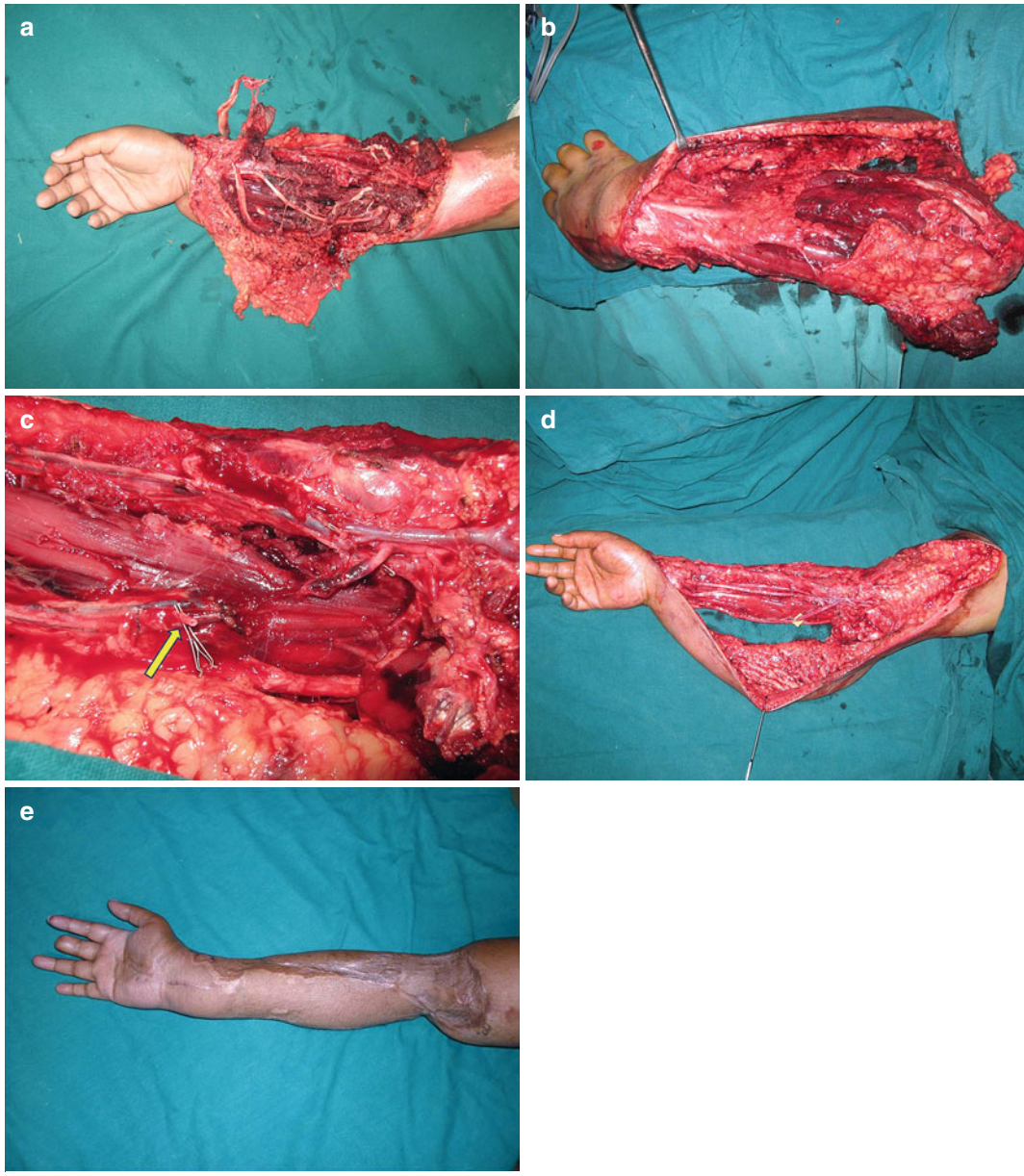


Fig. 18.6 (**a**, **b**) A major crush injury of right forearm with circumferential degloving injury with doubtful viability of the skin flap. The flow in the radial artery rendered the hand viable. (**c**) There was segmental loss of the proximal part of the ulnar artery *with the arrow pointing to the distal cut end of the ulnar artery*. (**d**) Repair of artery *with vein graft* made most of the long bipedicle flap viable through the perforators of the ulnar artery attached at the distal third of the forearm. (**e**) This provided the adequate skin to cover all the critical structures and obviated any need for the flap cover

allowing easy dressing changes and rehabilitation. The fractures may be severely comminuted and if there is no good soft tissue attachment the bone fragments may have to be removed. Bone fixation has to be done in the presence of bone loss.

It is an advantage if both the Plastic and the Orthopaedic team members are available at the time of debridement. It will help in deciding the impact of additional incisions in the viable flaps to provide exposure for skeletal fixation. If external

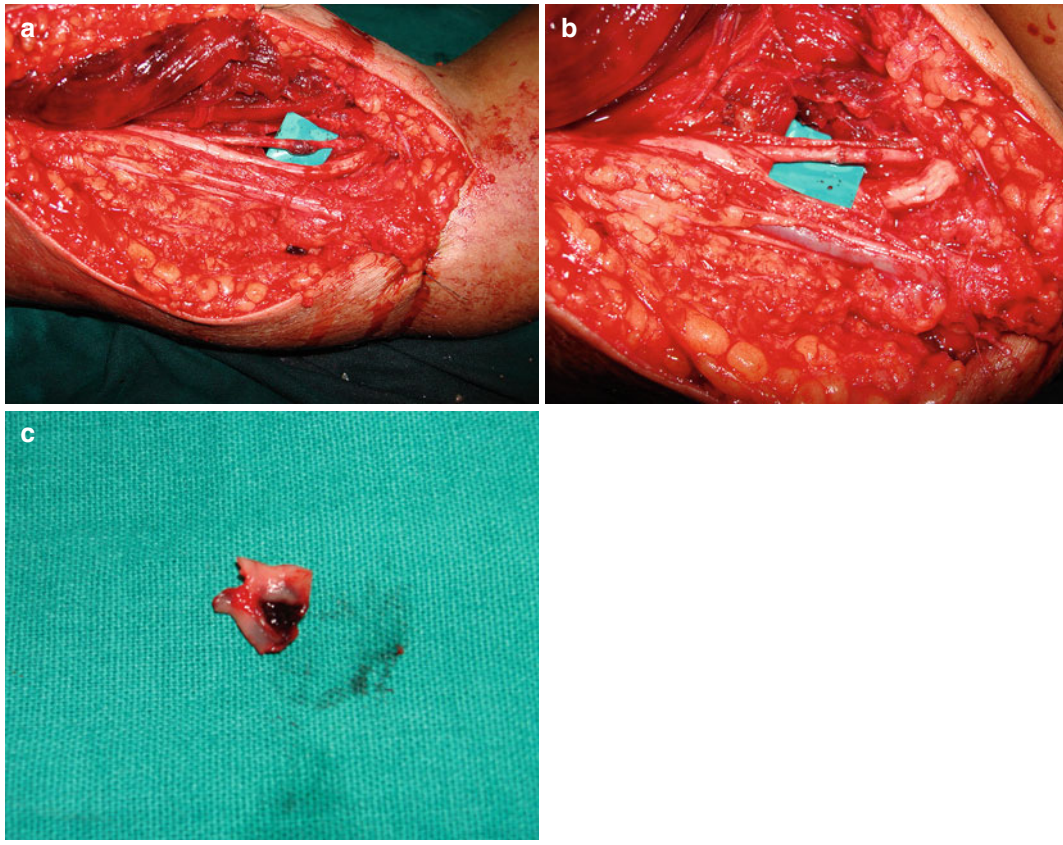


Fig. 18.7 (**a**) Crush injury at the elbow level with absent pulses due to thrombosis of the Brachial artery, which was excised and repaired (**b**). (**c**) The excised segment when cut open shows damage to intima which might lead to rethrombosis if only thrombectomy is done

fixation is chosen it should be planned without compromising the ease of subsequent flap cover.

Whilst precise indicators to assess the viability of skin and soft tissues like bright subdermal bleed for skin viability and contractility for muscle viability during debridement exists, such indicators have not been defined for bone. The assessment of bone viability is difficult and requires experience of judgment. Cancellous bone may be viable if there is about 50 % of soft tissue attached to it but cortical bone should have a substantial amount of soft tissue attached to it or else it will not be viable. The non-viable bone needs to be removed as it can be a source of infection but one should also not excise the bone too radically, as it may make the skeletal reconstruction difficult. Crucial fracture fragments like the articular fragment of the distal humerus should be debrided very carefully and as much soft tissue attachment as possible should be retained to maintain adequate blood supply to these fragments. For these fragments we err towards preservation as the removal of these would preclude restoration of the joint articular surface and hence the movements at that joint.

When there is severe comminution of bone involving the joint surface, associated skin loss must be addressed as early as possible. Exposure leads to loss of these bone fragments. Severe comminution at the distal radius and complex carpal injury may at times require fusion of the wrist joint. Patients tolerate wrist fusion well and are able to maintain good hand function (Fig. 18.3). If there is too much comminution or bone loss at the diaphysis, bone shortening and fixation is a simple and effective technique. The amount of bone shortening which can be done is

debatable, but about 7 cm of bone shortening gives an acceptable functional and aesthetic outcome. Shortening is relatively simpler in the humerus but technically more difficult in the forearm, as shortening has to be done in two bones while maintaining the symmetry of the distal radio-ulnar joint. In cases of fractures at different levels with bone loss, creation of a one bone forearm is a good option. If bone loss is extensive the skeleton may be stabilized externally to maintain the gap and later the bone gap can be filled with a free fibular graft (Fig. 18.8).

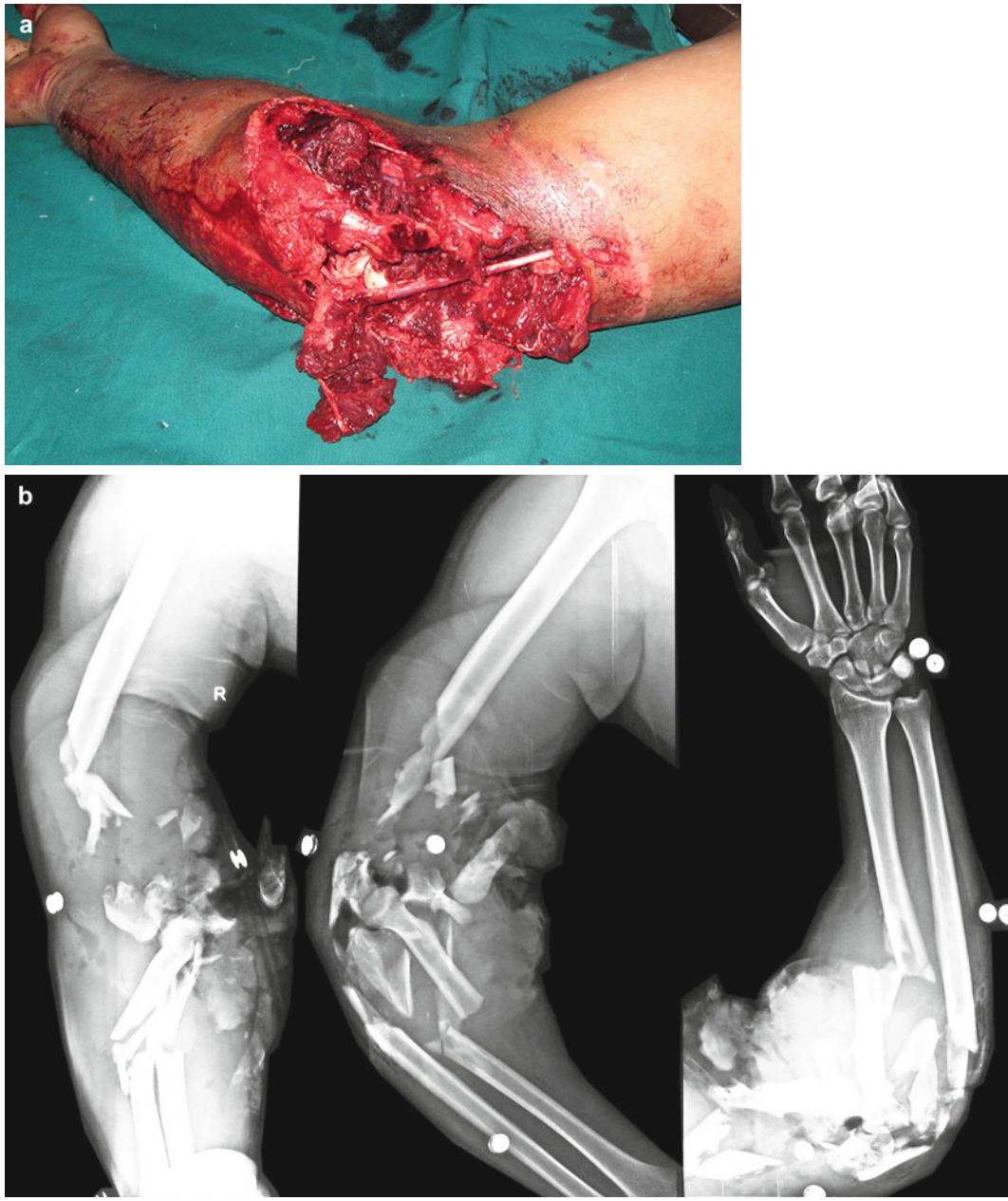


Fig. 18.8 (**a**) Sideswipe injury to the right elbow with soft tissue loss and severe communion of bone. (**b**) Debridement also involved removal of loose bone fragments and the elbow was stabilized by an external fixator. (**c**, **d**) Wound healing was achieved with an abdominal flap. After 3 months the bone gap was bridged with a free fibular graft (**e**) which went in for good union (**f**) and the patient gained useful function of the limb (**g**)

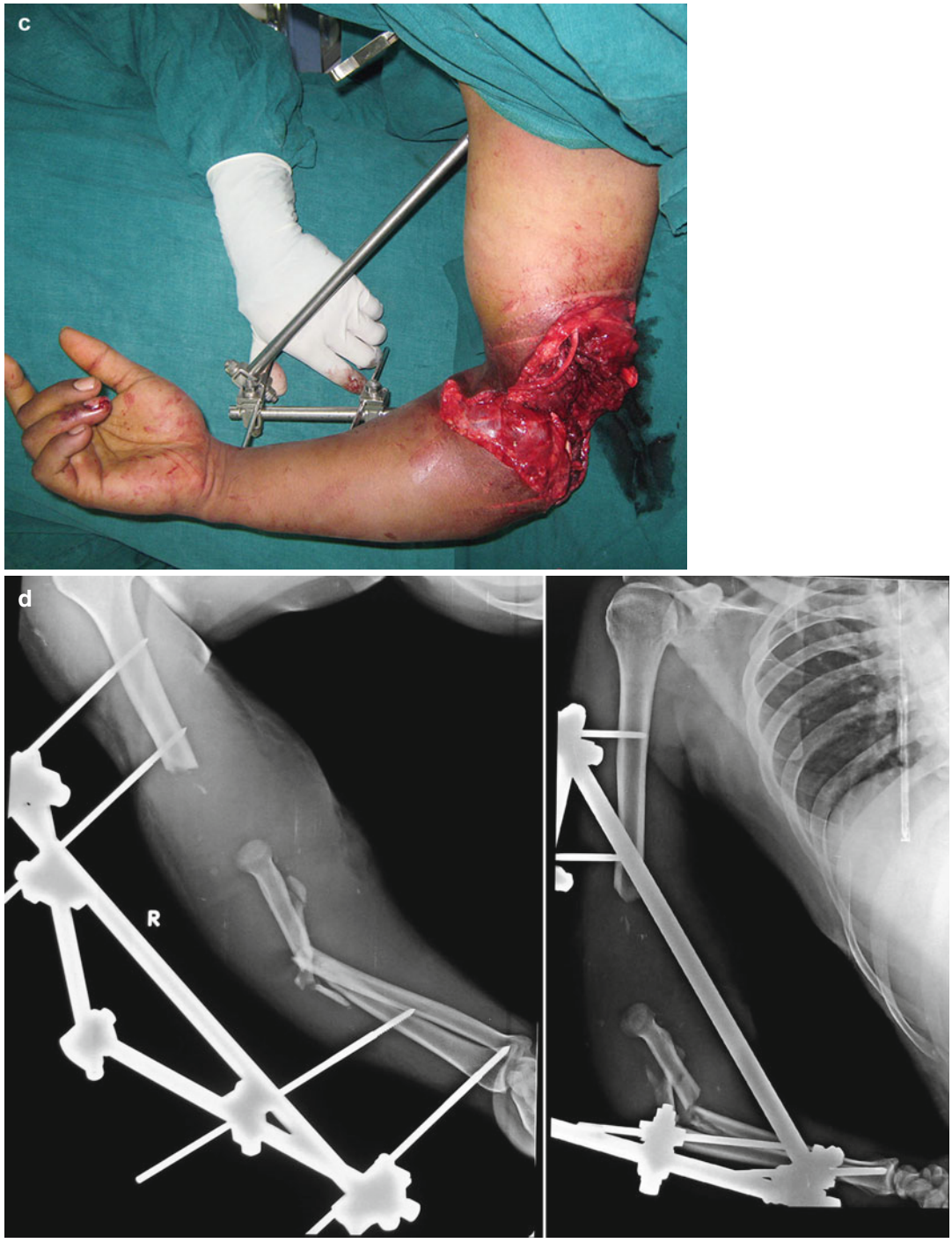


Fig. 18.8 (continued)

Fig. 18.8 (continued)

Repair of Functional Units

The functional outcome of salvage of a massively injured upper extremity depends upon the integrity or the quality of repair of functional units of the limb – the musculotendinous units and the nerves. While the survival of the limb depends upon the vascular repair, once vascularity is restored it does not affect the functional outcome. That is determined by the stability of the bone and the joints and the status of the musculotendinous units and the integrity or the quality of repair of the nerves. Every effort must be made to repair structures primarily. Standard techniques of muscle and tendon repair are used.

In a massively injured limb, musculotendinous avulsion is common. Muscle units tend to be avulsed at the musculotendinous junction and lie free attached at their insertion. All the muscle fibers attached to the tendons must be excised, since these muscles cannot be revascularised (Fig. 18.9). As much length of distal tendon as possible should be retained for use as distal attachment at the time of secondary reconstruction. It is also possible to weave the tendons through the viable proximal muscle and to secure these with non-absorbable sutures. It might result in some useful action. In massive injuries of the upper extremity segmental loss of musculotendinous units can occur. It is important to note and record the status of all important muscles as well as the level and location of the proximal and distal ends. This information is very useful when planning secondary reconstruction.

Primary repair of nerves improves outcome. Viable nerve ends need to be approximated without tension. Bone shortening at the time of skeletal fixation aids primary repair. In long segment avulsion of nerves, cross suturing of the available nerves like repair of the proximal ulnar nerve to distal median nerve may be done. Once the nerves are repaired, the level and the type of nerve repair done must be documented. If the nerves are not repaired it is important to document the position of the proximal and distal ends of the nerves. While planning secondary reconstruction, the proximal end of the nerve can be localized with Tinel's sign, but there is no clinical sign to localize the distal nerve end. Proper documentation will avoid unnecessary long exploration incisions during secondary reconstruction.

Total Primary Reconstruction Versus Staged Reconstruction

In segmental loss of musculotendinous units and nerves, debate exists about the role of primary tendon and nerve grafting and tendon transfers. Performing primary reconstruction has definite advantages in terms of early recovery of function, lesser number of surgeries and reduced cost. Sundine and Scheker [18] compared seven patients who underwent staged tendon and bone reconstruction of composite dorsal defects of the hand with seven patients who underwent immediate reconstruction of the bone and tendon defects and immediate soft tissue cover. The early reconstruction group had a significantly faster return to maximum range of movement, significantly fewer operations and a greater chance of returning to work compared to patients with staged reconstruction. The crucial factor for feasibility of primary reconstruction is the confidence of the surgeon in providing good soft tissue cover and the chances of primary wound healing. Whilst infection is a complication in other surgery, it is a disaster for a patient who has undergone total primary reconstruction as structures used as graft materials can be lost. Hence the deciding factor is not the technical feasibility of reconstruction of musculotendinous units or the nerves, but the quality of debridement and the status of the soft tissue cover. Primary reconstruction has to be followed by immediate flap cover. Associated injuries and the extra time under anaesthesia are also considerations in this decision making process.

Most units would defer primary nerve grafting unless the gap distance is small since the sources of nerve grafts are limited and one would prefer to use them in an ideal setting. Once nerve grafts have been done, further secondary reconstruction runs the risk of injuring these grafts and hence reconstruction of other structures has to be completed before performing nerve grafts.

Fig. 18.9 (**a**) A major crush injury of the forearm and arm with avulsion of the muscles. The muscles attached to the tendons are to be excised leaving the tendons for primary or secondary reconstruction. (**b**) The forearm was covered with a flap and the arm was covered with skin graft to achieve primary healing

While discussing the timing of reconstruction it will be safe to end with the words that whatever the nature of injury, debridement and skeletal fixation has to be done on day 1 and soft tissue cover has to be provided as early as possible and certainly before infection sets in. If tendon or nerve grafts are used the soft tissue reconstruction has to be immediate.

This also applies when the upper limb injury is associated with severe head, abdominal or chest injuries.

Provision of Soft Tissue Cover

Early replacement of skin and soft tissue loss is a vital component of the management of most massively injured upper extremities. There are several options available including skin graft, local flaps, distant pedicled flaps and free flaps. If at the end of debridement it is felt that flap cover is necessary, almost always the critical area would benefit from flap cover. Regular dressings and VAC application may reduce the need for flap cover, but if the area requires secondary reconstruction, provision of flap cover makes subsequent access easier. Though the choice of the flap is usually influenced by the training and experience of the surgeon, the following principles need to be kept in mind while choosing and providing soft tissue cover.

The first principle is to achieve soft tissue cover as early as possible. Primary radical debridement is a pre requisite for success. Exposed denuded bone, tendons and nerves do not tolerate desiccation and early cover prevents loss of these tissues. Godina in 1986 emphasized the importance of radical debridement and "early" tissue cover within the first 72 h. Though the work was mainly done in the lower limb the principles are also applicable to the upper limb [19]. Lister and Scheker took it forward and coined the term emergency free flaps where flap cover of the wound was achieved within 24 h [20]. In their series of 31 cases, the average hospital stay was 11.8 days. The flap survival rate was 93.5 % and 27 of the 31 patients returned to work, 18 of them to their original work. This approach has many proponents now but it requires a skilled team and infrastructure support. Ninkovic et al. [21] reported on 29 patients who underwent 27 emergency free flaps and three emergency toe transfers, after radical debridement and primary reconstruction of all injured structures. There was no flap failure or wound healing related complications.

In choosing the type of cover we have moved away from choosing the simplest to one which will offer the best possible outcome. In our experience we have not found a significant difference in the incidence of infection between local flaps, pedicled flaps or free flaps. What matters is directly related to the quality of debridement which is done prior to flap cover. In massively injured upper extremities which have extensive soft tissue loss, soft tissue reconstruction is made with the needs of the future reconstruction in mind. If secondary reconstruction procedures are to be done, then a skin flap is better for making access incisions and for the passage of tendon grafts and nerves. Muscle flaps waste and become thinner with time while the skin flaps may become bulky when the patient puts on weight at a later period. Subsequent thinning of the flap may be necessary. Secondary thinning of flaps in major injuries has to be done with care so as not to damage the reconstructed structures.

In extensive losses, reconstruction is easier if we divide the raw area into critical and non critical areas. By critical, we mean areas which would definitely need flap cover. Non-critical areas can be covered with skin grafts (Fig. 18.10). In this way the reconstructive challenge could be managed better. Flaps like the radial forearm flap, anterolateral thigh flap and lateral arm flaps in addition to providing cover can also serve as flow through flaps to revascularise the distal part [22] (see Chaps. 5 and 6). A plethora of choices are available at the present time to the hand surgeon to make the ideal choice of cover.

Clinical Pearl – Reconstruction

Primary Reconstruction reduces cost and ensures quick recovery

Staged reconstruction would also yield good outcome

Stable skeletal fixation is important

Internal fixation is preferred in the upper limb.

Provide soft tissue cover as early as possible

Quality of tendon and nerve repairs determines the functional outcome

Secondary reconstruction when induration subsides and soft tissue becomes supple

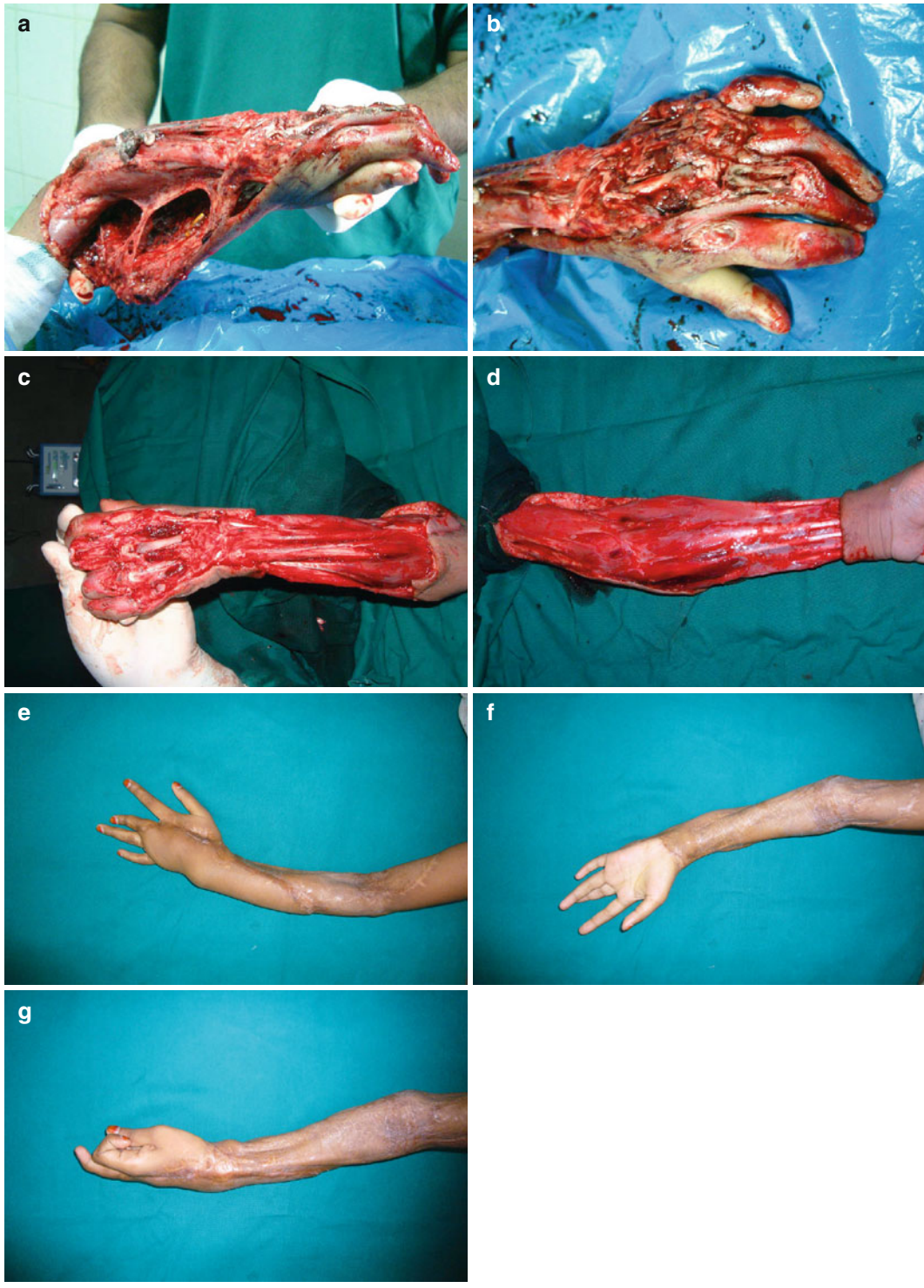


Fig. 18.10 (**a**, **b**) Crush injury of the forearm and hand in a 6 year old child with circumferential degloving of the entire forearm skin and dorsal composite tissue loss of the hand. (**c**, **d**) Post debridement pictures showing circumferential skin loss in the forearm and on the dorsum of the hand. (**e**–**g**) The critical raw areas are provided with flap cover and the rest of the raw area covered with skin graft. Dividing huge raw areas into critical and non critical helps in simplifying the reconstruction demand

Rehabilitation

Massively injured upper extremities would require long term supervised physiotherapy. A good rapport between the therapist and the patient is essential for success. Though in the overall care of the patient, the therapist would spend longer time with the patient than the surgeon, the surgeon is the first contact for any injured patient. The surgeon therefore has the opportunity to guide the patient regarding the need for therapy from day 1. We have found that acceptance of therapy protocols and compliance is better if the type of therapy and the duration of rehabilitation are briefly discussed with the patients while explaining about their surgery. Periodic evaluation of the progress of the patient in a combined clinic by the surgeon and the therapist is of great advantage. One can complement the other in guiding the patient and the timing of secondary reconstructions can be chosen at the appropriate time.

Secondary Reconstruction

The functional status of most patients with massively injured upper extremities can be improved by secondary procedures. The timing of the secondary procedure is important. The prime determinant is the status of the skin cover and subsidence of induration that accompanies any major injury. Usually the limb is ready for secondary procedures by about 3 months. Common procedures are bone grafting for non-union and reconstruction of functional units. If nerve reconstruction is to be done, it is done within 3 months. All other procedures are done along with nerve reconstruction particularly if nerve grafts are used. This is because, the flimsy nerve grafts may be damaged during access for later procedures. Tendon transfers, free functional muscle transfers and nerve transfers have increased the potential for enhancing function in massively injured upper extremities (Fig. 18.11).

Outcome

The outcome for these major injuries has much improved with the regular practice of radical debridement, stable skeletal fixation techniques and early soft tissue cover. Many disastrous situations have been turned into useful recovery. While the vascular status and repair determine survival, functional outcome depends upon the status of nerve and tendon repair. Managing these injuries requires attention to detail and each component of repair has to be done with utmost care and attention to get the best possible outcome. Objective measurement of range of movement and use of various scoring systems may variably project the outcome, but most individuals use their hand for their daily activities much more than the charts of range of movement would predict. This would involve keeping up the morale of the patient throughout the long process of rehabilitation. That demands greater time and effort from the surgeon and therapist than just doing the procedures themselves.

Even with primary fracture stabilization and early soft tissue management these cases sometime end with significant functional deficits if objectively measured in term of range of movement and scores but most would be using their reconstructed limb for day to day activities. That is considered as the success of the efforts of the Hand surgeon.

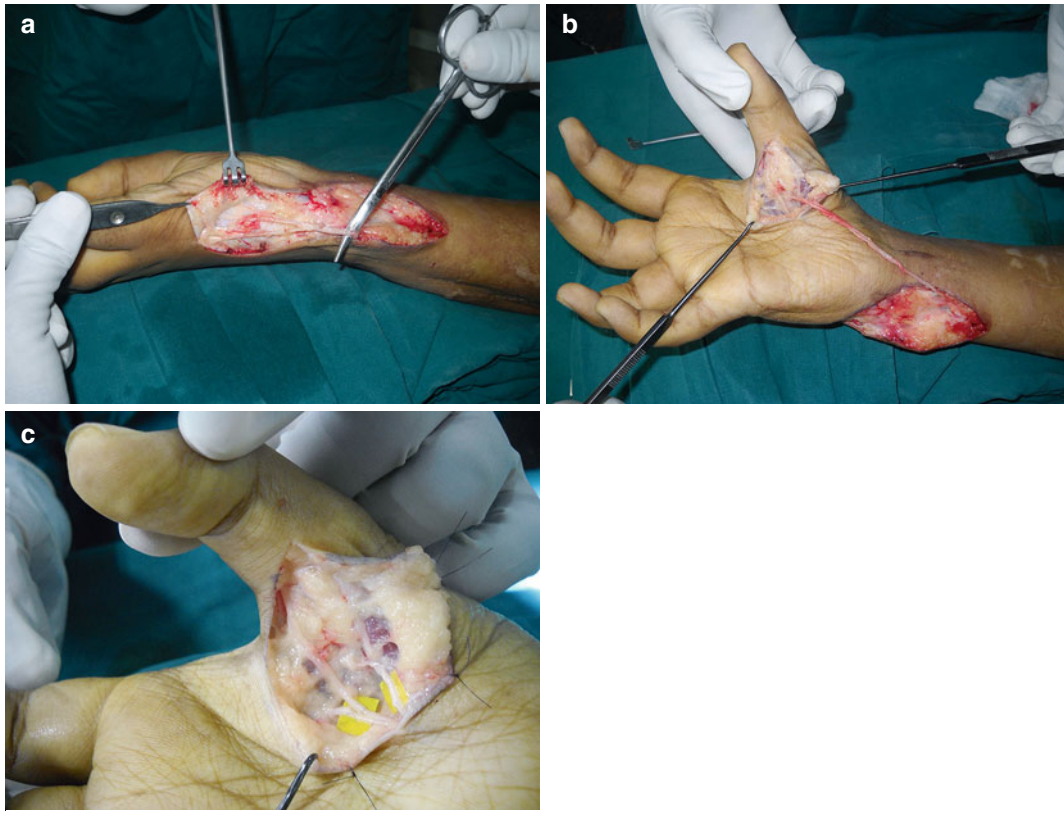


Fig. 18.11 (**a**) The dorsal sensory branch of the ulnar nerve was dissected in a patient who had a loss of 30 cm long length of median nerve. (**b**, **c**) Two branches of the nerve were repaired to the ulnar side digital nerve of the thumb and the radial side digital nerve of the index finger providing sensation to the contact surface for pinch activities

References

1. Gebhard F, Huber-Lang M. Polytrauma-pathophysiology and management principles. Langenbecks Arch Surg. 2008;393:825–31.
2. Patzakis MJ, Wilkins J. Factors influencing infection rate in open fracture wounds. Clin Orthop Relat Res. 1989;243:36–40.
3. Lovrincevic M, Kotob F, Santarosa J. Pain management in the trauma setting. Semin Anaesth. 2005;24:34–40.
4. Bhat VR, Maheshwari K, Sabapathy SR. Cardiac arrest following brachial plexus block in a patient with missed brachial plexus injury. Indian J Trauma Anesth Crit Care. 2003;4:177–8.
5. Wright TW, Hagen AD, Wood MB. Prosthetic usage in major upper extremity amputations. J Hand Surg Am. 1995;20A:619–22.
6. Biddiss EA, Chau TT. Upper limb prosthesis use and abandonment: a survey of the last 25 years. Prosthet Orthot Int. 2007;31:236–57.
7. Chung KC, Oda T, Saddawi –Konefka D, Shauver MJ. An economic analysis of hand transplantation in the United States. Plast Reconstr Surg. 2010;125:589–98.
8. Prichayudh S, Verananvattna A, Sriussadaporn S, Sriussadaporn S, Kritayakirana K, Pak-art R, Capin A, Pereira B, Tsunoyama T, Pena D. Management of upper extremity vascular injury: outcome related to the Mangled Extremity Severity Score. World J Surg. 2009;33:857–63.
9. Togawa S, Yamami N, Nakayama H, Mano Y, Ikegami K, Ozeki S. The validity of the mangled extremity severity score in the assessment of upper limb injuries. J Bone Joint Surg Br. 2005;87:1516–9.
10. Steele JT, Hoyt DB, Simons RK, Winchell RJ, Garcia J, Fortiage D. Is operation room resuscitation a way to save time? Am J Surg. 1997;174:683–7.
11. Scheker LR, Ahmed O. Radical debridement, free flap coverage, and immediate reconstruction of the upper extremity. Hand Clin. 2007;23:23–36.
12. Gupta A, Shatford RA, Wolff TW, Tsai TM, Scheker LR, Levin LS. Treatment of the severely injured upper extremity (Instructional course lectures, The

American Academy of Orthopedic Surgeons). J Bone Joint Surg. 1999;81A:1628–51.
13. Brown RE, Wu TY. Use of "spare parts" in mutilated upper extremity injuries. Hand Clin. 2003;19:73–87.
14. Dirschl DR, Wilson FC. Topical antibiotic irrigation in the prophylaxis of operative wound infections in orthopedic surgery. Orthop Clin North Am. 1991;22:419–26.
15. Rosenstein BD, Wilson FC, Funderburk CH. The use of bacitracin irrigation to prevent infection in postoperative skeletal wounds. An experimental study. J Bone Joint Surg Am. 1989;71:427–30.
16. Bakri K, Moran SL. Initial assessment and management of complex forearm defects. Hand Clin. 2007;23:255–68.
17. Sabapathy SR, Venkatramani H, Bharathi RR, Dheenadhayalan J, Bhat RV, Rajasekaran S. Technical considerations and functional outcome of 22 major replantations. (The BSSH Douglas Lamb Lecture, 2005). J Hand Surg Eur Vol. 2007;32B:488–501.
18. Sundine M, Scheker LR. A comparison of immediate and staged reconstruction of the dorsum of the hand. J Hand Surg Br. 1996;21B:216–21.
19. Godina M. Early microsurgical reconstruction of complex trauma of the extremities. Plast Reconstr Surg. 1986;78:285–92.
20. Lister GD, Scheker LR. Emergency free flaps to the upper extremity. J Hand Surg Am. 1988;13A:22–8.
21. Ninkovic M, Deetjen H, Ohler K, Anderl H. Emergency free tissue transfer for sever upper extremity injuries. J Hand Surg Br. 1995;20B:53–8.
22. Yildirim S, Taylan G, Eker G, Akoz T. Free flap choice for soft tissue reconstruction of the severely damaged upper extremity. J Reconstr Microsurg. 2006;22:599–609.

Hand Infections

19

Anthony Barabas and Andrew N.M. Fleming

Keywords

Hand infection • Paronychia • Herpetic whitlow • Felon • Flexor Tenosynovitis • Bursal infections • Deep space infections • Septic arthritis • Osteomyelitis • Necrotising fasciitis • Bite injuries • Mycobacterial infections • Fungal infections

Introduction

The hand is vulnerable to injury as mankind's primary tactile interface and tool for manipulating the physical world. The hands unique, compartmentalised anatomy and complexity of structure results in an abundant variety of possible hand infections. Each infection has its own characteristic presentation and patho-physiology which must be recognised and understood for successful treatment.

Acute infections of the hand normally commence following violation of the protective barrier of the skin or nail complex of the hand, beneath which an infecting organism may become established and multiply. The initial event is usually recalled by the patient and may be a puncture, burn, laceration, crush, or degloving injury. However the inoculation may also be through a barely discernible breach in the skin or even from a small hangnail.

Several non-infectious conditions may present with similar signs and symptoms to a hand infection. Crystalline deposition disease (e.g., gout, pseudogout), pyogenic granuloma, acute calcium deposition, acute nonspecific flexor tenosynovitis, rheumatoid arthritis, and foreign-body reactions can mimic an acute hand infection and should be considered in the differential diagnosis [1].

This chapter describes the aetiology and appropriate treatment of common and atypical hand infections which should optimise the outcomes in patients suffering from an infection of the hand.

A. Acute Infections
   1. Paronychia
   2. Herpetic whitlow
   3. Felon
   4. Pyogenic Flexor Tenosynovitis and bursal infections
   5. Deep space infections
   6. Septic arthritis

A. Barabas, BM, BSc, MRCS (Eng), FRCS
Department of Plastic Surgery, Hinchingbrooke Hospital, Huntingdon, UK
e-mail: tonybarabas@gmail.com; tonybarabas@hotmail.com;

A.N.M. Fleming, FRCS(Edin), FCS(SA)Plast (✉)
Department of Plastic Surgery, St Georges Hospital, Blackshaw Road, London SW17 0QT, UK
e-mail: andrew.fleming@stgeorges.nhs.uk

I.A. Trail, A.N.M. Fleming (eds.), *Disorders of the Hand: Volume 1: Hand Injuries*,
DOI 10.1007/978-1-4471-6554-5_19, © Springer-Verlag London 2015

7. Osteomyelitis
8. Necrotising fasciitis
9. Bite injuries

B. Atypical Infections
1. Mycobacterial infections
2. Fungal infections

Acute Infections

Introduction

Prior to the advent of antibiotics, there were few effective ways of combating infections caused by bacteria, other than surgery, often resulting in stiffness and contracture of the hand, and even selective amputation. However since the introduction of antibiotics in the mid 1930s, antibiotics are almost ubiquitously used to treat hand infections either as the primary treatment, or in conjunction with surgery.

Bone and soft tissue radiographs should be obtained early to evaluate for osteomyelitis or a foreign body. Tetanus prophylaxis should be administered when necessary. Rings should be removed from the affected finger and other fingers of the hand as soon as possible.

When diagnosed early hand infections may be amenable to treatment with elevation, oral antibiotics, and splintage for comfort [2]. The most commonly isolated organism is *Staphylococcus aureus*, implicated in up to 80 % of hand infections [3]. Empiric antibiotic coverage with a first-generation cephalosporin or anti-staphylococcal penicillin is usually adequate treatment for uncomplicated infections. Alternatively, a parenteral beta-lactamase inhibitor may be used as monotherapy. Thereafter Gram stain should guide initial antibiotic therapy. Severe hand infections require hospital admission for parenteral antibiotics [2, 4]. If an abscess is present, or if there is no improvement within 12–24 h, surgical intervention is mandatory.

Methicillin-resistant *S. aureus* (MRSA) has been increasingly reported in felons and other hand infections. Clinical response, as well as results of bacterial cultures, should be followed closely [5, 6].

With appropriate management intravenous therapy is not usually required beyond 3–5 days prior to conversion to a course of oral antibiotics. The recommended length of treatment varies from 5 to 14 days and depends on the clinical response and severity of infection [2].

Paronychia

The nail complex is composed of eponychium, bilateral paronychial (nail) folds, germinal matrix, sterile matrix, nail plate and hyponychium. Infection of the perionychium (the epidermis of the eponychium and paronychium bordering the nail plate) is called a paronychial infection, or more commonly just "paronychia". Paronychia may be either acute or chronic (longer than 6 weeks in duration) and is one of the most common infections of the hand. Paronychia is a superficial infection or abscess that develops after an invading organism gains access to the potential space between the perionychium and the nail plate. Paronychia frequently occurs after minor trauma (such as during dishwashing or a manicure), in nail biters and finger/thumb suckers (and hence frequent amongst children) and in the immunocompromised, but may occur from simply having a hangnail [7].

The infection develops in the groove between the paronychial or eponychial fold and the nail plate. There is localised erythema and swelling and is usually exquisitely painful. Fluctuance and local purulence indicate abscess formation, and may discolour, distort or raise the nail plate as it tracks above the sterile matrix. If the pus extends from one paronychial fold to the contralateral side it is termed a "run-around abscess" [8]. *S. aureus* and *streptococcus pyogenes* are the commonest infecting organisms followed by pseudomonas organisms. Gram negative organisms, herpes simplex virus, dermatophytes and yeasts have also been reported as causative agents. Finger suckers and nail biters are likely to culture oral organisms as a result of direct inoculation [9].

In the absence of an abscess oral antibiotics and warm soaks three to four times a day may be effective [7]. Children, finger suckers and nail

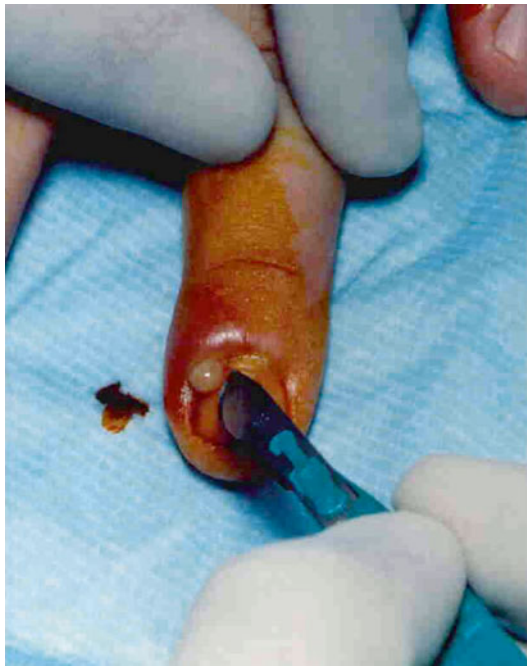


Fig. 19.1 Release of paronychia by elevating lateral nail fold

biters should be treated for anaerobes in addition to anti-staphylococcal therapy. Surgical treatment is required if an abscess has developed. Release of the pus may be achieved through a local anaesthetic digital block and simply by elevating the paronychial fold off the nail plate to induce spontaneous discharge (Fig. 19.1). If an abscess is pointing incision of the infarcted skin is painless even without anaesthetic, but for a thorough washout and debridement local anaesthetic is recommended, and the wound lightly packed to keep the edges open and prevent reaccumulation. More severe infection will likely require a wedge excision of the nail plate on the affected side, or if involving the whole of the nail complex, removal of the entire nail plate. All interventions should be followed by an appropriate course of antibiotics.

When chronic paronychia develops it is usually non-suppurative. People who are repeatedly exposed to water containing irritants or alkali, and those who are regularly exposed to moist environments (e.g. bartenders, dishwashers and swimmers) are at increased risk, and inflammation may be episodic associated with water exposure. Diabetics and immunosuppressed patients are also at higher risk [10]. Chronic paronychia leads to discolouration and thickening of the nail, with pronounced transverse ridges, and the nail folds may separate from the nail plate, forming a space for various microbes to invade. *Candia albicans is* cultured from 95 % of cases of chronic paronychia, however other pathogens, including atypical mycobacteria, gram-negative rods and gram-negative cocci, have also been implicated in chronic paronychia [8, 11].

Conservative treatment is the first-line treatment of chronic paronychia, with patients advised to avoid prolonged exposure of the hands to moist environments or other predisposing factors when identified. A topical antifungal combined with a steroid cream will often resolve a chronic paronychia through eradicating *C. albicans* infections. Oral antifungals are rarely necessary, and surgical intervention is only indicated when medical therapy fails. Removal of the entire nail and the use of an eponychial marsupialisation technique with application of an antifungal and steroid ointment to the nail bed had been reported to have excellent results [8, 9, 11]. In cases of recalcitrant chronic paronychia a sub-ungual or perionychial malignancy should be considered and biopsy sent for histological examination.

Clinical Pearls

Herpetic whitlow is treated non-operatively and should be clinically excluded prior to incision and drainage of a suspected paronychia, as this encourages bacterial superinfection

Incise paronychia's with blade facing away from the nail bed to prevent damage to the germinal matrix

Herpetic Whitlow

Herpetic whitlow results from inoculation of type 1 or type 2 herpes simplex virus into broken skin, and is the only common acute viral infection of the hand. Individuals at risk include dentists, oral hygienists, anaesthetists, nurses and other healthcare professionals. Auto-inoculation, particularly

amongst nail biters and finger suckers, can occur from direct contact with orolabial or genital infections, and infections following human bites have also been described [12, 13]. Contact sportsmen, especially wrestlers and rugby players, are also at increased risk of Herpes gladiatorum (otherwise known as herpes rugborium, scrumpox, wrestler's herpes or mat pox) by HSV-1, although the hand is much less frequently involved than the head or trunk [14–16].

Patients present with a throbbing pain out of proportion with initial physical signs, erythema and significant localised tenderness, followed by an eruption of vesicles as the virus infects epithelial cells and replicates. The vesicles coalesce and drain a clear to turbid fluid, and may mature into ulcers. Patients may also complain of systemic viral symptoms such as a temperature, malaise and lymphadenitis. The outbreak is normally self-limiting and resolves over a 1–3 week period. Diagnosis is clinical, but can be confirmed by a Tzanck smear (microscopy of scraping show multinucleated giant Tzanck cells), DNA amplification or viral cultures from the outer base of an ulcer [12, 17, 18].

Antiviral therapy with acyclovir, famciclovir, or valacyclovir treatment within the first 48 h of symptom onset is reported to lessen the severity of infection. Blisters should not be de-roofed or debrided as this can predispose to bacterial infection and may induce a viraemia. Antibiotics are therefore only of benefit if secondary bacterial infection occurs. The virus passes up affected nerves and remains latent in the dorsal root ganglion. 30–50 % of patients will experience recurrent infections, before which a tingling prodrome is often felt at which point starting antiviral therapy (topical or oral) may shorten or even prevent recurrence [19]. Viral shedding continues until any epidermal lesions have healed, and therefore affected areas should be covered to prevent further transmission.

Clinical Pearls

Warn patients of high chance of recurrence and provide a prescription for anti-viral treatment to be started as soon as tingling is experienced

Felon

A felon is an infection of the distal finger pulp. The most commonly isolated organism is *S. aureus* by direct inoculation from penetrating trauma. Common predisposing causes include splinters, glass and other minor puncture wounds. A felon may also arise when an untreated paronychia spreads into the fingertip pulp. Felons have been reported following multiple fingerstick blood tests [11]. The index finger and thumb are the most commonly involved digits [20].

The fingertip pulp is an enclosed fibro-fatty space which is subdivided by 15–20 fibrous septae running between the periosteum and overlying skin. The enclosed nature of the pulp results in rapid onset of a throbbing like pain. Septae prevent spread of infection to the distal interphalangeal joint (DIPJ). Instead the pulp appears swollen and erythematous, with high pressures within the pulp leading to tissue necrosis and a spontaneously draining sinus. Osteomyelitis of the distal phalanx may develop [21] and should be excluded by x-ray review.

An early felon can be treated with oral antibiotics and elevation alone. However, due to the rapid evolution of a felon, most patients present with fluctuance and established abscess formation and require surgical intervention. Incision and drainage should be performed under a digital block and sterile conditions. The drainage incision should be positioned over areas of tissue necrosis in a pointing or discharging felon, which will anyway require debridement. Otherwise a number of incisions have been described for closed felons. The preferred techniques are a single volar longitudinal incision or a high lateral incision (Fig. 19.2). Incisions that are not recommended are those that extend onto the hyponchyium/fingertip, such as a fishmouth incision or extended lateral "J" incision, as these produce painful sensitive scars [2, 11, 20, 22, 23]. Lateral incisions should be placed on the radial side of the thumb and ulnar side of the index, middle and ring finger distal phalanges to avoid pain or reduced sensation during pinch grip. The radial aspect of the little finger is preferable so as not to violate the "resting"

Fig. 19.2 Incisions for decompressing a felon: midlateral – *solid line*; volar pulp – *dashed line*

ulnar border of the hand. The incision location should avoid injury to the flexor tendon sheath, digital neurovascular structures and nail matrix, and should not need to extend proximal to the DIPJ. All septae are disrupted through blunt dissection, the wound debrided and irrigated and then lightly packed to allow drainage. Empiric antibiotics are started, and the digit dressed and elevated. The pack should be removed in 24–48 h, the digit inspected, and if satisfactory it is allowed to heal in by secondary intention [12, 18].

Clinical Pearls
Avoid sensitive fingertip incisions
Ensure decompression of all fibrous septae
X-ray distal phalanx to exclude osteomyelitis

Pyogenic Flexor Tenosynovitis

The flexor sheath is a fibro-osseous tunnel, lined by synovium, through which the flexor tendons of the fingers pass. The synovial fluid lubricates the sheath reducing friction and resistance to tendon glide. The flexor sheath of the fingers run from just proximal to the A1 pulley overlying the metacarpal head, to the A5 pulley at the base of the distal phalanx. The flexor sheath of the thumb extends from around 3 cm distal to the volar wrist crease to the distal thumb phalanx. The proximal half of this tendon sheath is commonly referred to as the radial bursa, although occasionally it is entirely separate from the distal flexor pollicis longus tendon sheath. The little finger tendon sheath (and occasionally the middle and ring finger sheaths) extend to and communicate with the ulnar bursa of the wrist. In the palm, the ulnar bursa extends laterally to envelop the superficial and deep tendons of the ring, middle, and index fingers, and extends 2–3 cm proximal to the proximal volar wrist crease. The radial and ulnar bursae communicate proximal to the carpal tunnel in 50–80 % of patients, and from here with the space of Parona in the forearm. Anatomic variations are frequent [12, 24].

Definition

Pyogenic flexor tenosynovitis is an infection between the visceral (on the flexor tendon) and parietal (the inner sheath wall) synovium of the flexor sheath. The most common organism is *S. Aureus* from penetrating trauma, especially at the volar joint creases where the flexor sheath lies superficial [12, 24, 25]. Spread to the radial and ulna bursa from thumb or little finger flexor sheath infections can occur, and where the two bursae are contiguous spread between them may result in a horseshoe abscess. Proximal spread into the space of Parona can also occur [24].

Clinical

Kanavel described four cardinal symptoms and signs of a flexor sheath infection, although not all need be present: (1) a symmetrically fusiform swelling of the digit; (2) pain on passive extension (most marked over the proximal volar end of the

Fig. 19.3 Fusiform swelling, semi-flexed position and palmar necrosis over A1 pulley in late presenting middle finger flexor sheath infection

digit); (3) tenderness from percussion over the flexor sheath; (4) the digit is held in a semi-flexed position [26] (Fig. 19.3). In a study of 75 patients, Pang et al. [27] found that fusiform swelling was most often present (97 % of patients), followed by pain on passive extension (72 %), semi-flexed digit posture (69 %), and tenderness along the flexor tendon sheath (64 %). Neviaser and Gunther [28] reported that the most reliable Kanavel sign is pain on passive extension of the digit. They also report that the inability of the finger to flex and touch the palm is another important clinical sign. Patients presenting early have less pronounced Kanavel signs, which will gradually evolve over time [24].

Once suspected urgent treatment should be instituted in treating flexor tenosynovitis. Early infections (presenting in less than 24–48 h) in physiologically healthy individuals can often be successfully treated with intravenous antibiotics, immobilisation and strict elevation under close observation [24, 28]. Most cases, however, require urgent operative intervention to prevent the potentially devastating complications of tendon adhesions, tendon necrosis or even devascularisation and necrosis of the digit, requiring amputation (Fig. 19.3).

Surgery

The flexor sheath is accessed via incisions at the distal palmar crease overlying the A1 pulley, and at the distal interphalangeal joint (DIPJ) where the flexor sheath ends. Mid-lateral, transverse and Brunners incisions have been described [2, 24]. We avoid Brunners incisions due to the risk of flap tip necrosis in the presence of infection. At the DIPJ we perform a transverse crease-line or mid-lateral incision, and a transverse incision proximally over the distal palmar crease. The flexor sheath is opened proximally and distally, the synovial fluid inspected and sent for culture. A narrow gauge catheter (commonly a paediatric feeding tube) is inserted proximally and distally, and the sheath flushed with at least one litre of normal saline, until clear. If the saline does not pass through the entire length of the sheath, or more importantly if the synovial fluid is very turbid, we advocate a third incision over the proximal interphalangeal joint to access the sheath. In cases of severe purulence, extensive exposure of the flexor tendons may be required in order to debride any infected or necrotic tissue.

Local protocol and the surgeon's preference will determine whether catheters are left in situ for continued closed tendon sheath irrigation on the ward post-operatively. No study had shown an advantage of post-operative intermittent or continuous tendon sheath irrigation over that of thorough intraoperative irrigation alone [29–31]. In contrast complications can occur from postoperative closed tendon sheath irrigation including increased digital stiffness by decreasing the patient's ability to participate in therapy, a foreign body reaction [24] and even tissue necrosis and digital amputation if the catheter tip is not correctly positioned within the sheath [32].

Intravenous antibiotics and elevation are continued post-operatively, and a further washout or inspection in theatre scheduled for 24–48 h if clinically indicated. The wounds are left to heal in secondarily or lightly tacked together, hand therapy is started prior to discharge and the patient sent home on a course of oral antibiotics.

Outcome

Despite treatment 10–25 % of patients fail to obtain a full range of motion in the digit [30, 33, 34]. Risk factors for a poor outcome include age over 43 years, diabetes mellitus, peripheral vascular disease, renal disease, subcutaneous purulence, digital ischaemia, and polymicrobial infections [27].

Radial and Ulnar Bursae and Space of Parona Infections

Normally as a consequence of a flexor sheath infection, surgical treatment for radial and ulnar bursal infections is combined with that for the flexor sheath. Symptoms may include acute carpal tunnel syndrome (CTS) and pain with finger or wrist motion. Management is similar to that of other deep-space infections, emphasising wide exposure and thorough drainage. Zig-zag or curvilinear palmar incisions should progress proximally into a standard carpal tunnel incision if CTS is present. A separate longitudinal incision is made radial to the flexor carpi ulnaris tendon at the wrist to proximally irrigate the ulnar bursa or to access the space of Parona. The proximal incision for irrigation of the radial bursa is just ulnar to the flexor carpi radialis in the distal forearm [2, 25].

Clinical Pearls

Kanavels signs of flexor tenosynovitis – (1) fusiform swollen digit; (2) pain on passive extension; (3) tenderness to percussion over sheath; (4) digit held in semi-flexed position – do not all have to be present and evolve with time.

Have very low threshold for exploration and surgical washout in diabetic and immunocompramised patients, even if Kanavels signs are equivocal.

Deep Space Infections

The deep spaces of the hand are the inter-digital web-spaces, thenar, mid-palmar and hypothenar spaces and the dorsal subcutaneous and sub-aponeurotic spaces. Infection may develop in each of these spaces usually as a result of penetrating trauma or contiguous spread from other areas, or occasionally from haematogenous spread [12]. They invariably require incision and drainage and are combined with appropriate antibiotic therapy.

Fig. 19.4 Second webspace abscess pointing dorsally and abducting the adjacent digits

Web Space Infections

Web space infections result in a subfascial "collar button abscess" usually as a result of a volar puncture to the web. The infection spreads from volar to dorsal through the space in the palmar fascia (just distal to the bifurcation of the neurovascular bundle), and presents with volar fullness and erythema with considerable dorsal webspace swelling [12] (Fig. 19.4). Patients may complain of a generalised pain in the palm, and still be able to flex and extend the fingers, but the affected web is held in abduction. Direct palpation or adduction of the web produces pain [35]. Separate incisions should be made through both the volar and dorsal web but not in the interdigital space which can cause contracture. A volar oblique, zigzag or chevron incision is recommended, whereas a longitudinal incision is acceptable on the dorsum [25, 35].

Thenar Space

Thenar space infections are the most common of the deep space infections. The boundaries of the thenar space are the adductor pollicus dorsally, the index finger flexor tendon volarly, the adductor insertion on the proximal phalanx radially, and the midpalmar septum ulnarly (which extends from the middle-finger metacarpal to the palmar fascia). The nomenclature is confusing as this space is separate from the compartment in which the thenar muscles are found within the thenar eminence. Infection in the thenar space is usually from index finger flexor tenosynovitis or a penetrating injury, and can spread between the adductor pollicis and first dorsal interosseous as a "pantaloon" abscess, or into the dorsal first webspace [36]. The thumb is held in palmar abduction as the thenar space is greatest in this position to accommodate the considerable thenar and first-web swelling seen with these infections. Pain is exacerbated by passive adduction or opposition.

We recommend a curved thenar crease incision (Fig. 19.5) which can be extended with zigzag incisions up to the index finger in cases of tenosynovitis. With a pantoloon abscess or dorsal involvement a longitudinal incision up to, but not across, the first webspace is performed.

Midpalmar (or Deep Palmar) Space

This space is bounded volarly by: the flexor tendons and lumbricals of the ulnar three digits; dorsally by the fascia overlying the middle, ring and little metacarpals and second and third palmar interossei; radially by the midpalmar septum; ulnarly by the hypothenar septum from the fifth metacarpal to the palmar aponeurosis. Infection results from direct penetration or spread from the middle and ring finger flexor sheaths. The normal concavity of the palm is flattened or even convex, however the tight volar fascia limits swelling. The looser dorsal connective tissues allow extensive dorsal swelling which may be so impressive that it often misleads clinical examinations. Pain is elicited on active and passive movements of the middle and ring fingers [25, 37]. For adequate exposure a large volar incision is required (Fig. 19.5), and may be combined with a longitudinal dorsal incision.

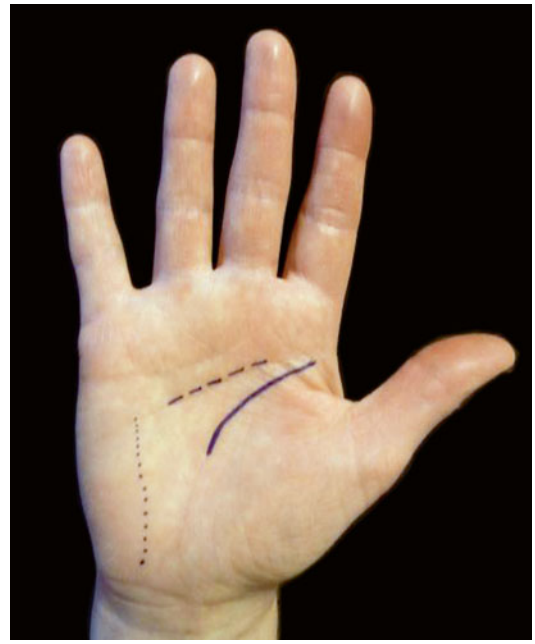


Fig. 19.5 Incision for deep space infection: Thenar crease incision – *solid line*; Proximal palmar crease incision for mid-palmar space – *dashed line*; Hypothenar incision – *dotted line*

Hypothenar Space

This is a potential space between the hypothenar septum and hypothenar muscles. The roof is formed by the palmar fascia and the floor by the periosteum over the fifth metacarpal. Infections of this space are extremely rare, and are drained by a longitudinal incision just ulnar to the hypothenar crease without extending beyond the distal wrist crease proximally or midpalmar crease distally (Fig. 19.5) [2, 37].

Dorsal Hand Spaces

The dorsal subaponeurotic space lies deep to the extensor tendons but above the periosteum of the metacarpals and fascia of the dorsal interosseous muscles. However the dorsal subcutaneous space, lying superficial to the extensor tendons, is an extensive area of loose connective tissue without distinct boundaries, in which pus can accumulate over the entire dorsum of the hand. Clinical differentiation between these infections in these two dorsal spaces, and that of other hand infections which manifest with dorsal swelling, is not

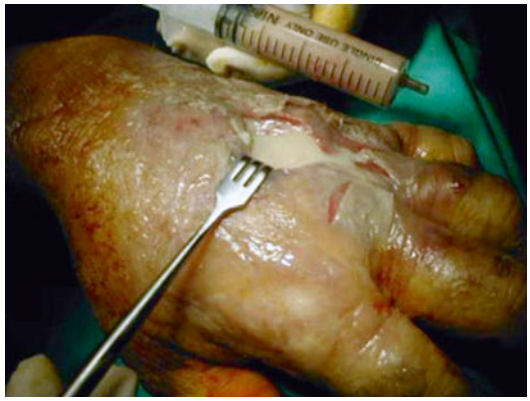


Fig. 19.6 Incision over fourth metacarpal to drain large dorsal hand abscess

Fig. 19.7 Septic arthritis of DIPJ with sinus formation and osteomyelitis

normally possible prior to exploration. Drainage incisions should be made in line with the second and/or the fourth metacarpals (Fig. 19.6) to allow exploration and decompression of all adjacent compartments, if necessary [37].

Clinical Pearls
Don't be reassured by a small skin defect, most deep space infections result from penetrating injuries

Septic Arthritis

Septic arthritis of the hand is most commonly from *S. Aureus* and Streptococcal infections as a result of direct penetration or contiguous spread (such as from a flexor sheath infection or periarticular osteomyelitis) [2]. However in non-traumatic cases haematogenous spread should be considered, in particular *Haemophilus influenzae* infections in children, and gonococcus in sexually active young adults with a monoarticular infection [25, 38]. Non-infective causes should also be considered, and where doubt exists, joint aspirates should be sent to exclude crystal arthropathies or other non-infectious aetiologies [12].

Septic arthritis results in destruction of joint cartilage through the release of bacterial toxins, proteolytic enzymes and free radicals [12]. Joints become erythematous and swollen. Infected joints are held in the position of "maximal joint space" to accommodate synovio-purulent expansion. Increasing intra-articular pressures decrease capsular blood flow and also directly damages articular cartilage [25]. This ultimately proceeds to osteomyelitis of the underlying bone, and possible sinus formation [38] (Fig. 19.7). Patients complain of pain exacerbated by movement. However the most useful clinical sign, which can help distinguish septic arthritis from soft tissue infections, is exquisite pain over the affected joint with axial loading [25].

Treatment is by expeditious incision, drainage and irrigation of the involved joint. Infected pannus and necrotic tissues should be excised. The wound is left open to allow continued drainage, and an irrigation catheter may be inserted for continued post-operative irrigation. We advocate a second washout in theatre at 24-48 h in all but the most mild cases of septic arthritis, and wounds only tacked closed if joints appear clear of infection. The hand is elevated and splinted, and intravenous antibiotics continued until local and systemic signs have resolved. Thereafter oral antibiotics, or less frequently community intravenous antibiotics, are given for 2–6 weeks [25] after discussion with a microbiologist. Early hand therapy should be instigated to prevent loss of joint motion.

The wrist joint is entered through a midaxial dorsal skin incision, followed by arthrotomy between the third and fourth compartment. A transverse dorsal wrist-crease incision is advocated by

some hand surgeons, as this leaves a discrete post-operative scar. Branches of the radial and dorsal ulnar nerve are at increased risk through this incision, and should be carefully avoided. A recent trend toward arthroscopic debridement of septic arthritis of the wrist has developed, with results reported superior to open techniques with respect to length of hospital stay and need for repeat surgeries [39, 40]. If acute carpal tunnel syndrome is present, volar release following carpal tunnel decompression may be considered. The metacarpophalangeal joints of the fingers are also entered through a dorsal midline incision over the joint. The joint is entered through the extensor expansion, or through the proximal part of the sagittal band [2, 12, 25], which is our preferred method. The interphalangeal joints and the metacarpal joint of the thumb are best approached through a midlateral incision, entering the joint between the volar plate and the accessory collateral ligament. This reduces adhesions and prevents damage to the extensor mechanism and subsequent deformities, such as boutonnière or mallet finger [12, 25, 38]. The DIPJ may alternatively be accessed through a dorsal H-flap incision (Fig. 19.8).

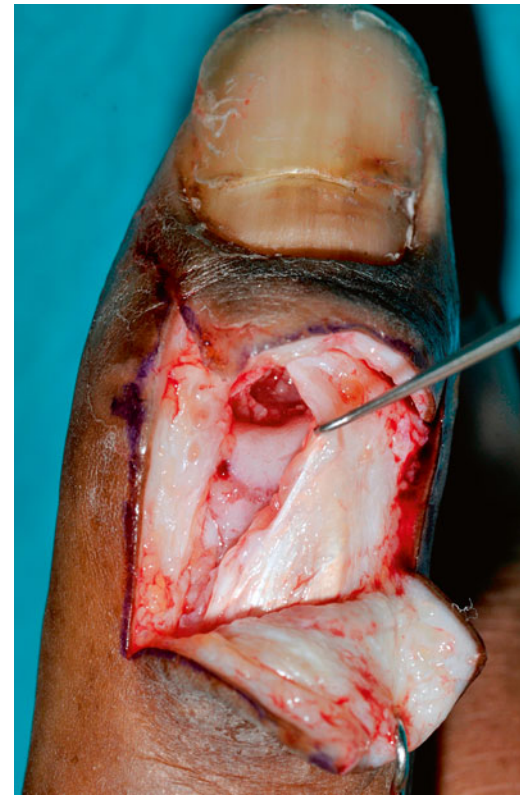


Fig. 19.8 DIPJ septic arthritis accessed through dorsal H-flap and retraction of extensor tendon to access and wash out the joint

Clinical Pearls

Septic arthritis is a surgical emergency. Delays to surgical washout are correlated with worse outcomes.

Osteomyelitis

Osteomyelitis of the hand most commonly arises from trauma, particularly when an open fracture communicates with a dirty wound. Osteomyelitis can also arise from contiguous spread from an adjacent abscess or septic arthritis. In rare cases osteomyelitis results from haematogenous spread in adults, but this route is more common in children [2, 25]. Iatrogenic osteomyelitis may complicate both clean (e.g. a closed fracture) and elective hand surgery, as well as dirty traumatic hand procedures. Operations using external k-wires are particularly implicted. Despite this there is little evidence that prophylactic or peri-operative antibiotics affect surgical site infection rates in either elective or emergency hand surgery, with the exception of operations lasting longer than 2 h [41–44].

The most common infecting organisms are *S. Aureus*, *S. Epidermidis* and streptococcus species. *Eikenella corrodens* and *Pasteurella multocida* are frequently isolated from human and animal bites. There is a higher incidence of Gram negative, anaerobic, and polymicrobial infections in immunosuppressed patients, diabetic patients, heavily contaminated wounds and those with mutilating injuries [43, 45].

Osteomyelitis can be acute or chronic. Acute osteomyelitis of the hand may occasionally resolve with antibiotic therapy alone [25], particularly from haematogenous spread [43], however most authors advise prompt surgical debridement of devitalised and infected bone [2, 46].

Fig. 19.9 Osteomyelitis of fourth metacarpal head following delayed presentation of a fight bite injury

Osteomyelitis causes localised pain, erythema and swelling, progressing onto sequestration of bone and sinus formation. Systemic symptoms are rare, and when present a haematogenous aetiology should be considered [25]. Plain x-rays may show osteolysis (Fig. 19.9), osteopenia, osteosclerosis, periosteal reaction, and sequestrum/involucrum [45], however nuclear medicine can identify changes before plain radiographs. Magnetic resonance imaging is the most sensitive and most specific imaging modality for the detection of infection in bone, and gives accurate information on the extent of the infectious process in bone and soft tissue [46].

Cierny classifies osteomyelitis into four stages which help to determine treatment options: (1) intramedullary; (2) superficial; (3) Localised; (4) Diffuse [46, 47].

Intramedullary infection is most commonly from intramedullary nailing, which in the hand equates to intramedullary k-wires, however haematogenous spread should also be considered. Treatment involves debridement of intramedullary contents by reaming the intramedullary canal (more appropriate in lower limb osteomyelitis) [46] or cortical windowing and curettage (the preferred method of most hand surgeons) [25, 38].

Superficial osteomyelitis requires decortication of the involved bone. An adjacent abscess or soft tissue infection should be drained and debrided, and if direct closure is not possible due to tissue loss, coverage should be provided by local or free flaps [46, 47].

Localised osteomyelitis occurs where infection penetrates the cortex with extension into the medullary canal, but the bone is axially stable. Saucerisation of the infected cortex and medulla is required, followed by appropriate soft tissue coverage. The need for bone grafting and orthopaedic stabilisation is determined by the location and extent of the resection [46, 47].

Infection extends extensively throughout the bone in diffuse osteomyelitis, resulting in axial instability. The techniques used in managing Stage IV osteomyelitis are conceived and executed with a stabilization procedure in mind. External or internal fixations are planned in combination with bone grafting or free bone flap, and soft tissue coverage [46, 47].

The treatment of osteomyelitis usually requires more than one surgical intervention, and prolonged immobilisation [45, 47]. Terminalisation or amputation of the affected digit, may provide the best solution in order to avoid disability to the unaffected parts of the hand [2] with amputation rates of 39 % in one study [45].

Antibiotic treatment should be guided by cultures from intraoperative bone biopsies. Intravenous antibiotics should be continued for at least 2 weeks after surgery, but are frequently required for 4–6 weeks based on the severity of the infection and virulence of the organism [42]. In less severe cases oral antibiotics are usually continued for 4 weeks in cases of acute osteomyelitis and for 4–6 weeks in cases of chronic osteomyelitis [38]. Normalisation of a previously elevated ESR and CRP are good indicators for completion of the antibiotic course [25, 42].

Bite Injures

Human Bites

Human bite injuries are either of the closed-fist "fight-bite" variety (typically with impaction of the knuckles onto the upper front incisors) or an occlusive injury from being intentionally bitten. Fight bites are the more frequent type of injury and far more common in young males under the influence of alcohol [48, 49], whereas women present with a greater proportion of occlusive

Fig. 19.10 Fight bite injury over fourth metacarpal head, with underlying extensor tendon laceration, MCPJ septic arthritis and dorsal hand abscess formation

injuries [50]. Fight bite injuries have a worse prognosis than occlusive injuries, as there is increased risk of deep seated infection and damage to underlying structures.

Skin lacerations from fight bite injuries are typically 3–5 mm in length, most commonly overlying the metacarpal heads (Fig. 19.10). These seemingly trivial skin wounds often result in under-estimation of the extent and severity of these injuries by both the patients, resulting in late presentation, and health care professionals [18]. The injury is sustained with the fingers fully flexed into a fist, whereas examination of the hand is routinely performed with fingers extended on a hand table. In this position extensor tendon and joint capsule injures remain undiagnosed as they retract proximally relative to the skin lacerations. Severe cellulitis, tissue necrosis, tendon laceration and septic arthritis are likely consequences of inadequate exploration, washout or debridement. Delayed treatment of septic arthritis predisposes to osteomyelitis, flexor tenosynovitis, secondary arthritis, stiffness and even amputation [51].

All fight bite injuries should have imaging to exclude a fracture or retained fragment of tooth. We then advocate surgical exploration with appropriate anaesthesia (block or GA), under tourniquet control and with wide exposure of the wound to allow assessment of the joint and the full excursion of the extensor tendon over the MCPJ.

A randomised control trial of uncomplicated human bite injuries (with no tendon or joint involvement) showed that debridement alone, in the absence of antibiotics, resulted in an increased infection rate. However there was no difference when either oral or intravenous antibiotics were combined with debridement [52]. Infected wounds require admission, debridement, elevation and intravenous antibiotics. Fractures and tendons should only be repaired once infection has been cleared. Wounds should be left open to heal in by secondary intention.

The potential for infective complications are well documented with a risk of local infection around 20 %. This infection rate is higher than most comparable wounds due to the considerable bacterial load of saliva, documented at over 900×10 [1] organisms/ml [53]. Over 40 different bacterial species have been cultured from human bites [54]. *Eikenella corrodens* is a gram-negative anaerobic bacillus commonly associated with human bites and contributes to 7–29 % of infections [25, 54]. It is resistant to clindamycin, erythromycin, the aminoglycosides, metronidazole and is variably susceptible to cephalosporins. Some are resistant to the tetracyclines, but most are susceptible to beta-lactam antibiotics, particularly the penicillins [55]. Group A Streptococcus and *S. aureus*, still remain the most commonly isolated organisms, with Bacteroides species the most commonly isolated anaerobes, usually associated with mixed cultures [54]. Hepatitis B [56], hepatitis C [57] and HIV [58] have all been recorded from human bite injuries. Vaccination

or prophylactic anti-viral treatment should be provided according to local protocol and the individual case, until microbiological results become available, along with tetanus vaccination [59].

Animal Bites

Roughly 1 % of all accident and emergency department visits are from animal bites, 80 % of which are dog bites [60]. Cats bites are the second most frequent animal bite with 30–50 % of cat bites becoming infected, more than twice as frequently as dog bites [61]. This is attributed to cat teeth being more sharp and slender which penetrate deeply, often through small puncture wounds, extending down to bone which may result in septic arthritis and osteomyelitis. The risk of being bitten varies with age, with children at greater risk due to threatening behaviour (often unintentional), or the deliberate teasing of animals. The upper limb, and particularly the hands, are the most common site of animal bite injuries, however the risk of head and neck and facial bites increases in younger children due to their shorter stature [62].

Although Pasteurella species are the most common isolates, dog and cat bites contain an average of 5 different aerobic and anaerobic bacteria per wound, including staphylococcus, streptococcus and bacteroides. Clinically, infection with *Pastuerella multocida* is characterized by a rapidly developing intense inflammatory response, with prominent pain and swelling developing within 24 h of the initial injury in 70 % of cases and by 48 h in 90 % [25, 60]. The decision to administer oral or parenteral antibiotics depends on the depth and severity of the wound and on the time since the bite occurred. It is recommended that all but the most minor bite wounds be surgically debrided and explored in theatre to exclude damage to underlying structures. Damaged structures should be repaired in a delayed fashion after a secondary inspection of the wound in theatre at 24–48 h, and skin wounds are left open to heal in secondarily.

Patients not allergic to penicillin should be treated with amoxicillin-clavulanate to provide adequate cover for *Pasteurella multocida* infections. Patients who are penicillin allergic can receive oral or intravenous doxycycline, trimethoprim-sulfamethoxazole, or a fluoroquinolone plus clindamycin [60]. Cat scratches typically inoculate the same organisms as cat bites and should be treated similarly. Rabies is uncommon in most western countries, but should be considered in bites from feral, wild or abnormally behaving animals, and rabies prophylaxis provided [62].

Necrotising Fasciitis

Necrotising fasciitis is a surgical emergency that is potentially limb or life threatening without prompt surgical intervention. Necrotising fasciitis is usually caused by toxin-producing, virulent bacteria, and is characterized by widespread fascial necrosis with relative sparing of skin and underlying muscle [63, 64].

The disease occurs more frequently in diabetics, alcoholics, immunosuppressed patients, IV drug users and patients with obesity and peripheral vascular disease. It can also afflict young, previously healthy individuals, particularly those suffering from chickenpox, or after surgery. It can occur in any region of the body, however the extremities, along with the abdominal wall and perineum, are the most common sites of infection [63–65]. In the upper limb trauma is the primary cause of infection, often after a relatively minor injury such as an insect bite. Haematogenous spread is less common [65, 66].

Necrotising fasciitis may initially present like a cellulitis, however it is then characterised by a rapidly advancing less distinct leading edge, with the skin beyond it appearing shiny and having pronounced non-pitting oedema. In the early stages pain out of proportion with the visible extent of infection, particularly on palpation outside the zone of cellulitis, should heighten concern for possible necrotising fasciitis [63, 64]. Soft tissue crepitus occurs almost exclusively from anaerobic infections. In later stages the skin becomes anaesthetic, with patchy or confluent areas of soft tissues necrosis, evolving from the point of initial infection, as the circulation to the skin is impaired (Fig. 19.11). The skin takes on a greyish hue, with patches of violet echymosis and bullae [12, 63, 64]. Systemic effects are more pro-

Fig. 19.11 Necrotising fasciitis of left arm following thorn injury to middle finger 48 h earlier, with eventual amputation of arm at mid-humeral level

nounced, and hemodynamic instability in the face of otherwise trivial appearing cellulitis should increase suspicion of necrotising fasciitis [63].

Polymicrobial necrotizing fasciitis is usually caused by enteric pathogens, whereas monomicrobial necrotizing fasciitis is usually due to skin flora [63]. Giuliano et al. divided necrotising fasciitis into two groups based on bacteriology results. Type 1 is poly-microbial and involves non-group A streptococci and anaerobes. Type 2 is group A beta-haemolytic streptococci alone or in combination with a staphylococcus [67], and is the more common type in the extremities [63]. However many different organisms have been recorded causing necrotising fasciitis, including fungi, viruses, and atypical mycobacteria [64, 68]. Tissue damage and systemic toxicity are believed to result from the release of endogenous cytokines and bacterial toxins. Muscle is usually spared, except with infections with clostridial species which causes myonecrosis, and "gas-gangrene" [60, 64].

Plain radiographs demonstrating free air in the tissues, and MRI scans can help with making a diagnosis of necrotising fasciitis, however these should only be ordered in haemodynamically stable patients with equivocal signs [64, 69]. The LRINEC (Laboratory Risk Indicator for Necrotizing Fasciitis) Score (see Table 19.1), derived from standard hospital blood tests, is described as a clinical tool for distinguishing early necrotising fasciitis from other soft tissue infections [70]. Certainly prompt treatment has been shown to reduce mortality [71].

The "finger test" can be performed at the bedside under local anaesthetic or in theatre. A lack of resistance of normally adherent fascia to blunt dissection is the classic finding, and may also include "dishwater pus" that is thin and foul smelling, with minimal bleeding due to subcutaneous vessel thrombosis. Frozen section sampling, combined with an urgent gram stain, provides the definitive diagnosis [12, 63, 64].

Treatment modalities include surgery, antibiotics, supportive care [63] and hyperbaric oxygen [72] (if available). Initial antibiotic therapy

Table 19.1

Test, units	Score
C-Reactive Protein, mg/L	
<150	0
≥150	4
Total white cell count, per mm^3	
<15	0
15–25	1
>25	2
Haemoglobin, g/dL	
>13.5	0
11–13.5	1
<11	2
Sodium, mmol/L	
≥135	0
<135	2
Creatinine, μmol/L	
≤141	0
>141	2
Glucose, mmol/L	
≤10	0
>10	1

The maximum score is 13; a score of ≥6 should raise the suspicion of necrotizing fasciitis and a score of ≥8 is strongly predictive of this disease [70]

should include broad aerobic and anaerobic coverage. Clindamycin is recommended due to its ability to suppress toxin production, and modulate cytokine production from toxin-producing strains of Gram-positive and Gram-negative organisms [60, 73, 74]. Fluid resuscitation should be commenced, but surgery should not be delayed as haemostability may only be achievable through excision of necrotic tissues. This may necessitate amputation of the limb in severe cases. Early and adequate surgical debridement and fasciotomy have been associated with improved survival [63].

Mortality rates are as high as 76 %. Delays in diagnosis and/or treatment correlate with poor outcome, with the cause of death being overwhelming sepsis syndrome and/or multiple organ system failure [63, 71]. A recommendation to use intravenous γ–globulin to treat streptococcal toxic shock syndrome cannot be made with certainty at present [60].

Atypical Infections

Introduction

Atypical infecting organisms of the hand are becoming more common in Western societies [75]. This is attributed to increasing migration to and from endemic areas, an aging population and increasing numbers of immunocompromised patients, including diabetics, transplant patients and patients affected with the immunodeficiency virus (HIV) [75, 76]. However it should be noted that in HIV and immune-suppressed patients staphylococcus aureus is still the most commonly encountered organism. Even so there is a tendency for the development of "atypical" manifestations of infections with "typical" organisms in such patients [77].

Atypical infecting agents include mycobacteria, viral, and fungal organisms. The standard antibiotic and surgical protocols for treating typical bacterial hand infections are often not succesful in eliminating those caused by atypical organisms [75].

Mycobacterial Infections

Mycobacterium Tuberculosis (TB)

Musculoskeletal tuberculosis is a rare form of extrapulmonary disease and occurs in between 1 and 3 % of cases [78, 79], of which around 10 % is tuberculosis of the hand [80].

Constitutional symptoms of tuberculosis are usually present, including low-grade fever, night sweats, weight loss and anorexia. TB infections of the hand have 4 general presentations: cutaneous, synovial infection, osteomyelitis or septic arthritis [75]. Specific symptoms of tuberculosis of the hand include swelling, stiffness, pain, carpal tunnel syndrome and chronic discharging sinuses [76]. Chest x-rays are usually clear in cases of hand tuberculosis, and diagnosis is frequently delayed resulting in a worse outcome [81].

An elevated ESR, a strongly positive Mantoux test (if patients have not had a Bacille Calmette-Guerin vaccination), and typical radiological

features indicate the presence of tuberculosis [76]. At operation fresh tissue should be sent for culture and Ziehl-Neelsen staining. Diagnosis from a culture usually takes from 3–6 weeks and, on suspicion, medical treatment should be started before the results have returned. Granulomatous lesions on open biopsy with the typical "rice bodies" are considered diagnostic [82].

Antibiotic treatment consists of 2 months of combination drug therapy (three or four different drugs), including isoniazid, rifampin, and pyrazinamide with or without ethambutol. Isoniazid and rifampin therapy is then continued for another 4 months. It is imperative that a full course of treatment is completed because partial treatment can lead to the development of resistant strains [75, 83].

Cutaneous Tuberculosis

Cutaneous TB of the hand is rare and seen mainly in healthcare workers exposed to TB patients [83]. Cutaneous TB has three stages. The nodular stage lasts 1–2 weeks, followed by the "tuberculous chancre" ulcerative stage, and finally the enlargement of regional lymph nodes after 3–4 weeks. Diagnosis is from cultures and histology demonstrating non-caeseating granumlomas. Mycobacterium marinum, sporotrichosis and orf all have similar presentations, and should be excluded [76].

Tuberculous Tenosynovitis

Synovial infection is the most common TB infection of the hand, and usually presents as extensor or flexor tenosynovitis. The flexors are involved more frequently than the extensors and those of the ulnar border more than the radial border. Flexor tendon involvement most commonly occurs in the palm and within the carpal tunnel. The synovitis can be dense and proliferative, resembling rheumatoid synovium (Fig. 19.12), and may result in tendon rupture [75, 76]. Despite its slow onset the most common presentation is from tenosynovial invasion of the carpal tunnel resulting in a "compound palmar ganglion" with painless swellings proximal and distal to the carpal tunnel. This will eventually lead to carpal tunnel syndrome. Isolated digital tuberculous tenosynovial infections are occasionally seen

Fig. 19.12 TB wrist with multiple tendon ruptures. Extensive thickened tenosynovium and rice bodies removed with ronger

[76]. A complete synovectomy is recommended, followed by long-term chemotherapy.

Tuberculous Osteomyelitis

Skeletal involvement in extrapulmonary TB accounts for 1–3 % of cases, with the metacarpals and phalanges rarely being involved [79, 82]. However in children bone and joint TB occurs in 1–5 % of those with untreated pulmonary TB. In fact 85 % of patients with TB dactylitis are younger than 6 years of age, with skeletal infections usually becomes symptomatic 1–3 years after the initial pulmonary infection [84].

The clinical presentations of TB osteomyelitis in the hand are: unifocal – a single lesion in a metacarpal or phalanx; multifocal – usually only seen in adults; and disseminated – seen in immunocompramised patients, with bony lesions throughout the body [76]. Radiographic appearances are of sclerotic and osteolytic lesions of a variable

Fig. 19.13 TB wrist, with osteopaenia of metacarpal bases and the carpus

nature (Fig. 19.13). The classic "spina ventosa" (Latin: *spina* = short bone, *ventosa* = filled with air) pattern is that of a massive spindle shaped metacarpal or phalanx [80]. Chronic discharging sinuses occur late on, with secondary bacterial infection resulting in sequestrum formation. Definitive diagnosis is made by biopsy, however there is no indication for extensive debridement as good medical treatment is sufficient, and the prognosis is usually good [76].

Tuberculous Arthritis

Tuberculous arthritis of the upper limb is rare. The pathophysiology is different in children, with metaphyseal osteomyelitis the initial pathology, which then crosses the growth plate into the joint [85], whereas in adults seeding of the synovium usually occurs first [76]. Clinical presentation is as mono-or polyarthritis of the metacarpal or interphalageal joints, or arthritis of the wrist. Monoarthritis usually takes a prolonged course, however involvement of the wrist or polyarthritis of digits usually progresses rapidly [76]. Sinus formation occurs late, and bony destruction results in joint subluxations, dislocations and finally ankylosis [80]. Diagnosis is confirmed through biopsy and cultures.

Conservative management (chemotherapy and immobilisation, followed by rehabilitation) usually gives good functional results. Outcomes depend on the clinical stage at the time that medical treatment is started. Ankylosis is expected from advanced stages, and functional splinting is reported to achieve better results than arthrodesis or excision of the joint. Corrective osteotomies or wrist arthrodesis may be required later once the infection has resolved [76, 80].

Finally clinicians should be aware of Poncet's disease, which is an aseptic polyarthritis without joint destruction, in which synovial biopsies are negative, in a patient with known active tuberculosis. This is a hypersensitive immune response to tuberculo-protein, often confused with connective tissue diseases, but shows a rapid therapeutic response after initiating anti-tuberculous drug therapy [76, 86].

Non-tuberculous Mycobacterial Infections

Non-tuberculous mycobacteria (NTM) can cause rare, but relatively resistant, hand infections [25]. NTM lead to similar manifestations in the hand as tuberculosis, including tenosynovitis, osteomyelitis, and septic arthritis [75] and should be managed similarly.

NTM are ubiquitous organisms present in the environment (soil, water) and in animal reservoirs, and a large number of different organisms have been recorded causing hand infections. *M. marinum* is the most common NTM to cause hand infections, and in immunocompetent humans is restricted principally to the extremities, with

few systemic infections [75, 87]. *M. marinum* lesions have three clinical presentations: verrucal skin lesions, subcutaneous granulomas (with or without ulceration), and deep-tissue infections. Tenosynovitis of the extensor or flexor tendons is the most common deep infection [88, 89]. Verrucal skin lesions are usually self-limiting, whereas subcutaneous granulomas and deep-tissue infections are treated by surgical excision/debridement, followed by antibiotic therapy [75].

M. avium causes the greatest number of human NTM infections overall, but is the second most common NTM infection of the upper-extremity. Prior to the AIDS epidemic *M. avium* infections were primarily pulmonary infections, however in AIDS patients the infection is usually disseminated, including to the hand.

Chemotherapy of NTM infections has been problematic because of the relative resistance of nontuberculous mycobacteria to a wide range of antibiotics. Following biopsy antibiotic therapy must be guided by culture and sensitivity results [75, 87].

Leprosy (*Mycobacterium leprae*)

Leprosy, commonly known as Hansen's disease, is caused by *M. leprae*, and is rarely seen in Western societies. Spread by person-to-person transmission, *M. leprae* grows better at lower temperatures and thus has a predilection for the skin and peripheral nerves, particularly of the upper extremity. The infection initially infects schwann cells, subepidermal cells, and perivascular skin macrophages. Initial nerve involvement of small sensory nerves produces patchy areas of sensory loss. Involvement of larger nerves will cause mixed motor and sensory loss, that is more severe and debilitating. The ulnar nerve is the most commonly involved upper extremity nerve, followed by the median and radial nerves. Ulnar nerve dysfunction leads to clawing proximal interphalangeal contractures and intrinsic thumb weakness. Mutilation of the hands can occur when they become completely insensate. Treatment consists of splintage and release of contractures. Treatment of the infection is medical [75, 90].

Fungal Infections

Fungal infections are either opportunistic or non-opportunistic. In the hand fungal infections are relatively uncommon, but are now being reported with increasing frequency, especially opportunistic infections in immunocompromised patients [91].

Fungal infections often present with an indolent onset resulting in a delay in diagnosis and treatment. Definitive diagnosis can only be made following biopsy and special fungal stains and cultures [92]. There are two types of fungi: yeasts and moulds, and both can cause infections in humans. Yeasts such as *Candida albicans* grow as single cells and reproduce by asexual budding. In contrast, moulds grow in long filaments called hyphae [93].

Fungal infections can broadly be subdivided into three main groups according to their anatomic depth of invasion: cutaneous, subcutaneous and systemic [93].

Cutaneous Fungal Infections

Cutaneous infections are caused by organisms that metabolise keratin and therefore cause infections of the skin, paronychia and nail, and are called tinea infections (Latin: tinea = gnawing worm).

Cutaneous dermatophytosis of the hand is called tinea manuum [25], or more commonly by the generic term "ringworm" due to the characteristic enlarging raised red rings. They generally occur in the glabrous skin of the palm and the intertrigenous skin, and pruritic scaling lesions with vesicle formation occurs. These are some of the most common hand infections, and are caused by dermatophytes (usually Trichophyton, Microsporum or Epidermophyton species) or *Candida albicans*. Dermatophytes are keratinophilic saprophytes that colonize the dead stratum corneum with histology demonstrating spores and septated hyphae. *Exophiliala werneckii* causes tinea negra, an asymptomatic browny-black lesion due to its pigmented hyphae, and can be confused with malignant melanoma. Infection by Candida species is suggested by the absence of hyphae. Treatment of cutaneous dermatophytosis is with topical imidazoles, such as ketoconazole, or tolnaftate for 2–3 weeks and is usually curative [75].

Onychomycosis (Greek: onycho = claw, mycosis = fungal infection), otherwise known as tinea unguium, accounts for around a third of fungal skin infections, with toenails affected much more often than fingernails. The most common cause of subungual onychomycosis is the dermatophyte *Trichophyton rubrum*. However onychomycosis is responsible for only about one half of nail dystrophies, and therefore a biopsy and cultures are required to exclude psoriasis, lichen planus, contact dermatitis, trauma or a nail bed tumour [94]. Onychomycosis produces a white lesion that migrates proximally under the nail, separating the nail from the bed, resulting in a brittle, cracked nail. Bacterial super-infection may cause discolouration of the nail, whereas chronic or recurrent paronychias are most likely caused by Candida species [95]. People working in moist environments, or whose hands are often in water, are at particular risk of Candidal nail infections [25, 75].

Treatment of onchomycosis requires oral antifungal treatment, such as terbinafine or itraconazole, as topical treatment alone is usually insufficient to eradicate infection [94]. Chronic paronychia due to Candida infections are commonly treated with marsupialization [25].

Subcutaneous Fungal Infections

Subcutaneous infections are typically caused by organisms of low virulence, such as Sporotrichosis [25]. Sporotrichosis, caused by *Sporothrix schenckii*, is the most common subcutaneous fungal infection, and occurs predominantly in the upper extremities [95]. Subcutaneous implantation of Sporothrix spores is usually from handling a variety of plants, such as rose thorns and cactii [25]. The spores produce a chronic granulomatous infection which develops into an ulcer at the site of inoculation. An ascending lymphadenopathy occurs with epitrochlear lymph nodes frequently involved. The lymph nodes can eventually ulcerate and drain seropurulent fluid. Sporotrichosis is difficult to diagnose with standard stains, and cultures are required to diagnose the organism. Recommended treatment consists of topical saturated potassium iodide solution or oral itraconazole therapy [25, 75].

Deep or Systemic Fungal Infections

Deep or systemic infections are usually caused by organisms that have two stages to their life cycle. They enter the body as spores, but then multiply in a different form causing infections of tendons, bones and joints [25]. They are rarely primary hand infections, but are disseminated infections from a pulmonary nidus [75]. Deep infections of the hand are nearly all opportunistic infections in immunocompramised individuals. Infections are those that always require an immunocompromised host (deep candidiasis, invasive aspergillosis, mucormycosis and cryptococcus infection) or those that usually required an immunocompromised host (extra-cutaneous sporotrichosis, disseminated coccidioidomycosis and disseminated histoplasmosis) [91]. Ulceration and sinus formation may communicate with deep tendons (flexor tenosynovitis) and synovial tissues (causing carpal tunnel syndrome) or may even cause osteomyelitis or septic arthritis. Early aggressive surgical treatment includes debridement of all involved soft tissues and bone, followed by adjuvant antifungal therapy [91, 92].

Clinical Pearls

In immunocompramised patients with hand infections request microbiological tests for mycobacteria and fungi

If mycobacteria or fungi are isolated unexpectedly, discuss with the patient the need for an HIV test.

Summary

Injuries to the hand and upper limb are a common presentation to accident and emergency and other acute service departments. The hand's reaction to infection is determined by anatomic considerations. It is therefore important that even seemingly insignificant injuries are assessed by individuals with a strong understanding of hand anatomy. Surgical exploration, washout and debridement using loupe magnification and tourniquet control greatly reduces the chance of missing

damage to underlying structures and reduces the probability of infection. Targeted antibiotics, hand elevation and early hand therapy should also help to minimise poor outcomes. Unfortunately hand infections frequently present late due to the trivial nature of many hand injuries or to the circumstances in which the injury was sustained (e.g. fight bite injuries), reducing the potential for a full functional recovery. Also as a consequence of increasing numbers of elderly and immunocompramised patients, once "atypical" infections are becoming less atypical, and should always be considered when ordering microbiological investigations. However *Staphlycoccus aureus* remains the most common infecting organism, and should be covered for in nearly all hand infections, until full antibiotic sensitivities have returned.

References

1. Louis DS, Jebson PJ. Mimickers of hand infections. Hand Clin. 1998;14(4):519–29, vii.
2. Brown DM, Young VL. Hand infections. South Med J. 1993;86(1):56–66.
3. Hausman MR, Lisser SP. Hand infections. Orthop Clin North Am. 1992;23(1):171–85.
4. Boles SD, Schmidt CC. Pyogenic flexor tenosynovitis. Hand Clin. 1998;14(4):567–78.
5. Connolly B, Johnstone F, Gerlinger T, Puttler E. Methicillin-resistant Staphylococcus aureus in a finger felon. J Hand Surg Am. 2000;25(1):173–5.
6. Karanas YL, Bogdan MA, Chang J. Community acquired methicillin-resistant Staphylococcus aureus hand infections: case reports and clinical implications. J Hand Surg Am. 2000;25(4):760–3.
7. Hochman LG. Paronychia: more than just an abscess. Int J Dermatol. 1995;34(6):385–6.
8. Canales FL, Newmeyer 3rd WL, Kilgore Jr ES. The treatment of felons and paronychias. Hand Clin. 1989;5(4):515–23.
9. Brook I. Aerobic and anaerobic microbiology of paronychia. Ann Emerg Med. 1990;19(9):994–6.
10. Rockwell PG. Acute and chronic paronychia. Am Fam Physician. 2001;63(6):1113–6.
11. Jebson PJ. Infections of the fingertip. Paronychias and felons. Hand Clin. 1998;14(4):547–55, viii.
12. McDonald LS, Bavaro MF, Hofmeister EP, Kroonen LT. Hand infections. J Hand Surg Am. 2011;36(8):1403–12.
13. Wu IB, Schwartz RA. Herpetic whitlow. Cutis. 2007; 79:193–6.
14. White WB, Grant-Kels JM. Transmission of herpes simplex virus type 1 infection in rugby players. JAMA. 1984;252(4):533–5.
15. Selling B, Kibrick S. An outbreak of herpes simplex among wrestlers (herpes gladiatorum). N Engl J Med. 1964;270:979–82.
16. Belongia EA, Goodman JL, Holland EJ, et al. An outbreak of herpes gladiatorum at a high-school wrestling camp. N Engl J Med. 1991;325(13):906–10.
17. Fatahzadeh MSRA. Human herpes simplex virus infections: epidemiology, pathogenesis, symptomatology, diagnosis and management. J Am Acad Dermatol. 2007;57(5):737–63.
18. Clark DC. Common acute hand infections. Am Fam Physician. 2003;68(11):2167–76.
19. Gill MJ, Arlette J, Buchan K, Tyrrell DL. Therapy for recurrent herpetic whitlow. Ann Intern Med. 1986; 105:631.
20. Stern PJ. Selected acute infections. Instr Course Lect. 1990;39:539–46.
21. Watson PA, Jebson PJ. The natural history of the neglected felon. Iowa Orthop J. 1996;16:164–6.
22. Moran GJ, Talan DA. Hand infections. Emerg Med Clin North Am. 1993;11(3):601–19.
23. Harrison BP, Hilliard MW. Emergency department evaluation and treatment of hand injuries. Emerg Med Clin North Am. 1999;17(4):793–822, v.
24. Reid W, Draeger RW, Bynum Jr DK. Flexor tendon sheath infections of the hand. J Am Acad Orthop Surg. 2012;20(6):373–82.
25. Abrams RA, Botte MJ. Hand infections: treatment recommendations for specific types. J Am Acad Orthop Surg. 1996;4(4):219–30.
26. Kanavel AB, editor. Infections of the hand. 7th ed. Philadelphia: Lea & Febiger; 1939.
27. Pang HN, Teoh LC, Yam AK, Lee JY, Puhaindran ME, Tan AB. Factors affecting the prognosis of pyogenic flexor tenosynovitis. J Bone Joint Surg Am. 2007;89(8):1742–8.
28. Neviaser RJ, Gunther SF. Tenosynovial infections in the hand: diagnosis and management. Instr Course Lect. 1980;29:108–28.
29. Gutowski KA, Ochoa O, Adams Jr WP. Closed-catheter irrigation is as effective as open drainage for treatment of pyogenic flexor tenosynovitis. Ann Plast Surg. 2002;49(4):350–4.
30. Lille S, Hayakawa T, Neumeister MW, Brown RE, Zook EG, Murray K. Continuous postoperative catheter irrigation is not necessary for the treatment of suppurative flexor tenosynovitis. J Hand Surg Br. 2000;25(3):304–7.
31. Henry M. Septic flexor tenosynovitis. J Hand Surg Am. 2011;36(2):322–3.
32. Wain RAJ, Shah HA, Laitung JKG. Continous flexor sheath irrigation: a cautionary tale. J Hand Surg Eur Vol. 2012;37(E)(9):904–5.
33. Pollen AG. Acute infection of the tendon sheaths. Hand. 1974;6(1):21–5.
34. Harris PA, Nanchahal J. Closed continuous irrigation in the treatment of hand infections. J Hand Surg Br. 1999;24(3):328–33.
35. Plancher KD, editor. Masterclasses: hand and wrist surgery. Thieme, 2004.

36. Burkhalter WE. Deep space infections. Hand Clin. 1989;5(4):553–9.
37. Wolfe SW, editor. Green's operative hand surgery. 6th ed. Churchill Livingstone, 2010.
38. Freeland AE, Senter BS. Septic arthritis and osteomyelitis. Hand Clin. 1989;5(4):533–52.
39. Sammer DM, Shin AY. Comparison of arthroscopic and open treatment of septic arthritis of the wrist. J Bone Joint Surg Am. 2009;91(6):1387–93.
40. Sammer DM, Shin AY. Arthroscopic management of septic arthritis of the wrist. Hand Clin. 2011;27(3):331–4.
41. Aydin N, Uraloglu M, Yilmaz Burhanoglu AD, Sensoz O. A prospective trial on the use of antibiotics in hand surgery. Plast Reconstr Surg. 2010;126(5):1617–23.
42. Gaston RG, Kuremsky MA. Postoperative infections: prevention and management. Hand Clin. 2010;26(2):265–80.
43. Rao N, Ziran BH, Lipsky BA. Treating osteomyelitis: antibiotics and surgery. Plast Reconstr Surg. 2011;127 Suppl 1:177S–87.
44. Kleinert JM, Hoffmann J, Miller Crain G, Larsen CF, Goldsmith LJ, Firrell JC. Postoperative infection in a double-occupancy operating room. A prospective study of two thousand four hundred and fifty-eight procedures on the extremities. J Bone Joint Surg Am. 1997;79(4):503–13.
45. Reilly KE, Linz JC, Stern PJ, Giza E, Wyrick JD. Osteomyelitis of the tubular bones of the hand. J Hand Surg Am. 1997;22(4):644–9.
46. Cierny 3rd G. Surgical treatment of osteomyelitis. Plast Reconstr Surg. 2011;127 Suppl 1:190S–204.
47. Cierny 3rd G, Mader JT, Penninck JJ. A clinical staging system for adult osteomyelitis. Clin Orthop Relat Res. 2003;414:7–24.
48. Marr JS, Beck AM, Lugo Jr JA. An epidemiologic study of the human bite. Public Health Rep. 1979;94(6):514–21.
49. Perron AD, Miller MD, Brady WJ. Orthopedic pitfalls in the ED: fight bite. Am J Emerg Med. 2002;20(2):114–7.
50. Wallace CG, Robertson CE. Prospective audit of 106 consecutive human bite injuries: the importance of history taking. Emerg Med J. 2005;22(12):883–4.
51. Mennen U, Howells CJ. Human fight-bite injuries of the hand. A study of 100 cases within 18 months. J Hand Surg Br. 1991;16(4):431–5.
52. Zubowicz VN, Gravier M. Management of early human bites of the hand: a prospective randomized study. Plast Reconstr Surg. 1991;88(1):111–4.
53. Henry FP, Purcell EM, Eadie PA. The human bite injury: a clinical audit and discussion regarding the management of this alcohol fuelled phenomenon. Emerg Med J. 2007;24(7):455–8.
54. Goldstein EJ, Citron DM, Wield B, Blachman U, Sutter VL, Miller TA, et al. Bacteriology of human and animal bite wounds. J Clin Microbiol. 1978;8(6):667–72.
55. Lacroix JM, Walker C. Characterization of a beta-lactamase found in Eikenella corrodens. Antimicrob Agents Chemother. 1991;35(5):886–91.
56. Hamilton JD, Larke B, Qizilbash A. Transmission of hepatitis B by a human bite: an occupational hazard. Can Med Assoc J. 1976;115(5):439–40.
57. Figueiredo JF, Borges AS, Martinez R, Martinelli Ade L, Villanova MG, Covas DT, et al. Transmission of hepatitis C virus but not human immunodeficiency virus type 1 by a human bite. Clin Infect Dis. 1994;19(3):546–7.
58. Andreo SM, Barra LA, Costa LJ, Sucupira MC, Souza IE, Diaz RS. HIV type 1 transmission by human bite. AIDS Res Hum Retroviruses. 2004;20(4):349–50.
59. Harrison M. A 4-year review of human bite injuries presenting to emergency medicine and proposed evidence-based guidelines. Injury. 2009;40(8):826–30.
60. Stevens DL, Bisno AL, Chambers HF, Everett ED, Dellinger P, Goldstein EJ, et al. Practice guidelines for the diagnosis and management of skin and soft-tissue infections. Clin Infect Dis. 2005;41(10):1373–406.
61. Aghababian RV, Conte Jr JE. Mammalian bite wounds. Ann Emerg Med. 1980;9(2):79–83.
62. Griego RD, Rosen T, Orengo IF, Wolf JE. Dog, cat, and human bites: a review. J Am Acad Dermatol. 1995;33(6):1019–29.
63. Green RJ, Dafoe DC, Raffin TA. Necrotizing fasciitis. Chest. 1996;110(1):219–29.
64. Andreasen TJ, Green SD, Childers BJ. Massive infectious soft-tissue injury: diagnosis and management of necrotizing fasciitis and purpura fulminans. Plast Reconstr Surg. 2001;107(4):1025–35.
65. Schecter W, Meyer A, Schecter G, Giuliano A, Newmeyer W, Kilgore E. Necrotizing fasciitis of the upper extremity. J Hand Surg Am. 1982;7(1):15–20.
66. Wilkerson R, Paull W, Coville FV. Necrotizing fasciitis. Review of the literature and case report. Clin Orthop Relat Res. 1987;216:187–92.
67. Giuliano A, Lewis Jr F, Hadley K, Blaisdell FW. Bacteriology of necrotizing fasciitis. Am J Surg. 1977;134(1):52–7.
68. Huang KF, Hung MH, Lin YS, Lu CL, Liu C, Chen CC, et al. Independent predictors of mortality for necrotizing fasciitis: a retrospective analysis in a single institution. J Trauma. 2011;71(2):467–73; discussion 73.
69. Schmid MR, Kossmann T, Duewell S. Differentiation of necrotizing fasciitis and cellulitis using MR imaging. AJR Am J Roentgenol. 1998;170(3):615–20.
70. Wong CH, Khin LW, Heng KS, Tan KC, Low CO. The LRINEC (Laboratory Risk Indicator for Necrotizing Fasciitis) score: a tool for distinguishing necrotizing fasciitis from other soft tissue infections. Crit Care Med. 2004;32(7):1535–41.
71. Golger A, Ching S, Goldsmith CH, Pennie RA, Bain JR. Mortality in patients with necrotizing fasciitis. Plast Reconstr Surg. 2007;119(6):1803–7.
72. Riseman JA, Zamboni WA, Curtis A, Graham DR, Konrad HR, Ross DS. Hyperbaric oxygen therapy for necrotizing fasciitis reduces mortality and the need for debridements. Surgery. 1990;108(5):847–50.
73. Kishi K, Hirai K, Hiramatsu K, Yamasaki T, Nasu M. Clindamycin suppresses endotoxin released by ceftazidime-treated Escherichia coli O55:B5 and subsequent production of tumor necrosis factor alpha and

interleukin-1 beta. Antimicrob Agents Chemother. 1999;43(3):616–22.
74. van Langevelde P, van Dissel JT, Ravensbergen E, Appelmelk BJ, Schrijver IA, Groeneveld PH. Antibiotic-induced release of lipoteichoic acid and peptidoglycan from Staphylococcus aureus: quantitative measurements and biological reactivities. Antimicrob Agents Chemother. 1998;42(12):3073–8.
75. Elhassan BT, Wynn SW, Gonzalez MH. Atypical infections of the hand. J Am Soc Surg Hand. 2004; 4(1):42–9.
76. Al-Qattan MM, Al-Namla A, Al-Thunayan A, Al-Omawi M. Tuberculosis of the hand. J Hand Surg Am. 2011;36(8):1413–21; quiz 22.
77. Wynn SW, Elhassan BT, Gonzalez MH. Infections of the hand in the immunocompromised host. J Am Soc Surg Hand. 2004;4(2):121–7.
78. Lakhanpal S, Linscheid RL, Ferguson RH, Ginsburg WW. Tuberculous fasciitis with tenosynovitis. J Rheumatol. 1987;14(3):621–4.
79. Evanchick CC, Davis DE, Harrington TM. Tuberculosis of peripheral joints: an often missed diagnosis. J Rheumatol. 1986;13(1):187–9.
80. Martini M, Benkeddache Y, Medjani Y, Gottesman H. Tuberculosis of the upper limb joints. Int Orthop. 1986;10(1):17–23.
81. al-Qattan MM, Bowen V, Manktelow RT. Tuberculosis of the hand. J Hand Surg Br. 1994;19(2):234–7.
82. Subasi M, Bukte Y, Kapukaya A, Gurkan F. Tuberculosis of the metacarpals and phalanges of the hand. Ann Plast Surg. 2004;53(5):469–72.
83. Hooker RP, Eberts TJ, Strickland JA. Primary inoculation tuberculosis. J Hand Surg Am. 1979;4(3):270–3.
84. Andronikou S, Smith B. “Spina ventosa”-tuberculous dactylitis. Arch Dis Child. 2002;86(3):206.
85. Wang MN, Chen WM, Lee KS, Chin LS, Lo WH. Tuberculous osteomyelitis in young children. J Pediatr Orthop. 1999;19(2):151–5.
86. Bhargava AD, Malaviya AN, Kumar A. Tuberculous rheumatism (Poncet’s disease): a case series. Indian J Tuberc. 1998;45:215–20.
87. Falkinham 3rd JO. Epidemiology of infection by nontuberculous mycobacteria. Clin Microbiol Rev. 1996;9(2):177–215.
88. Hurst LC, Amadio PC, Badalamente MA, Ellstein JL, Dattwyler RJ. Mycobacterium marinum infections of the hand. J Hand Surg Am. 1987;12(3):428–35.
89. Chow SP, Stroebel AB, Lau JH, Collins RJ. Mycobacterium marinum infection of the hand involving deep structures. J Hand Surg Am. 1983;8(5 Pt 1): 568–73.
90. Scollard DM, Adams LB, Gillis TP, Krahenbuhl JL, Truman RW, Williams DL. The continuing challenges of leprosy. Clin Microbiol Rev. 2006;19(2):338–81.
91. al-Qattan MM. Opportunistic mycotic infections of the upper limb. A review. J Hand Surg Br. 1996;21(2): 148–50.
92. Eo S, Jones NF. Fungal infections of the hand and upper extremity. J Am Soc Surg Hand. 2004;4(4): 250–5.
93. Amadio PC. Fungal infections of the hand. Hand Clin. 1998;14(4):605–12.
94. Rodgers P, Bassler M. Treating onychomycosis. Am Fam Physician. 2001;63(4):663–72, 77–8.
95. Hitchcock TF, Amadio PC. Fungal infections. Hand Clin. 1989;5(4):599–611.

Pain Management in Disorders of the Hand

20

Carmel Martin, Richard Kennedy, and Jeremy Cashman

Keywords
Upperlimb • Hand • Pain • Nociception • Acute • Chronic • Trauma • Nociceptive • Neuropathic • Analgesia • Preventive • Multimodal • CRPS • Phantom • Biopsychosocial • Pain management

The ability of an individual's somatosensory system to detect noxious and potentially tissue damaging stimuli is an important protective mechanism. The multiple interrelated peripheral and central processes that detect these noxious stimuli are defined as nociception. But it is the interpretation of those multiple processes within the central nervous system (CNS) that results in the sensation of pain. Pain is defined by the International Association for the Study of Pain (IASP) as:

> An unpleasant sensory and emotional experience associated with actual or potential tissue damage.

Acute pain is defined as:

> Pain of recent onset and of probable limited duration. It usually has an identified temporal and causal relationship to injury or disease. [1]

Whereas chronic pain is described as a pain which:

> Commonly persists beyond the time of healing of an injury and frequently there may not be any clearly identifiable cause [1]

However, it is now recognised that rather than being separate entities, acute and chronic pain may represent different aspects of a single pain continuum. Furthermore pain is a multifactorial condition that, in addition to somatosensory stimuli, is affected by an individual's previous pain experience, psychology and environment. Therefore it is vital for the effective clinical management of any patient with pain that these factors are also considered.

C. Martin, MB BCh
Anaesthesia and Acute Pain Management, Wrightington Hospital, Hall Lane, Appley Bridge, Wigan, Lancashire WN6 9EP, UK

R. Kennedy, MB BS, FRCA
Anaesthesia and Pain Management, St George's Hospital, Blackshaw Road, London SW17 0QT, UK
e-mail: rcpkennedy@hotmail.com

J. Cashman, MB BS, BA, BSc, MD, FRCA, FFPMRCA (✉)
Anaesthesia and Acute Pain Management, St George's Hospital, Blackshaw Road, London SW17 0QT, UK
e-mail: jeremy.cashman@stgeorge's.nhs.uk

I.A. Trail, A.N.M. Fleming (eds.), *Disorders of the Hand: Volume 1: Hand Injuries*,
DOI 10.1007/978-1-4471-6554-5_20, © Springer-Verlag London 2015

Pain Physiology

Nociceptive Pain

Peripheral Nociception

In acute pain the detection of noxious stimuli caused by trauma and/or surgery requires the activation of peripheral ion channels and transduction to a nerve action potential for conduction to the central nervous system. These primary afferent nociceptors are distributed widely throughout skin, muscle, joints and viscera and include the transient receptor potential (TRP) superfamily of cell membrane ion channels that are implicated in thermo-, chemo- and mechanoreception. Cell membrane voltage-gated sodium channels are involved in the conduction of the nerve action potentials to the spinal cord in sensory nerve fibres. Two types of nerve fibre conduct the action potential; small unmyelinated, slow conducting C fibres and larger myelinated, faster conducting A-delta fibres. C fibres are more numerous and transmit a broad range of stimuli including heat, cold, pressure and chemical. Local anaesthetic agents which act by blocking voltage-gated sodium channels can cause not only sensory nerve blockade but also motor nerve and sympathetic nerve blockade as well.

Central Nociception

Primary afferent nociceptors project to the dorsal root ganglia (DRG) of the spinal cord. The terminals of these neurons contain excitatory amino acids (glutamate and aspartate) and peptides (calcitonin gene related peptide [CGRP] and substance P) that act as neurotransmitters. The N-methyl-D-aspartate (NMDA) receptor is central in pain transmission to higher centres. Presynaptic NMDA receptors located on the terminals of small diameter pain fibres facilitate and prolong the transmission of nociceptive messages through the release of substance P and glutamate. The spinothalamic tract transmits nociceptive signals to the thalamus and somatosensory cortex. The thalamus also receives afferent input from the amygdala, which explains the affective and emotional components of pain. In addition to the excitatory processes outlined above there are descending inhibitory pathways that modulate C fibre activity in the DRG. These can be activated by higher brain function (distraction, cognitive input etc.) or by endogenous neurotransmitters such as endorphins, enkephalins, noradrenaline and serotonin. Analgesia may be achieved by reducing excitation either peripherally (e.g. NSAIDs, local anaesthetics) or centrally (e.g. paracetamol, NSAIDs, local anaesthetics, ketamine) or by enhancing inhibition (e.g. opioids, antidepressants).

Neuropathic Pain

Neuropathic pain has been defined as:

> Pain initiated or caused by a primary lesion or dysfunction of the nervous system. [1, 2]

Although commonly considered a cause of chronic pain, neuropathic pain is present in an estimated 3 % of trauma and surgical patients and may be seen early in the postoperative period [3]. Plasticity of the nervous system is fundamental to the mechanism of neuropathic pain [4]; the nervous system demonstrates a great ability to alter it's threshold to a painful stimulus and the amount of afferent nociceptive transmission to that stimulus.

Nerve injury results in structural and functional changes at multiple points in the nociceptive pathways. Peripherally, alteration of sodium channel sensitivity and "cross talk" of nociceptors results in increased excitability ('*allodynia*' and '*hyperalgesia*' see section "Pre-emptive and preventive analgesia") and spontaneous pain. Centrally, sensitisation occurs, reducing transmission threshold with loss of descending inhibition. Continued stimulation of nociceptors in the spinal cord results in activation of nociceptors adjacent to those already affected leading to an increase in the proportion of the limb affected by pain. This is referred to as "*wind up*".

Progression of Acute Pain to Chronic Pain

Inadequately treated severe acute pain has an increased risk of becoming chronic with the

Table 20.1 Incidence of acute pain following surgery

Pain severity	Incidence of acute pain (%); Mean (95 % confidence interval)
Moderate-to-severe at rest	29.7 % (26.4–33.0)
Moderate-to-severe on movement	32.2 % (24.8–39.6)
Severe	10.9 % (8.4–13.4)

attendant risk of social disability and isolation in adults [5]. However, a survey of published studies has shown that post-operative pain is an under managed condition with an unacceptable level of poor pain control after major surgery (Table 20.1) [6].

Many patients with chronic pain can relate their pain to an acute injury. Indeed, chronic pain is often referred to as persistent pain. There is a well-established link between acute post-traumatic and post-surgical pain and chronic pain conditions (Table 20.2) [7, 8]. Furthermore a number of risk factors for the development of chronic pain have been identified (Table 20.3) [7, 8].

Chronic or persistent pain represents a significant source of physical and emotional disability with considerable economic consequences [7]. Chronic pain often has both nociceptive and neuropathic components. Few controlled studies have investigated the aetiology, prevention and treatment of the transition between the two pain states.

Pre-emptive and Preventive Analgesia

The concept of pre-emptive analgesia is based on the hypothesis that pre operative analgesia may enhance postoperative analgesia and hence reduce progression to chronic pain conditions. However clinical studies have reported conflicting outcomes [9, 10]. This has led to shifting the focus from the timing of the intervention to the intervention itself. The concept of protective or preventive analgesia has evolved from this. Preventive analgesia describes a technique that reduces sensitisation in the acute phase and the persistence of analgesia beyond the duration of the intervention.

Table 20.2 Incidence of chronic pain following surgery

Surgery	Incidence of chronic pain (%)	Incidence of severe (>5/10) chronic pain (%)
Amputation	30–85	5–10 %
Thoracotomy	5–65	10 %
Mastectomy	11–57	5–10 %
Inguinal hernia	5–63	2–4 %
Coronary bypass	30–50	5–10 %
Caesarean section	6–55	4 %
Cholecystectomy	3–50	Not estimated
Vasectomy	0–37	Not estimated
Dental surgery	5–13	Not estimated

Source Kehlet et al. [7] and Macrae [8]; Review of studies with >50 patients

Table 20.3 Risk factors for the development of chronic pain

Preoperative risk factors	Persistent pre-existing pain; moderate-to-severe or worse
	Younger age
	Female gender
	Genetic predisposition
	Patient anxiety trait
	Patient tendency to 'catastrophise'
	Previous litigation/compensation
	Repeat surgery
Intraoperative risk factors	Surgical approach associated with risk of nerve damage
Postoperative risk factors	Acute pain; moderate-to-severe or worse
	Pain radiation to surrounding area
	Patient anxiety state
	Patient depression
	Patient neuroticism
	Patient psychological vunerability
	Neurotoxic chemotherapy

Source Kehlet et al. [7] and Macrae [8]

There is increasing evidence that early analgesic interventions in the perioperative period reduce the incidence of chronic pain after surgery (Table 20.4) [11].

It is thought that preventive analgesia reduces central sensitisation and "wind up". Current opinion considers that for this strategy to be effective, it should be instituted as early as pos-

Table 20.4 Influence of pre-emptive and preventive analgesia on the incidence of chronic pain after surgery

	Number of studies	Pre-emptive analgesia		Preventive analgesia		Opposite effects
Medication		Positive	Negative	Positive	Negative	
Local anaesthetic agents	13	3	3	6	1	2
Gabapentin	6	0	1	4	1	0
Mutimodal technique	5	1	1	2	2	0
NMDA antagonists	14	2	1	9	4	1
NSAIDs	14	7	3	4	2	0
Opioids	5	3	1	0	1	0

Source Katz and Clark [11], p. 165

sible and continued for as long as the sensitising stimulus persists.

Assessment of Pain

Reliable, accurate and reproducible assessment of pain is fundamental to the process of diagnosing a patient's pain, selecting the correct analgesic therapy, evaluating response and modifying the therapy accordingly. Pain is an individual experience and many factors including previous pain experiences, cultural background, disease/surgical prognosis, coping strategies, fear, anxiety and depression will interact to produce what the patient then describes as pain. There is often a poor correlation between the patient's assessment of the pain and the nursing or medical staff's estimate of the pain that the patient is experiencing.

Pain should be assessed using a biopsychosocial model that recognises physiological, psychological and environmental factors that influence the overall clinical picture [12].

The assessment of pain should include a thorough medical history and physical examination that may indicate further investigation.

Table 20.5 The pain history

Site of pain
Primary location
Radiation
Circumstances associated with pain onset
Including details of trauma or surgery
Character of pain
Nociceptive descriptors; sharp, throbbing, aching etc
Neuropathic descriptors; LANSS questionnaire
Intensity of pain
At rest and on movement
Exacerbating or relieving factors
Associated symptoms
Headache, nausea etc
Effects of pain on activity and sleep
Treatment
Current and previous medications; details of dose, frequency, efficacy, side effects
Other treatments; injections, TENS etc
Other health professionals consulted
Relevant medical history
Prior or coexisting pain conditions and their outcomes
Prior medical or coexisting conditions; Multiple sclerosis, HIV, diabetes, cancer
Other factors
Belief concerning the cause of pain
Knowledge and expectations of treatment
Presence of anxiety or psychiatric conditions

The Pain History

A good pain history provides important diagnostic information that will help distinguish between nociceptive and neuropathic pain (Table 20.5).

Character of Pain

Nociceptive pain is often described as sharp, hot or stinging and is generally well localised. It is usually associated with surrounding tissue inflammation and tenderness.

Features suggestive of neuropathic pain include:

- Surgery or trauma with a high risk of nerve injury.
- Descriptors such as burning, shooting or stabbing.
- Paroxysmal or spontaneous pain with no clear precipitating factors

- Allodynia; pain due to a stimulus that is not normally painful (e.g. light touch).
- Dysaesthesia; spontaneous or evoked unpleasant abnormal sensations
- Hyperalgesia; increased response to a normally painful stimulus
- Hypoaesthesia; reduced sensation to a stimulus
- Autonomic features; changes in skin colour, temperature and sweating
- Phantom phenomena

Intensity of Pain

Pain intensity should be assessed regularly during the postoperative period and the frequency of assessment should be increased if the pain is poorly controlled or if the pain stimulus or treatments are changing. In adults, three common methods of self-reported pain measurements are used.

- Visual Analogue Scale
- Numerical Rating Scale
- Verbal Rating Scale

Each of these methods of measurement is reasonably reliable as long as the endpoints and adjectives are carefully selected and standardized.

Visual Analogue Scale

The visual analogue scale (VAS) uses a 10 cm line with endpoint descriptors "*no pain*" marked at the left end of the line and "*worst pain imaginable*" marked at the right end of the line. Patients are asked to mark a point on the line that best represents their pain. The distance from "*no pain*" to the patients mark is measured and this value equates to the VAS score. The advantage of the VAS is that it avoids imprecise descriptive terms. However, it can be more time consuming than other simple scoring methods and some patients may have difficulty understanding or performing this score, especially in the immediate postoperative period [13].

Numerical Rating Scale

The numerical rating scale (NRS) is a Likert scale and as such is similar to the VAS pain measurement system. Patients are asked to imagine that "*0*" equals "*no pain*" and "*10*" equals "*the worst pain imaginable*" and then to assign a number on this scale that best represents their pain. The advantage of the NRS is that it does not require any equipment. However, problems may occur if there is a language barrier or the patient has some other difficulty in understanding the scoring system.

Table 20.6 Physical examination

Inspection
Scars, symmetry with contra-lateral hand, temperature, erythema, sweating, nail changes, oedema, muscle wasting, fasciculation
Palpation
Scar tenderness, palpable mass under scar (neuroma)
Dermatomal/non-dermatomal distribution of pain
Allodynia, dysaesthsia, hyperalgesia
Passive movement
Joint tenderness, range of movement
Neurology[a]
Sensory function; light touch, temperature, pin prick, tuning fork
Motor function

[a]Should include spinal cord and brachial plexus as well as peripheral nerves

Verbal Rating Scale

The verbal rating scale (VRS) uses different words to rate pain; "*none*", "*mild*", "*moderate*", "*severe*", and "*very severe*" or "*worst pain imaginable*". The VRS is a categorical system of pain measurement but it is common for a number to be assigned to each descriptor. The advantage of the VRS is that it is quick and simple to administer. However, the small number of descriptors may be a disadvantage.

Examination

Physical examination can be tailored to assist in a pain diagnosis. The list is not exhaustive (Table 20.6).

Acute Pain

Postoperative Pain

The goals of postoperative pain management are to:

Table 20.7 Drug treatment and the World Health Organisation (WHO) analgesic ladder

		Step 3: **Strong opioids**	**Adjuvants**
		Buprenorphine	Amitriptyline
		Diamorphine	Carbamazepine
		Fentanyl	Clonidine
	Step 2: **Weak opioids**	Hydromorphone	Dexamethasone
	Codeine	Morphine	Gabapentin
Step 1: **Non opioids**	Tramadol	Pethidine	Ketamine
Paracetemol	Codeine-Paracetamol combinations	Oxycodone	Neostigmine
NSAIDs		Tapentadol	Pregabalin

- provide subjective comfort (rather than complete abolition of pain)
- inhibit trauma-induced nocioceptive impulses in order to blunt autonomic and somatic reflex responses to pain
- enhance restoration of function by allowing the patient to breathe, cough and move more easily.

In addition, the effective treatment of acute post-operative or post-traumatic pain may reduce the incidence of chronic pain [14]. Acute post-operative pain differs from chronic or cancer pain because it is transitory. There may also be an affective component relating to anxiety about the outcome of the surgical procedure and concern regarding suboptimum analgesia. Studies have shown that for patients awaiting surgery the possibility of severe acute postoperative pain is a major concern. In addition, uncontrolled postoperative pain can lead to delayed recovery from surgery, pulmonary dysfunction and hypoxia, together with decreased mobility and subsequent increased risk of thromboembolism.

Diagnosis

Acute pain, whether due to a medical or surgical condition, should be relieved as soon as possible. Simultaneously investigate and treat the underlying cause; it is rare for analgesia to mask a diagnosis

Management: Patient Information and Education

Patient education is important for optimal pain management. The surgeon also has a significant responsibility in the prevention and treatment of postoperative pain. A team approach is necessary to effectively prevent and treat peri-operative pain. An Acute Pain Service (APS) is the optimal structure for postoperative pain management but surgeons and nurses must be included. Most UK hospitals now have an APS which develops strategies for staff training, quality control and auditing.

Management: Pharmacotherapy

Drugs used in acute pain and chronic pain management are generally the same and all follow the World Health Organisation (WHO) analgesic ladder (Table 20.7). The general principle for treatment of pain is to start at the bottom of the ladder and work up the ladder. However, in severe postoperative pain it may be necessary to start at the top of the ladder. Thus, strong opioids (step 3) may be required at the outset, superimposed on a background of step 1 and 2 drugs. As the pain subsides it is then appropriate to step down the ladder to eventually use only non-opioid drugs. The oral route is preferred for all steps.

Simple (Non Opioid) Analgesics

Paracetamol: Paracetamol (acetaminophen), an acetanilide derivative, is one of the most commonly used analgesic drugs worldwide, due in part to an excellent safety record. Paracetamol has analgesic and antipyretic activity, but lacks anti-inflammatory activity. It is believed to be a centrally acting cyclo-oxygenase (COX) inhibitor with weak peripheral effects. Current evidence points to multisite activity in the CNS involving inhibition of prostaglandin synthesis, and interaction with both serotonergic and cannabinoid pathways.

Paracetamol can be given orally, rectally and parentally. Oral Paracetamol is well absorbed from the proximal small bowel and is not subject to significant first-pass metabolism with oral bioavailability estimated at between 63 and 89 % in adults. In contrast, rectal administration is restricted by slow onset time and unpredictable bioavailability. An intravenous formulation provides rapid and predictable therapeutic plasma concentrations and is particularly useful in patients under anaesthesia or in patients who are unable to take oral medication. The recommended dose of intravenous paracetamol is 15 mg/kg, with a maximum daily dose of 60 mg/kg. Peak plasma concentration (*Cmax*) is achieved approximately 25 min after a 1 g intravenous infusion compared to 45 min after a 1 g oral dose. Clinically this difference has been shown to resul in a faster onset of analgesia. A meta analysis of single dose intravenous paracetamol involving 36 studies showed that 37 % of patients receiving paracetamol experienced at least 50 % pain relief over 4 h compared with 16 % receiving placebo [15].

As paracetamol does not cause gastric irritation, is relatively non-toxic in therapeutic doses and has minimal side effects it should be considered as a basic building block of most postoperative analgesic regimens. Paracetamol is an effective and well tolerated agent in the management of mild-to-moderate pain and as it has none of the renal or cardiovascular side effects that characterize anti-inflammatory drugs. It can be used in both NSAID- and opioid-sparing roles. Combining paracetamol with NSAIDs appears to result in a synergistic interaction but so far there is only limited evidence that the interaction is advantageous in the clinical setting. In contrast studies have shown that paracetamol has a significant morphine sparing effect in the order of 20 % in the first 24 h. However, this is not associated with a reduction in morphine related side effects.

Non-Steroidal Anti-Inflammatory Drugs (***NSAIDS***): Postoperative analgesia comparable with that of opioids has been demonstrated with the NSAIDs. An opioid-sparing effect in the order of 30–50 % has also been observed, as well as a reduction in opioid induced nausea, vomiting and respiratory depression. This reduction in opioid requirement and side-effects may benefit the patient by producing superior postoperative analgesia and even reduced hospital stay.

The analgesic, anti-inflammatory and antipyretic effects of NSAIDS as well as their most notable side effects are attributed to inhibition of cyclo-oxygenase (COX) 1 and 2 receptors, thereby reducing production of mediators of the acute inflammatory response. Classic NSAIDs such as diclofenac, ibuprofen and ketorolac are widely prescribed as analgesics and anti-inflammatory agents that exert their inhibition of prostanoids synthesis through blockade of both COX-1 and COX-2 receptors. Unfortunately these drugs can have a number of undesirable adverse effects.

Erosions of the gastrointestinal tract (especially the stomach) can develop. These are due not only to local irritation of the mucosa but also to decreased gastric mucus production, reduced mucosal blood flow and increased gastric acid production. The problems may be diminshed but not avoided altogether if the drugs are administered by parenteral or rectal routes. Gastric irritation, dyspepsia and ulceration may develop at any time but are less likely with short-term treatment. Pre-existing peptic ulcer disease may also be exacerbated.

NSAID induced reduction in prostaglandin levels may result in decreases in renal blood flow and acute kidney injury (AKI). Pre-existing renal impairment may increase this risk. In the healthy patient, renal blood flow is not normally prostaglandin dependent. However, renal blood flow may be altered during anaesthesia. NSAIDs can also cause sodium, potassium and water retention which may lead to oedema in some patients. Abnormalities in liver function tests may also occur but are usually transient.

Aggregation of platelets depends on a balance between prostacyclin (from endothelial cells) and thromboxane A2 (from platelets). The former is a vasodilator and inhibits platelet aggregation. NSAIDs inhibit the synthesis of both these factors and the net balance will determine the tendency to bleed. NSAIDs reversibly inhibit

platelet cyclo-oxygenase and the effect lasts only as long as the drug remains in the blood. Increases in bleeding have been reported with the use of NSAIDs [16].

NSAID induced bronchospasm can occur in some asthmatic patients. Up to 20 % of adult asthmatics may develop aspirin-induced bronchospasm and a cross-sensitivity does exist between aspirin and other NSAIDs. As a result NSAIDs should be used with caution in these patients.

Skin reactions are the second most common unwanted effect of NSAIDs. Patients can present with a variety of skin conditions ranging from mild rashes, urticaria and photosensitivity to occasionally more serious and potentially fatal varieties.

COX-2 Inhibitors: Selective COX-2 inhibitors (Coxibs) were developed in an effort to reduce the incidence of potentially serious gastrointestinal and renal adverse effects associated with traditional NSAIDs. The coxibs were expected to have similar analgesic efficacy to traditional NSAIDs but with reduced risk of bleeding and of gastrointestinal toxicity. However, large outcome and epidemiological studies suggest that while COX-2 inhibitors do confer improved gastrointestinal safety they are not devoid of gastrointestinal side effects during long term use. In addition, concerns have also been raised regarding the increased incidence of thrombotic complications associated with selective COX-2 inhibitors. This can lead to myocardial infarction and stroke. Selective COX-2 inhibition may increase the risk of vascular thrombus formation by upsetting the balance between pro- and anti-platelet aggregation effects [17]. Two coxibs have already been withdrawn because of safety concerns; rofecoxib because of cardiovascular problems and valdecoxib because of serious skin reactions [18].

The role of coxibs in postoperative pain management is still not clearly defined. Also there is a cost implication as they are considerably more expensive than the older non-selective NSAIDS. In contrast, there have been many studies done using different NSAIDS pre-emptively (see section "Pre-emptive and preventive analgesia") and while there have been some mixed results, it is common practice in orthopaedic patients to commence NSAIDs pre-emptively.

Opioid Analgesics

Opioid drugs are effective postoperative analgesics and are the principle drugs used to combat moderate-to-severe pain. The most commonly used drugs are buprenorphine, codeine and dihydrocodeine, fentanyl, hydromorphone, morphine, oxycodone and tramadol. Pethidine is no longer recommended for postoperative analgesia because repeat doses may lead to accumulation of its metabolite, norpethidine, which can cause confusion and seizures. It also has a very high incidence of nausea and vomiting.

Opioids can be given orally, intramuscularly, intravenously, subcutaneously, transdermally, transmucosally and even intra-articularly. They can also be administered by the spinal and epidural routes. Oral dosing of opioids is usually the most convenient and least expensive route of administration. It is appropriate as soon as the patient can tolerate oral intake and is the mainstay of pain management in the ambulatory surgical population. Intravenous patient-controlled analgesia (PCA), in which the patient has control over the timing of each dose, has become the standard method of providing postoperative analgesia after major surgery.

Pharmacology: Morphine and codeine are alkaloids of opium. These naturally occurring substances are called *opiates*. All drugs that have morphine-like actions, whether naturally occurring or synthetic, are called *opioids*. Unlike less potent drugs, opioids influence the emotional aspects of pain such as anxiety and fear as well as altering the pain threshold, making pain more tolerable. Despite all the advances in developing new analgesics morphine remains the gold standard against which all new analgesics are compared.

A number of different opioid receptors have been identified; mu (μ), delta (δ), kappa (κ) and sigma (σ). There are two subtypes of μ receptors, μ1 and μ2 with subtype μ1 mediating analgesia and μ2 responsible for respiratory depression. All currently available μ receptor agonist opioids activate both subtype μ1 and μ2. The effects of

Table 20.8 Effects of activation of opioid receptors

Mu receptor
Analgesia, bradycardia, euphoria, inhibition of gut motility, miosis, nausea and vomiting, respiratory depression, inhibition of gut motility
Kappa receptor
Analgesia, miosis, sedation
Delta receptor
Analgesia
Sigma receptor
Dysphoria, confusion, hallucinations, mydriasis

activating the different receptors are outlined in Table 20.8.

Opioid drugs are categorised according to endorphin receptor affinity and activity as follows.

- *Agonists*: drugs that bind to and stimulate opioid receptors e.g. codeine, diamorphine, morphine, oxycodone, pethidine
- *Antagonists*: drugs that bind to but do not stimulate opioid receptors e.g. naloxone
- *Partial Agonists*: drugs that bind to and stimulate opioid receptors but have a ceiling (submaximal) effect e.g. buprenorphine
- *Agonist-Antagonists*: drugs that are agonists at one opioid receptor type and antagonist at another e.g. nalbuphine, pentazocine.

Agonist-antagonist drugs derive their analgesic actions principally from k receptor activation while acting as antagonist at the μ receptor. Most of these drugs also activate (at least partially) the δ receptor. These drugs are associated with a higher incidence of psychomimetic effects (e.g. dysphoria) and are far less commonly used in clinical practice

Side Effects: All of the commonly used opioid drugs act primarily at the μ receptor and have a similar spectrum of side effects when used in equi-analgesic doses (Table 20.9).

The most serious side effect of opioid drugs is respiratory depression which may be severe and result in hypoxia. Therefore it is routine practice to monitor respiratory rate and to use continuous pulse oximetry in all postoperative patients in the Recovery room or Post Anaesthetic Care Unit (PACU). Sedation should be routinely monitored postoperatively using a sedation score. Opioid

Table 20.9 Side effects of opioids

Cardiovascular
Bradycardia, myocardial depression, vasodilation
Central nervous system
Euphoria/dysphoria, miosis, muscle rigidity, nausea and vomiting, sedation,
Cutaneous
Pruritus (common with morphine)
Gastrointestinal
Constipation (very uncommon), delayed gastric emptying
Genitourinary
Urinary retention
Respiratory
Respiratory depression

drugs are implicated as one of the risk factors in post operative nausea and vomiting (PONV) [19]. In all cases where opioids are used intra-operatively or post-operatively, antiemetic prophylaxis should be given. Combinations of anti-emetic prophylaxis with drugs having different mechanisms of action have been shown in many studies to markedly reduce the incidence of PONV. Ondansetron and cyclizine have been shown to achieve a response rate of 95 % [20].

Routes of Opioid Administration – Oral: The most commonly used drugs for mild to moderate pain are codeine, codeine plus-paracetamol combinations, oxycodone and tramadol.

Codeine, like morphine, is a naturally occurring alkaloid. It is metabolized in the liver where 5–10 % of the dose is converted to morphine which accounts for its main analgesic effect. Oral codeine is routinely given for the treatment of mild to moderate pain. Codeine plus paracetamol preparations are the most commonly used opioid analgesics for postoperative pain and day case surgery procedures. However with fixed dose combinations of codeine plusparacetamol there is a relatively flat dose-response curve and a clear ceiling effect. The analgesic potential of fixed-dose combinations is generally limited by unacceptable side effects associated with the paracetamol component. Fixed-dose combination preparations must be given every 4–6 h and a delay in administration, especially when ordered on a "*on patient request*" basis, may result in lower plasma opioid concentration and thus the

re-emergence of pain. For this reason it is recommended that codeine and paracetamol are prescribed separately on drug charts.

Oxycodone is a thebaine derivative that is used for the treatment of moderate to severe pain. It is available in immediate and sustained release formulatations. Sustained release oxycodone (oxycontin®) has been shown to be very effective in post-operative pain relief. The biphasic pattern of absorption of oxycontin® following oral administration results in an initial rapid absorption of approximately 40 % of the dose, and onset of analgesia over the first hour. This is then followed by sustained release of the drug and stable plasma levels over a 12-h period, therefore enabling twice daily dosing. A trial of oxycontin® in orthopaedic patients has shown that it provides effective analgesia, shortened hospital stay and a reduced frequency of analgesic administration after hip and knee arthroplasty [21].

Tramadol provides effective analgesia in patients undergoing minor or intermediate surgery. It provides analgesia by two mechanisms, an opioid effect and an enhancement of descending serotonergic and adrenergic inhibitory pathways. It is a weak opioid and therefore has fewer of the typical opioid side-effects. Little respiratory depression occurs unless it is given in large doses. It causes less constipation and has less addiction potential. It can be given orally or intravenously, usually in a dose of 100 mg. As nausea is common after intravenous administration, antiemetics should be coadministered.

Routes of Opioid Administration – Intramuscular: Traditionally, intramuscular (IM) opioids, particularly morphine, have been ordered 4-hourly "on patient request" as the mainstay of postoperative analgesia. Opioids given by iIM injection are generally not able to maintain steady analgesic plasma levels for 2–4 h. A reluctance and a fear for patient safety has played a major role in the lack of effectiveness of IM analgesia regimens. This method of administration has many disadvantages and is no longer recommended for the following reasons

- Intramuscular injections are painful
- It is difficult to sustain steady blood concentration
- There are prolonged intervals when the patient experiences pain
- The patient may be unaware that pain relief is available
- There may be delays between request for pain relief and the actual time of administration

In addition considerable input from nursing/ancillary staff, often a scarce resource, is required.

Routes of Opioid Administration – Intravenous: For an opioid to be effective it must reach a certain concentration in the blood and dose titration is needed to individualize treatment for each patient. The best way to monitor the effectiveness of opioid treatment is the pain score. The aim of pain treatment is to make the patient comfortable without undue sedation. Analgesia is usually obtained more rapidly if intravenous (IV) bolus doses are administered to "load" the patient.

Intravenous patient-controlled analgesia (PCA) using opioids, usually morphine, has become the standard of care in many hospitals for the management of post-operative pain. The term PCA is used to describe a method of analgesia which employs sophisticated infusion devices that allow patients to self-administer opioids, usually intravenously. PCA machines incorporate microprocessor-driven syringe pumps that within preset limits will deliver a bolus dose of drug when the patient presses a demand button. Access to the syringe and the microprocessor programme are key or access code protected. Variables are programmed into the PCA machine to control how much opioid the patient can receive. Most PCA machines can also deliver a continuous or "background" infusion. Patients are instructed to push the demand button whenever they are uncomfortable. Alternative demand devices (pneumatic device, pressure-sensitive pad, etc.) may be required if the patient is unable to use either hand. The inherent safety of PCA lies in the fact that as long as the machine is in PCA mode only (i.e. there is no continuous infusion; see below) no further doses will be delivered if the patient become over-sedated and makes no further demands.

Programmable PCA variables include

- *Loading Dose*: The dose of opioid needed to make the patient comfortable before PCA

maintenance therapy is started. Due to wide inter patient-variation, the dose is individualized for each patient and titrated against their pain score
- *Bolus Dose*: The amount of opioid that the PCA machine will deliver when the demand button is pressed. The standard dose of morphine is 1 mg. However the dose may be increased or reduced according to the patient's age
- *Lock-Out Interval*: The time from the end of the delivery of one dose until the machine will respond to another demand. An interval of 5 min is the standard
- *Continuous* (*Background*) *Infusion*: This feature is not routinely used as it is associated with an increased risk of side-effects with no improvement in the quality of analgesia.

Many studies have been carried out comparing PCA to conventional methods of analgesia. Systemic reviews in 1992 and 2001 found conflicting results regarding the effectiveness of PCA compared to traditional analgesia. However, a Cochrane review in 2007 of 55 studies with a total of 3,861 patients found that, overall, more patients in the PCA group were satisfied with it as a method of analgesia. Also, pain intensity during the first 24 h was eight points lower in the PCA group than in the group receiving conventional analgesia, nine points lower between 25 and 48 h and 13 points lower between 49 and 72 h [22].

Adjuvants

Although opioid analgesics along with paracetamol, NSAIDs and local anaesthetics are important during the immediate perioperative period, the use of other adjuvant drugs is likely to assume a greater role in post-operative pain management.

Gabapentin and Pregabalin: The anticonvulsants gabapentin and pregabalin can be used as components of a multi modal approach to postoperative pain. Results from a number of clinical trials have demonstrated analgesic efficacy and opioid sparing effects associated with the gabapentin and pregabalin. A meta-analysis has shown that gabapentin given preoperatively decreases pain scores and analgesic consumption in the first 24 h after surgery. However, a significant reduction in the incidence of side effects could not be demonstrated [23].

Ketamine: The role of ketamine as a postoperative analgesic adjuvant is unclear with a number of studies of its effectiveness providing conflicting findings. However, a meta–analysis of studies has found that ketamine administered intravenously during anaesthesia in adults was associated with decreased postoperative pain intensity for up to 48 h, decreased cumulative 24 h morphine consumption and a delay in the time to first request for rescue analgesic [24]. Nevertheless the usefulness of ketamine as a component of a perioperative analgesic regimen may be limited by its potential side effects. Ketamine may, however, have a role in the treatment of persistent (chronic) pain.

Other Drugs: Clonidine, dexamethasone, magnesium and neostigmine may all have potential benefits in reducing pain but so far are not used routinely in clinical practice.

Management: Interventional Procedures

Local Anaesthetic Blockade

The hand is innervated by three major nerves (median, ulnar and radial), which in turn are terminal branches of the three cords of the brachial plexus.

The following local anaesthetic techniques for achieving upper limb analgesia may be employed:
- Local Infiltration
- Intra-Articular Joint Injection
- Intra-Venous Regional Anaesthesia
- Plexus Blocks
- Peripheral Nerve Blocks

Blocks can be extended by using catheter techniques and either continuous infusion or intermittent (patient-controlled) boluses of a long-acting local anaesthetic.

Local Infiltration: Single peri-operative injection of a local anaesthetic drug into wounds and incisions provides effective analgesia following minor soft tissue operations. Bupivacaine, a long acting local anaesthetic, is being replaced by its isomer levobupivacaine

(chirocaine®) and by ropivacaine, another long acting local anaesthetic, both of which have a better cardiovascular safety profile.

Intra-Articular Joint Injection: Intra-articular injection of local anaesthetic has been used for many years for arthroscopic knee surgery with good results. For shoulder, elbow and wrist arthroscopic surgery, intra-articular injection of local anaesthetic has been shown to be superior to surgical wound infiltration. Insertion of an intra-articular catheter at the end of the operation by the surgeon under direct vision is relatively simple. Dilute solutions of local anaesthetics are infused through the catheter postoperatively. The technique provides good pain relief whilst preserving motor function of the joint. A refinement is to connect the catheter to a PCA machine which allows the patient to self administer boluses of a long acting local anaesthetic.

Intravenous Regional Anaesthesia: Intravenous regional anaesthesia (IVRA), Biers Block, is a straightforward, reliable and simple method of providing anaesthesia for minor superficial surgery of the forearm and hand including reduction/fixation of fractures. Published series report successful anaesthesia in 96–100 % of patients with a low incidence of side-effects. The technique is only suitable for use in short operations (less than 30 mins) It is contra-indicated in children and is unsatisfactory in patients with fat arms and in hypertensive (systolic BP >200 mmHg) patients. The preferred local anaesthetic agent is 0.5 % prilocaine (up to 300 mg) but neither bupivacaine nor levobupivacaine should ever be used.

Plexus Blocks: Over 40 techniques of performing a proximal, regional plexus or cord block have been described but they are all modifications of four basic approaches as follows:

- Interscalene
- Supraclavicular
- Axillary
- Infraclavicular

No one technique is ideal, as they have different benefits and complications and their use depends on which is most appropriate for the intended surgery. For surgery on the hand and forearm axillary plexus block is the most widely used technique.

Axillary Plexus Block is easy to learn, has the lowest incidence of complications and is ideally suited to hand surgery. Traditionally the block was performed by eliciting paraesthesia using a blind needle technique around the axillary artery. Later a trans-arterial approach was used, which was then replaced by a nerve stimulator and a single injection technique. However, incomplete anaesthesia is reported in 10–40 % of patients when a paraesthesia, transarterial technique or stimulation with single injection techniques is used. A multiple stimulation technique of each individual nerve produces faster and more extensive block with success rate between 90 and 94 %. Also if the patient is to remain awake for surgery it is important to supplement the block of the musculocutaneous nerve in order to avoid tourniquet pain during the procedure.

Complications include inadvertent vascular injection and nerve damage.

Transient post-operative neurological symptom associated with nerve blocks are relatively frequent at day 10 (8 %) but are infrequent after 1 month (2–4 %). Whilst block–related neurological sequelae lasting more than 6 months are exceedingly rare [25].

Peripheral Nerve Blocks: The use of a single injection combined with a continuous infusion peripheral nerve block for post-operative analgesia following upper limb surgery is popular. Peripheral nerve blocks provide excellent analgesia with minimal motor blockade. This facilitates early joint mobilization and physiotherapy. Peripheral nerve blocks avoid the side effects associated with opioid drugs.

A continuous peripheral nerve block technique involves the percutaneous insertion of a "catheter" directly adjacent to the peripheral nerve(s) supplying the surgical site. Dilute local anaesthetic solution are then infused via the catheter to provide potent site specific analgesia. The technique has been shown to be safe even for outpatient use with the use of disposable elastomeric pumps.

Ultrasound Visualisation

The use of high resolution real time ultrasound (US) guided nerve blocks has become popular in regional anaesthesia. The advantages of US

includes the visualisation of anatomical structures and the spread of local anaesthetic. Using US visualisation during regional and peripheral nerve block may increase success rates, decrease latency of the block, reduce volume of local anaesthetic used, reduce the time to perform the block and potentially reduce or eliminate the risk of accidental intravascular or intra-neural injection. A large number of new studies support these claims. For example a retrospective analysis of axillary blocks found that the block success rate was higher in the US group compared to the traditional group (91.6 % versus 81.9 %) [26]. The US group also employed a lower volume of local anaesthetic and had a faster onset time.

Persistent (Chronic) Pain

Persistent pain occurs when symptoms exceed in both magnitude and duration the expected clinical course of the initial injury and often results in significant disability. The transition from acute pain to chronic pain is not a finite one. Patients who develop persistent pain often have symptoms and signs during the acute phase after surgery or trauma. Table 20.10 lists the differential diagnoses for persistent pain. The list is not exhaustive.

Table 20.10 Differential diagnoses for persistent pain

Amputation; phantom or stump pain
Arthritis/arthrosis
Bony or soft tissue injury; fracture, ligament damage, instability
Cancer; tumour infiltration or chemotherapy
Compartment syndrome
Complex Regional Pain Syndrome (CRPS)
Erythromelalgia
Infection; bony, HIV, joint, skin, soft tissue
Neuropathic pain; peripheral nerve damage, central nervous or spinal lesion
Self-harm/malingering
Thoracic outlet syndrome; nerve or vascular compression
Vascular; athersclerosis, raynauds, thromboangitis obliterans

Post Amputation Pain

Amputation of the upper limb is much less common than amputation of the lower limb with trauma as the reason for over 50 % of upper limb amputations. Other causes include burns, congenital abnormality, infection, and tumours. Post amputation pain is almost universal and epitomises the overlap between acute and chronic pain. Thus following amputation, or indeed a deafferentation injury such as brachial plexus avulsion, a number of phenomena can occur:

- Stump pain
- Phantom limb sensation.
- Phantom limb pain.

It is important to recognised that all three phenomena can coexist.

Stump pain is well localised to the site of amputation. Acutely it is nociceptive and if persistent usually neuropathic. The risk of stump pain is increased by the presence of severe pre amputation pain. Phantom limb sensation is defined as any sensory perception of the missing body part with the exclusion of pain. Almost all patients undergoing amputation experience phantom sensations. Sensations vary from vague to complete sensory awareness including size, position, movement and temperature. Phantom limb pain is defined as any noxious sensory phenomenon in the missing body part. Phantom limb pain varies in its severity and in its impact on patients' lives (Table 20.11).

Epidemiology

The prevalence of phantom limb pain differs based on the location of the amputation. The prevalence of phantom pain in upper limb amputees is nearly 82 %, compared with 54 % in lower limb amputees [27]. The incidence of phantom pain is 30–80 % and is more common in the distal parts of the limb i.e. hands/fingers rather than elbow. As many as 75 % of patients develop pain within the first few days after amputation. The pain typically diminishes with time. However, 10 % of patients develop moderate or severe persistent pain. Pre-amputation pain seems to increase the risk of phantom pain

Table 20.11 Classification of severity of phantom pain

Group 1
Mild, intermittent paraesthesias that do not interfere with normal activity, work, or sleep
Group 2
Paraesthesias that are uncomfortable and annoying but do not interfere with activities or sleep
Group 3
Pain that is of sufficient intensity, frequency, or duration to be distressful;
Group 4
Nearly constant severe pain that interferes with normal activity and sleep
Some patients in Group 3 have pain that is: bearable; intermittently interferes with lifestyle; and responds to conservative treatment

after amputation and the intensity of pre-amputation and post-operative pain are predictors of intensity of chronic pain.

Management: Overview

Prevention

There is evidence to support the use of peri-operative local anaesthetic agents to reduce the severity of persistent phantom limb pain as they provide excellent post-operative pain relief [28]. However, local anaesthetic agents have not been shown to reduce the incidence of phantom limb pain *per se*. For upper limb amputation local anaesthetics can be provided in the form of continuous brachial plexus blockade or a catheter inserted under direct vision intra-operatively. The severity of long-term phantom limb pain may also be reduced by the use of intravenous peri-operative ketamine for 72 h (see section "Adjuvants") [29].

Management: Pharmacotherapy

Simple (Non Opioid) Analgesics

In the acute phase, post-amputation pain is predominantly nociceptive. Therefore simple analgesia should be started early. The use of paracetamol, NSAIDs and weak opioids such as codeine and tramadol should be considered.

Opioid Analgesics

Morphine, whether as intravenous PCA or oral sustained release tablet (MST), may be effective in reducing phantom limb pain.

Antineuropathic agents

Amitriptyline and gabapentin are effective in reducing stump and phantom limb pain. They should be prescribed as described in section "First line treatment".

Calcitonin

Calcitonin, whether by intravenous, subcutaneous or intranasal route, is effective in the treatment of acute phantom limb pain. It is not effective for persistent pain.

Ketamine

Ketamine is effective in reducing the severity of stump pain but there is no oral formulation currently available.

Management: Non-pharmacological

Non- pharmacological techniques including sensory discrimination training, mirror therapy and mental imagery of limb movement are also effective.

Neuropathic Pain

Neuropathic pain is generally more distressing than other forms of pain and can be very challenging to treat due the heterogeneity of aetiologies and underlying mechanisms. Conditions causing peripheral neuropathies such as diabetes mellitus, post-herpetic neuralgia, radicular compression, brachial plexopathy, trauma and cancer can all have neuropathic pain as a symptom. Conditions causing central neuropathic pain as a symptom include stroke, spinal cord injury and multiple sclerosis. The majority of patients with neuropathic pain following surgery or trauma will also have a nociceptive component depending on the underlying pathophysiology.

Epidemiology

The population prevalence of neuropathic pain is approximately 1 %. However, after surgery, trauma and amputation rates can be as high as 85 % for persistent pain and 10 % for severe persistent pain [7].

Diagnosis

The diagnosis of neuropathic pain in clinical practice is not easy. It can be thought of as pain occurring in an area of abnormal or absent sensation.

Symptoms

Classically patients complain of spontaneous pains (arising without detectable stimulation) and evoked pains (abnormal responses to stimulation). Pain can be continuous and steady or intermittent and paroxysmal. Continuous pain is often described as "burning", "cutting", "tingling" or "stabbing". Paroxysmal pain is often described as "shooting" or "lancing"

Signs

Physical examination should focus on the location of the pain and identifying any abnormal responses to sensory stimulus. The following phenomena may be present:

- Positive Phenomena: Allodynia, hyperalgesia, hyperpathia, autonomic dysfunction
- Negative Phenomena: Loss of light touch, pinprick, vibration and thermal sensations

The Leeds Assesment of Neuropathic symptoms and Signs (LANSS) pain scale is a useful tool to discriminate between neuropathic and nociceptive pain [30].

Management: Patient Information and Education

Information should be supplied to the patient and carers to promote self-management. Patients should be reassured that physical and occupational therapy is safe and central to effective management. Pacing of activity and goal setting should be discussed early on. The British Pain Society (www.britishpainsociety.org.uk) provides patient information.

Management: Pharmacotherapy

First Line Treatment

Options for first line drug treatment include amitriptyline, gabapentin and pregabalin [31]. Commence oral amitriptyline 10 mg at night increased slowly to an effective dose not exceeding 75 mg. Aim for a maintenance dose of at least 25 mg at night. Or commence oral gabapentin 300 mg at night increased slowly to 300 mg three times a day and then slowly to an effective dose not exceeding 3,600 mg daily. Aim for a maintenance dose of at least 600 mg three times a day. Alternatively commence oral pregabalin 75 mg at night increased to 75 mg twice daily and then slowly to an effective dose not exceeding 600 mg daily. Aim for a maintenance dose of at least 150 mg twice a day.

Whichever drug is chosen the treatment effect should be assessed at 2-weekly intervals until pain is controlled. If a drug is ineffective or not tolerated switch to another first line drug. However, amitriptyline and either gabapentin or pregabalin can be combined if one is found to be partially effective.

Second Line Treatment

Options for second line drug treatment include duloxetine and carbamazepine. Commence oral duloxetine 30 mg at night increased slowly to a maintenance dose not exceeding 120 mg. Aim for a maintenance dose of at least 60 mg at night. Alternatively commence oral carbamazepine 100 mg twice a day increased to an effective dose not exceeding 1,600 mg daily. Aim for a maintenance dose of 200 mg four times a day.

Third Line Treatment

Options for third line drug treatment include tramadol and other opioid analgesics. Commence oral modified release tramadol 50 mg twice a day increased slowly to an effective dose not exceeding 400 mg daily. Tramadol should be used with extreme caution in patients taking Selective Serotonin Reuptake Inhibitor (SSRI) antidepressants as there is potential risk of inducing serious serotonergic crisis. Other opioids can be considered and may have value if the practitioner is confident with equivalent doses and long-term management of opioids and problems arising. Consideration of these agents should also prompt a referral to a pain specialist.

Other Agents

A variety of other drugs and different delivery routes may be considered including 5 % lido-

caine plasters (versatis®), topical 8 % capsaicin patches (qutenza®) and oral ketamine.

Management: Interventional Procedures

A history suggestive or pain in the distribution of a peripheral nerve should be urgently reviewed because of the possibility of nerve damage due to surgery or trauma in which case surgical exploration may be necessary. Interventional pain therapies such as stellate ganglion block, intravenous regional anaesthesia (IVRA) and spinal cord stimulation may be indicated but only after referral to a pain specialist.

Management: Physical and Vocational Rehabilitation

Physical therapy should be delivered by therapists experienced in the management of persistent and neuropathic pain. The emphasis of treatment is on the restoration of function and normal activities through acquisition of self-management skills. Patient and carers should be actively involved in goal setting. If pain persists or treatment is ineffective referral to a pain specialist should be considered.

Complex Regional Pain Syndrome (CRPS)

Complex Regional Pain Syndrome (CRPS) is a debilitating, painful condition associated with sensory, motor, autonomic, skin and bone abnormalities [32]. The cause of CRPS is not known. There is interplay between peripheral and central pathophysiologies. The concept of sympathetic dysfunction as the predominant problem is now obsolete (Table 20.12). CRPS can be divided into two types based on the absence (type 1) or presence (type 2) of a lesion to a major nerve. This distinction has no relevance for management but may have medico-legal importance.

Epidemiology

The available evidence suggests that transient CRPS is common after limb fractures and surgery (up to 25%) [33]. The pain improves in most cases after a few months. CRPS commonly arises after injury although there is no relationship to the severity of trauma. In 9 % of cases there is no history of trauma. The European incidence of CRPS is 1 in 25,000 with a prevalence of 1 in 1,500. CRPS usually affects one limb but affects the contra-lateral limb in 7 % of cases [34]. The onset of symptoms for the majority occurs within 1 month of injury or immobilisation. Approximately 15 % of sufferers will have persistent pain and functional impairment at 2 years [35]. A transient episode of CRPS may result in long term disability due to functional and structural changes.

Table 20.12 Previous names for CRPS

Algodystrophy
Algoneurodystrophy
Causalgia
Fracture disease
Reflex neurovascular dystrophy
Reflex sympathetic dystrophy
Shoulder-hand syndrome
Sudeck's atrophy

Diagnosis

CRPS is a diagnosis of exclusion. The Budapest criteria (Table 20.13) [36] were developed to differentiate CRPS from the other causes of pain seen in a pain clinic. The Atkins criteria (Table 20.14) [37] were developed for a surgical setting. They both provide similar results in the diagnosis of CRPS.

Common alternative causes of pain include infection, poor fracture fixation, instability, arthritis, arthrosis and nerve entrapment or nerve damage (see Table 20.10).

Plaster tightness and/or disproportionate pain whilst in plaster may be early warning signs for CRPS or even a compartment syndrome. Sensory and motor neglect ("my hand feels like is does not belong to me" or "I cannot move my hand the way I want to") are features of CRPS. The routine use of technetium-99 three-phase bone scans is no longer recommended [38].

Management: Overview

CRPS should be managed with an integrated interdisciplinary approach [39]. For patients with

Table 20.13 Budapest (modified IASP) clinical diagnostic criteria for CRPS[6]

Continuing pain which is disproportionate to any inciting event			
≥**1** symptom of **3 categories** plus ≥**1** sign of **2 categories**			
No other diagnosis that better explains the symptoms and signs			
Category 1	**Category 2**	**Category 3**	**Category 4**
Symptoms:	**Symptoms**:	**Symptoms**:	**Symptoms**:
Spontaneous pain	Temperature asymmetry	Swelling	Motor weakness
Mechanical hyperalgesia	Skin colour changes	Hyperhydrosis	Tremor/dystonia
Thermal hyperalgesia		Hypohydrosis	Coordination deficit
Deep somatic hyperalgesia			Nail/hair changes
			Skin atrophy
			Joint stiffness
			Soft tissue changes
Signs:	**Signs**:	**Signs**:	**Signs**:
Pinprick hyperalgesia	Vasodilatation	Swelling	Motor weakness
Allodynia	Vasoconstriction	Hyperhydrosis	Tremor/dystonia
	Temperature asymmetry	Hypohydrosis	Coordination deficit
	Skin colour changes		Nail/hair changes
			Skin atrophy
			Joint stiffness
			Soft tissue changes

CRPS pain, loss of function and psychological distress predominate. There are four "pillars" of care (Fig. 20.1).

Early referral to a specialist centre offering multidisciplinary pain management is recommended.

Management: Patient Information and Education

Information should be supplied to the patient and carers to promote self-management.

Patients should be reassured that pain will completely or partially resolve in 85 % of cases. Patients should be reassured that physical and occupational therapy is safe and central to effective management. Pacing of activity and goal setting should be discussed early.

Management: Pharmacotherapy

Simple* (*non opioid*) *analgesia: Simple analgesia should be started early. The use of paracetamol, NSAIDs and weak opioids such as codeine and tramadol should be considered. Whilst these drugs to do not treat the specific pain of CRPS they may treat ongoing nociceptive pain and assist with physical therapy.

Table 20.14 Atkins clinical diagnostic criteria for CRPS

Neuropathic pain
Non dermatomal; burning; without cause but with associated allodynia and hyperpathia
Vasomotor instability and abnormalities of sweating
Extremity red and dry; cool, blue and clammy; increase in temperature sensitivity; abnormal temperature difference between the limbs
Swelling
Loss of joint mobility with associated joint and soft tissue contracture
Skin thinning; hair and nail dystrophy

Opioid analgesia: Opioids can be considered and may have value if the practitioner is confident with equivalent doses and long-term management of opioids and problems arising. Consideration of these agents should also prompt a referral to a pain specialist.

Anti-neuropathic agents: These agents should be prescribed as described in section "Management: Non-pharmacological".

Other agents: Intravenous pamidronate 60 mg may be considered in patients with CRPS of less than 6 months duration as a one off treatment. If pain persists or treatment is ineffective referral to a pain specialist should be considered.

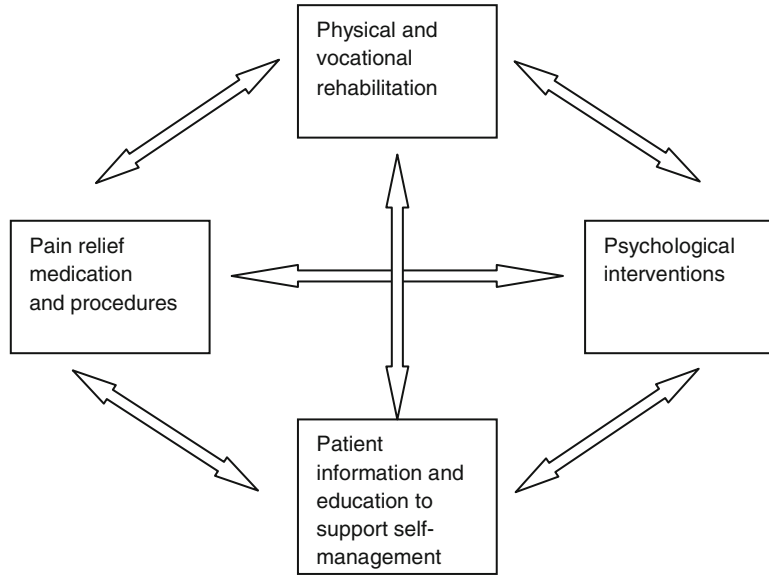


Fig. 20.1 The four pillars of care

Management: Interventional Procedures

Interventional pain therapies such as stellate ganglion block and intravenous regional anaesthesia (IVRA) are not recommended for the management of CRPS. Spinal cord stimulation may be indicated for patients who have not responded to integrated management after referral to a pain specialist.

Surgery: Amputation (see section "Post amputation pain") should only be considered in cases of untreatable infection of the limb. Early surgery should be avoided on a CRPS affected limb and deferred for at least 1 year after the active process has resolved if unavoidable. Surgery may be indicated in some cases of CRPS type 2 if an identifiable nerve lesion is identified. There is no evidence to support that any one anaesthetic technique is superior in the prevention of CRPS recurrence.

Management: Physical and Vocational Rehabilitation

Therapy should be delivered by therapists experienced in the management of persistent and neuropathic pain. The emphasis of treatment is on the restoration of function and normal activities through acquisition of self-management skills. Patient and carers should be actively involved in goal setting. The program may include: pacing and relaxation strategies, self administered desensitisation strategies to tactile and thermal stimuli, mirror therapy and management of CRPS related dystonia.

Management: Psychological Intervention

Psychological intervention is based on an individualised assessment of mood (anxiety or depression) expectations and behaviours (avoidance, catastrophising). Management usually follows the principles of cognitive behavioural therapy (CBT) on an intermittent one-on-one basis or a more intensive residential pain management program (PMP) that some specialist centres offer.

References

1. Merskey H, Bogduk N. Classification of chronic pain, IASP Task Force on Taxonomy. Seattle: IASP Press; 1994.
2. Loeser JD, Treede RD. The Kyoto protocol of IASP basic pain terminology. Pain. 2008;137(3):473–7.
3. Hayes C, Browne S, Lantry G, et al. Neuropathic pain in the acute pain service: a prospective study. Acute Pain. 2002;4:45–8.
4. Cousins MJ, Brennan F, Carr DB. Pain relief: a universal human right. Pain. 2004;112(1–2):1–4.
5. Kehlet H, Dahl JB. Anaesthesia, surgery, and challenges in postoperative recovery. Lancet. 2003; 362(9399):1921–8.

6. Dolin SJ, Cashman JN. Effectiveness of acute postoperative pain management: I. Evidence from published data. Br J Anaesth. 2002;89:409–23.
7. Kehlet H, Jensen TS, Woolf CJ. Persistent postsurgical pain: risk factors and prevention. Lancet. 2006; 367(9522):1618–25.
8. Macrae WA. Chronic post-surgical pain: 10 years on. Br J Anaesth. 2008;101(1):77–86.
9. Kissin I. Preemptive analgesia: terminology and clinical relevance. Anesth Analg. 1994;79(4):809–10.
10. Katz J, McCartney CJ. Current status of preemptive analgesia. Curr Opin Anaesthesiol. 2002; 15(4):435–41.
11. Katz J, Clarke H. Preventive analgesia and beyond: current status, evidence, and future directions. In: Macintyre PE, Walker SM, Rowbotham DJ, editors. Clinical pain management: acute pain. 2nd ed. London: Hodder Arnold; 2008.
12. Scott DA, McDonald WM. Assessment, measurement and history. In: Macintyre PE, Rowbotham D, Walker S, editors. Textbook of clinical pain management. 2nd ed. Acute pain. London: Hodder Arnold; 2008.
13. Murphy D, McDonald A, Power C, Unwin A, MacSullivan R. Measurement of pain: a comparison of the visual analogue with a nonvisual analogue scale. Clin J Pain. 1988;3:197–9.
14. Australian and New Zealand College of Anaesthetists and Faculty of Pain medicine: Scientific evidence, 2nd ed. Melbourne: Australian and New Zealand College of Anaesthetists; 2005.
15. McNicol ED, Tzortzopoulou A, Cepeda MS, Francia MBD, Farhat T, Schumann R. Single-dose intravenous paracetamol or propacetamol for prevention or treatment of postoperative pain: a systematic review and meta-analysis. Br J Anaesth. 2011;106(6): 764–75.
16. Dsida R, Cote C. Nonsteroidal anti-inflammatory drugs and hemorrage following tonsillectomy: do we have the data? Anaesthesiology. 2004;100:749–51.
17. Kearney PM, Baigent C, Godwin J, et al. Do selective cyclo-oxygenase-2 inhibitors and traditional nonsteroidal inflammatory drugs increase the risk of atherothrombosis? Meta-analysis of randomised trials. BMJ. 2006;332:1302–3.
18. Nussmier NA, Whelton AA, Brown MT, et al. Complications of the COX-2 inhibitors parecoxib and valecoxib after cardiac surgery. N Engl J Med. 2005;352:1081–91.
19. Apfel CC, Greim CA, Haubitz I, Usadel J, Sefrin P, Roewer N. A risk score to predict the probability of postoperative vomiting in adults. Acta Anaesthesiol Scand. 1998;42:495–501.
20. Ahmed AB, Hobbs GJ, Gurran JP. How to study postoperative nausea and vomiting. Acta Anaesthesiol Scand. 2002;46:921–8.
21. De Beer Jde V, Winemaker MJ, Donnelly GAE, et al. Efficacy and safety of controlled-release oxycodone and standard therapies for postoperative pain after knee or hip replacement. Can J Surg. 2005;48: 277–83.
22. Hudcova JC, McNicholl E, Quah C, et al. Patient controlled analgesia versus conventional opioid analgesia for postoperative pain. Cochrane Database Syst Rev. 2006;(4):CD003348.
23. Seib RK, Paul JE. Preoperative gabapentin for postoperative analgesia: a meta-analysis. Can J Anaesth. 2006;53:461–9.
24. Elia N, Tramer MR. Ketamine and postoperative pain-a quantitative systematic review of randomised trials. Pain. 2005;113:61–70.
25. Fredrickson MJ, Kilfoyle DH. Neurological complication analysis of 1000 ultrasound guided peripheral nerve blocks for elective orthopaedic surgery: a prospective study. Anaesthesia. 2009;64:836–44.
26. Lo N, Brull R, Perlas A, et al. Evolution of ultrasound guided axillary brachial blockade: retrospective analysis of 662 blocks. Can J Anaesth. 2008;55(7): 408–13.
27. Kooijman CM, Dijkstra PU, Geertzen JH, et al. Phantom pain and phantom sensations in upper limb amputees: an epidemiological study. Pain. 2000; 87(1):33–41.
28. Lambert AW, Dashfield AK, Cosgrove C, et al. Randomized prospective study comparing preoperative epidural and intraoperative perineural analgesia for the prevention of postoperative stump and phantom limb pain following major amputation. Reg Anesth Pain Med. 2001;26(4):316–21.
29. Dertwinkel R, Heinrichs C, Senne I, et al. Prevention of severe phantom limb pain by perioperative administration of ketamine – an observational study. Acute Pain. 2002;4(1):9–13.
30. Bennett M. The LANSS Pain Scale: the Leeds assessment of neuropathic symptoms and signs. Pain. 2001;92(1–2):147–57.
31. NICE. The pharmacological management of neuropathic pain in adults in non-specialist settings. 2010. www.nice.org.uk/cg96.
32. Baron R, Fields HL, Jänig W, Kitt C, Levine JD. National Institutes of Health Workshop: reflex sympathetic dystrophy/complex regional pain syndromes-state-of-the-science. Anesth Analg. 2002;95:1812–6.
33. Dijkstra PU, Groothoff JW, ten Duis HJ, Geertzen JH. Incidence of complex regional pain syndrome type I after fractures of the distal radius. Eur J Pain. 2003;7(5):457–62.
34. Veldman PH, Reynen HM, Arntz IE, Goris RJ. Signs and symptoms of reflex sympathetic dystrophy: prospective study of 829 patients. Lancet. 1993; 342(8878):1012–6.
35. Schasfoort FC, Bussman JG, Stam HJ. Impairments and activity limitations in subjects with chronic upper limb complex regional pain syndrome type I. Arch Phys Med Rehabil. 2004;85(4):557–66.
36. Harden RN, Bruehl S, Stanton-Hicks M, Wilson PR. Proposed new diagnostic criteria for complex regional pain syndrome. Pain Med. 2007;8(4): 326–31.
37. Thomson McBride AR, Barnett AJ, Livingstone JA, Atkins RM. Complex regional pain syndrome (type

1): a comparison of 2 diagnostic criteria methods. Clin J Pain. 2008;24(7):637–40.
38. Atkins RM, Tindale W, Bickerstaff D, Kanis JA. Quantitative bone scintigraphy in reflex sympathetic dystrophy. Br J Rheumatol. 1993;32(1):41–5.
39. Bruehl S, Harden RN, Galer BS, et al. External validation of IASP diagnostic criteria for complex regional pain syndrome and proposed research diagnostic criteria. International Association for the Study of Pain. Pain. 1999;81(1–2):147–54.

Index

A
Abductor pollicis longus (APL), 227
Acute DRUJ instability
  aetiology, 382
  anatomy
    ECU, 380
    fibrocartilaginous lip, 379–380
    IOM, 380
    pronator quadratus, 380
    TFCC, 380–381
    ulnar styloid, 380, 381
    vascular supply, 381
  biomechanics, 381–382
  imaging
    arthroscopy, TFCC, 384–385
    CT scan, volar subluxation, 384
    MRI, TFCC tear, 384
    plain radiographs, 383
  physical examination, 383
  presentation, 382–383
  treatment options
    TFCC repair, 385–386
    ulnar styloid fracture repair, 386–387
Acute pain
  adjuvants
    gabapentin and pregabalin, 447
    ketamine, 447
  to chronic pain, progression, 438–439
  diagnosis, 442
  local anaesthetic blockade
    intra-articular joint injection, 448
    IVRA, 448
    local infiltration, 447–448
    peripheral nerve blocks, 448
    plexus blocks, 448
  non opioid analgesics
    COX-2 inhibitors, 444
    NSAIDS, 443–444
  opioid analgesics
    intravenous routes, 446–447
    oral administration, 445–446
    pharmacology, 444–445
    side effects, 445
  patient information and education, 442
  pharmacotherapy, 442
  ultrasound visualisation, 448–449
Acute scaphoid fracture
  anatomy, 250
  assessment of union, 253–254
  clinical examination
    inspection, 251
    movement, 251
    palpation, 251
    tests, 251
  description, 249
  diagnosis, 250
  history, 251
  imaging, 251–252
  mechanism, 249–250
  non-operative management, 252–253
  operative management, 254
  outcome, 256
  proximal pole fractures, 254–255, 257
  soft tissue injuries, 257
  surgical techniques, 255–256
  vascularity/displacement, 257
Advanced Trauma Life Support (ATLS), 90, 393
Amputation
  avulsed hand, 96–98
  complex upper limb trauma, 395, 397
  distal phalanx, 171, 173
  of fingertip, 168–169
Anaesthesia/analgesia. *See also* Analgesia
  distal phalanx, 169
  management, 90
Analgesia
  non opioid, 450, 453
    COX-2 inhibitors, 444
    NSAIDS, 443–444
  opioid, 450, 453
    intravenous administration, 446–447
    oral, 445–446
    oral administration, 445–446
    pharmacology, 444–445
    side effects, 445
  pre-emptive and preventive, 439–440

I.A. Trail, A.N.M. Fleming (eds.), *Disorders of the Hand: Volume 1: Hand Injuries*,
DOI 10.1007/978-1-4471-6554-5, © Springer-Verlag London 2015

'Anatomic' plates, 279
Aneurysms, 15
Angiography, 12
Angular stability
  advantages, 279
  non-locking plates, 278
  palmar surface, 273
  volar plate, 271
Arterial system
  anatomical variations, 3–4
  axillary artery, 2
  brachial, 2
  deep palmar arch, 3
  digital, 3
  hand and wrist, 3
  interosseous, 2
  radial, 2
  superficial palmar arch, 3
  thumb, 3
  ulnar, 2
  upper limbs, 1
Arthritis
  septic, 423–424
  tuberculous, 431
Articular fractures
  extra, 270–272
  intra, 272–274
Associated injuries
  interventional approach, 261
  wrist fractures
    arthroscopic study, 277
    median nerve dysfunction, 278
    radiographic clues, 277
    scapholunate (SL) ligament tears, 277–278
Avascular necrosis, 238, 239, 244
Avulsion
  bony, 182
  head metacarpal fractures, 223–224
  metacarpophalageal joint fractures
    base of proximal phalanx, 212
    description, 211
    fixation, dorsal approach, 213, 214
    flexion, 212
    metacarpal head, 212–213
    non-union, 211
    palmar approach, 212, 213
    palmar plate, 211
  nail (*see* Nail avulsions)
  of nail, 165
  volar rim fractures, 280

B
Bands of Fontana, 31
Bennett's fracture, 229, 233
Bilateral lateral VY flaps
  advantages, 137
  indications, 136
  limitations, 137
  outcomes, 137
  technique and refinements, 136
Biopsychosocial model, 440
Bite injuries
  animal bites, 427
  human bites
    bacterial species, 426
    fight bites, 425–426
    infective complications, 426
    vaccination or anti-viral treatment, 426–427
Blood disorders, 19
Blood vessels
  arterial system, 1–4
  compartment syndrome, 10–13
  conditions, acute occlusive, 12–19
  injuries
    blood disorders, 19
    causes, 7–8
    connective tissue, 18–19
    renal insufficiency, 19
    sepsis, 19
    vasospastic (*see* Vasospastic injuries)
  lymphatic, 4–5
  physiology, 5
  trauma
    blunt, 8–9
    management, 7
    penetration, 7–8
    surgical treatment, 9–10
    vascular, 7
  vascular disorders, 1
  venous, 4
Blunt trauma, 8–9
Bone loss
  bone forearm, creation, 403–404
  in fracture, 273
  metacarpal fractures, 231–232
Boutonnière injury
  Carducci's, Elson's and Boyes' tests, 78
  description, 77
  middle phalanx, 188
  PIP joint, 77–78, 179–180
  shape, 78
  treatment, 78
'Boxer's fracture, 224
Boye's test, 70, 74, 75, 78
Bursal infections, 421

C
Cancer reconstruction
  adjuvant therapy, 96
  MDT, 95
  radiotherapy, 95–96
Capitate fractures
  imaging, 245
  incidence, 245
  mechanism, 245
  treatment, 245–246

Capsular injuries
MCPJ of thumb
clinical features and diagnosis, 338
complications, 339
epidemiology, 337–338
outcome and lit review, 338–339
surgical treatment, 338
treatment options, 338
PIPJ
classification, 328
clinical features and diagnosis, 328
complications, 330–335
epidemiology and pathomechanics, 327–328
outcomes, 335
type III injuries, 329, 331, 332
type II injuries, 328–330
type I injuries, 328, 329
type IV injuries, 329–330, 333–335
Carducci's test, 70, 73, 74, 78
Carpal fracture/dislocation
aetiology
intrinsic and extrinsic components, 368
prevalence, 368
radio-scapholunate ligament, 368–369
reverse perilunate instability, 368
stages of lunate instability, 368
arthroscopy, 371–372
characteristics, 237
classification
of carpal dislocations, 372
CID, CIND and CIC, 373
dorsal subluxation, 373
static and dynamic instability, 373
total radiocarpal dislocation, 372, 373
transradial perilunar dislocation, 372, 373
ulnar translocation, 373
CT and MRI scans, 370–371
description, 237
perilunar dislocation, 369
scapholunate gap, 370, 371
scapholunate interosseous ligament, 369–371
surgical techniques and rehabilitation
closed reduction, 374
immobilizing wrist, 373–374
isolated lunotriquetral dissociation, 377
isolated scapholunate dissociation, 376–377
open reduction and repair technique, 374–376
transcaphoid perilunar dislocation, 369
Carpal instability complex (CIC), 373
Carpal instability dissociative (CID), 373
Carpal instability non-dissociative (CIND), 373
Carpometacarpal joint (CMCJ)
of fingers
bony architecture, 357–358
clinical features and diagnosis, 358–360
complications, 360
dorsal dislocations, 358–360
epidemiology, 358
extrinsic joint support, 358
joint capsule, 358
outcome and lit review, 359–360
pathology, 358
surgical treatment, 359
treatment options, 359
fracture-dislocations
articular incongruity, 229
base fractures, 227
CT scan, 222
intra-articular base fractures, 229
metacarpal shaft fractures, 228
post-traumatic osteoarthritis, 231
transcarpal, 227
of thumb
bony architecture, 361
clinical features and diagnosis, 361–362
complications, 363
dorsal dislocations, 361–363
epidemiology, 361
extrinsic joint support, 361
joint capsule, 361
outcome and lit review, 363
surgical treatment, 362, 363
treatment options, 362
Children
assessment, 286, 287
complex and mutilating hand injuries, 305–306
distal phalanx injury, 289
epidemiology/aetiology, 285–286
fingertip pulp injury, 289
fractures
carpal, 294
distal radius and ulna fractures, 294–297
metacarpal, 293–294
phalangeal, 291–293
Salter Harris classification, 289, 290
joint injury, 297–298
management, 286–288
nail bed injury, 288–289
nerve injury, 301–304
tendon injury
closed, 300
diagnosis, 299
extensor, 301
flexor, 300–301
jersey finger, 299
management, 299
vascular injury, 304–305
Chinese flap
Allen's test, 108
extensor tendon reconstruction, 108
PIPJ's, 109–110
skin marking, 106–108
volar surface, fingers and distal palm, 109, 111
Choke vessels, 105
Chromatolysis, 301
Chronic pain
CRPS, 452–454
neuropathic pain, 450–452
post amputation pain, 449–450
CMCJ. *See* Carpometacarpal joint (CMCJ)

Coaptation neurosynthesis, 31
Collateral ligament tear
  clinical features, diagnosis and treatment, 317–318
  PIPJ
    clinical features and diagnosis, 324
    complications, 326
    conclusions/personal view, 326–327
    epidemiology and pathomechanics, 324, 325
    outcome and lit review, 326
    surgical treatment, 325, 326
    treatment options, 324
  radial, MCPJ of thumb
    clinical features and diagnosis, 348
    complications, 349
    epidemiology, 347
    outcome and lit review, 349
    pathology, 348
    surgical treatment, 348–349
    treatment options, 348
  thumb (acute) instability, 317–318
  ulnar, MCPJ of thumb
    clinical features and diagnosis, 343–345
    complications, 346
    epidemiology, 342
    pathology, 342–343
    surgical treatment, 345–346
    treatment options, 345
Columnar theory, 260–261, 263, 273
Comminuted fractures, 209–210
Comparative studies, soft tissue cover
  fingertip reconstruction, 145–147
  Microvascular Toenail Transfer, 142
  thumb reconstruction, 154
  VY advancement flaps, 135
Compartment syndrome
  conditions, 10
  consequences, 11
  fasciotomies
    dorsal, 12, 13
    forearm and palmar, 12
  incisions, 12
  pain, 12
  relationship, LBF and arteriovenous gradient, 10–11
  swollen forearm after crush injury, 12
  symptoms, 10
Complex regional pain syndrome (CRPS)
  anti-neuropathic agents, 453
  diagnosis, 452
  epidemiology, 452
  interventional procedures, 454
  opioid analgesia, 453
  patient information and education, 453
  physical and vocational rehabilitation, 454
  psychological intervention, 454
  simple (non opioid) analgesia, 453
  surgery, 454
Complex upper limb trauma
  assessment
    amputation, 395, 397
    hand transplantation, 395
    MESS, 397
    of patient, 393
    repair of functional units, 406
  debridement
    contaminants, removal, 398
    degloved skin edges, assessment, 398–399
    fasciotomy, 400
    'on arrival block,' 397
    polytrauma patients, outcome, 397
    radical, 398
    serial or staged, 399–400
    skeletonization of longitudinal structures, 398
    use of tourniquet, 398
    wound excision, 398
  microsurgical techniques, 391–393
  outcome, 411–412
  pain relief (concept of 'on arrival block'), 393–394
  reconstruction
    revascularization considerations, 400–401
    skeletal fixation considerations, 401–406
  rehabilitation, 410
  repair of functional units, 406
  secondary reconstruction, 411, 412
  soft tissue cover, 409–410
  total primary *vs.* staged reconstruction, 407–408
Composite grafts, non-flap, 133–134
Condylar fractures
  articular surface, 206
  dorsal approach, 207
  K-wires, 206–207
  lateral approach, 207–208
  open reduction and internal fixation, 206, 207
  timing of surgery, 208
  un-displaced position and non-operative treatment, 206
Condyle, 205, 207–208, 312–313
Cross finger flap
  advantages, 138
  indications, 138
  limitations, 138
  outcomes, 139
  technique and refinements, 138, 140
Crush injury upper limb, 393–394

D
DASH. *See* Disabilities of Arm, Shoulder & Hand (DASH)
Deep space infections, 421, 433
Denervation
  injury, axonotmesis, 36
  Schwann cells, 27
  spontaneous spike potential, nerve transfer, 41
Diaphyseal fractures
  displaced
    closed K-wire fixation, 198, 199
    fixation, 198
    precise restoration of the anatomy, 198
    X-rays, 198

K-wires, 199–200
lag screws, 201
plates, 201
undisplaced
buddy-taping, 197
custom-made, thermoplastic splint, 197, 198
intrinsic-plus position with MCP, 197
Digital (distal) dislocations
collateral ligament injury, 317–318
DIPJ of fingers, 312–313
IPJ of thumb, 313
joint dislocation, 318–319
mallet finger, 313–315
mallet thumb, 315–317
Disabilities of Arm, Shoulder & Hand (DASH), 156, 226, 265
Distal interphalangeal joint (DIPJ)
dislocation, 168
of fingers
bony architecture, 312
joint capsule, 312–313
fusion, 173
Distal phalanx
anatomy, 163–164
avulsion of nail, 165
fingertip, anatomy, 163
fractures
amputations of fingertip, 168–169
DIP joint dislocation, 168
intra-articular, 166–167
Jersey finger or FDP tendon avulsions, 167, 168
of shaft, 166
of tuft, 165
injury, 289
investigations, 164
presentation, 164
subungual haematoma, 164–165
surgical techniques and rehabilitation
amputation, 171
anaesthesia/analgesia, 169
bony mallet finger fixation techniques, 170
fixation of the nail, 170, 171
fixation techniques, 170
Jersey finger, 171
nail removal, 170
outcomes, 171–172
replacing the nail, 170
subungual haematoma, 169
Tourniquet, 169
treatment, complications
absence of nail, 172
amputation, 173
DIP joint fusion, 173
eponychial deformities, 172
fingertip deformities, 172
hyponychial deformities, 172–173
nail spikes and cysts, 172
outcomes, 174
paronychia, 173
split nail, 172
technique, 173
Distal radioulnar joint (DRUJ)
carpal malalignment, 265
component skeletal structures, 263
disruption, tilt and translation, 281
extra articular fractures, 270–272
injury
distal ulna, 263
forearm rotation, 263
skeletal structures, 263
instability (*see* Acute DRUJ instability)
ligament injury, 298
ligaments, 270
malalignment or ligament injury, 265
plain x-ray, 276
radial component, 261
radial shortening, 270
radial translation, 266, 271
sigmoid notch, 261
stability, 270
and TFC detachment, 275
ulna, 263
weight bearing fulcrum, 274
Distal radius fractures
absorption characteristics, 280
acceptable reduction, 265
adaptive carpal instability, 265
anatomic plates, 279
angular stability, 279
articular (*see* Articular fractures)
bone mineral density, 265
carpal malalignment, 264–265
characterizes, 259
classification
AO comprehensive classification, 263
distal ulnar fractures, 275
Frykman's classification, 260, 274
Melone, 262
Universal System, 263–264
external fixators, 279
fixed angle locking plates, 279
fixed-angle stability, 279
imaging, 266–270
injury
articular fragmentation, 262
articular surface, 261–262
bending' fracture, 262
classification of Melone, 262
3-column concept, 260–261
CT analysis, 261
dye-punch injury, 261, 262
extrinsic ligament attachments, 262
Frykman's classification, 260
intermediate column, 260, 261
load pattern, 261
properties of bone and ligament, 262
types, 262
volar extrinsic ligaments, 262

Distal radius fractures (*cont.*)
internal fixation implants, 279, 280
management, 260
overpenetration of screw tips, 279, 280
palmar surface, 280
Radial styloid fractures, 280
radiographic parameters, 264
scoring systems, 265
and ulna fractures
forearm fractures, 294–295
remodelling, 295, 296
unicortical or bicortical fractures, 295
volar rim avulsion fractures, 280
Distant flaps, 140
Distant pedicled flaps
cross-arm flaps, 116
inferiorly based abdominal flaps
intra-operative technique, 113–114
large defect, dorsum, 112–113, 115
SIEA, 112
pedicled groin flap, 114–120
Donor nerves, 38
Dorsal fracture subluxation
dynamic external fixator, 186–187
manipulation and K wiring, 186
open reduction and internal fixation, 187
radiograph, 182–183
Dorsal metacarpal artery perforator flaps (Quaba flap), 144–145
"Dropped knuckle," 219
DRUJ. *See* Distal radioulnar joint (DRUJ)

E
Elson's test, 73–75, 78
Embolism
acute onset ischaemia, 14
anticoagulation, 14
conditions, 14
distal, 14
evaluation, 14
intra arterial injection injuries, 14–15
management, 15
upper limb, 14
Escharotomy, 92
Extensor carpi ulnaris (ECU), 380
Extensor pollicis brevis (EPB), 227
Extensor pollicis longus (EPL) tendon, 227, 313
Extensor tendon injuries
anatomy
blood supply, 69
brachioradialis and carpi radialis longus, 69
compartments, retinaculum, 68
Digitorum Communis, 68
excursions, 69
finger, 69
flexor system, 68
gliding surfaces, 69
oblique retinacular ligament, 68–69
proximal and middle phalanx, 68
superficial and deep group, 67
wrist, 67–68
carpi radialis longus and brevis, 72, 73
classification, 79
closed
mallet finger, 76–77
rupture, central slip (*see* Boutonnière injury)
controlled active rehabilitation, 85
Digitorum Communis, 70–71
fingers extention, 71, 72
indicis and digiti minimi, 71
long-term outcomes, 84–85
Miller's rating, 84
mobilisation programmes, 86
observation, 69–70
open injuries
open zone 1 (distal interphalangeal joint), 81
open zone 2 (middle phalanx), 82
open zone 3 (proximal interphalangeal joint), 82–83
open zone 4 (proximal phalanx), 83
open zone 5 (metacarpophalangeal joint), 83
open zone 6 (metacarpal), 86
open zone 7 (wrist), 84
open zone 8 (distal forearm), 84
Pollicis Brevis test, 70, 71
rehabilitation, 84, 85
sagittal band rupture, 78–79
snuffbox, 70
surgical treatment, open injuries, 79–81
tests
Boyes', 74, 75
Carducci's, 71, 72
Carpi Ulnaris, 73, 74
Elson's, 73–75
Pollicis Longus, 70, 71
Extensor tendon repair
Braided and non-absorbable sutures, 80
flexor, 79–80
general anaesthesia, 79
injury, 301
Kessler core suture, 80
mobilisation technique, 80
prophylactic antibiotics, 79
schemes, open injuries, 80–81
surgical treatment, 79
technique, 79
tourniquet and loupe magnification, 79
Extra articular fractures
carpal deformity, 270
description, 270
and intra articular fractures
closed K-wiring, 185
condylar fracture, 181
K-wires, 183–184
open K-wiring, 185
open reduction and internal fixation, 185–186
unicondylar fracture of head, 181–182

loss of palmar tilt, 270
non-surgical treatments, 271
percutaneous K-wires, 271
Pulsed low-intensity ultrasound, 271
radial shortening, 270
radial translation, 270, 271
shortening, 270
stable reduction, 270
ulnar sided wrist pain, 270
undisplaced and stable injuries, 271

F
Fascicular neurosynthesis, 31
Fascio-cutaneous flaps
distally based
dorsal ulnar, 111–113
PIIF, 109–112
radial forearm/Chinese flap, 108–111
upper and lower limb, 106
perforator, 105–107
proximally base, 105, 106
Felon, 418–419
Finger instability
CMCJ, 357–360
DIPJ, 312–313
mallet finger, 313–315
MCPJ, 349–357
Fingertip pulp injury, 289
First dorsal metacarpal artery flap (FDMCA/Foucher flap)
advantages, 151
indications, 150
limitations, 150
outcomes, 151
technique and refinements, 150, 151
First webspace free flap/dorsalis pedis free flap
advantages, 159
indications, 158
limitations, 159
outcomes, 159
technique and refinements, 158–159
Fixation techniques
bony mallet finger, 170
distal phalanx, 170
of nail, 170, 171
Flap(s)
algorithm, soft tissue defects, 147
fingertips (index to little)
dorsal middle phalanx defects, 142
dorsal proximal phalanx defects, 142, 144–145
heterodigital flaps, 138–140
homodigital flaps (*see* Homodigital flaps)
Ishikawa's classification, 134, 135
nailbed defects, 140–142
outcomes, 145–147
surgical techniques, 134
volar finger defects, 145
functional considerations, 134
non-flap
composite grafts, 133–134
healing by secondary intention, 133
skin grafts, 133
sensory considerations, 134
small free (*see* Small free flaps)
thumb (*see* Thumb)
Flap cover
primary radical debridement, 409
secondary reconstruction procedures, 409
skin graft, 409–410
Flap reconstruction
angiosome, 105
axial pattern flap, 105
blood supply, 102
choke vessels, 105
design flaps, 105
distant pedicled flaps, 112–118
fascio-cutaneous flaps, 105
hallux transfer, 128–130
local flaps, 105–112
micro vascular free flap transfers, 118–128
myocutaneous flaps, 105
pedicled abdominal flaps, 104
planning and evolution, 127, 129–130
random pattern flaps, 102, 104
robust healing, 102
skin ref, 105
Taylor's anatomical studies, 105
Tintle pedicled flap cases, 127
upper limb defect, 105
Flexor Digitorum Profundus (FDP) tendon, 167, 313
Flexor pollicis longus (FPL) repairs, 52–53
Flexor tendon
adhesions effects, 45
anatomy
pulley system, sheaths, 47
Verdan classification, 47–48
zones 1 and 2, 48–49
classification, injuries, 60–61
closed rupture, 45
division, 47
finger, 45
healing process, 45
injury, 300–301
mobilisation, 46
post-operative management, 54–58
primary repair (*see* Primary flexor tendon repair)
proximity, 46–47
radiological investigations, 45
repair
methods, 51–53
primary (*see* Primary flexor tendon repair)
techniques, 49–50
timing of, 49
zone 2 injuries, 58–59
scarring, 46

Flexor tendon (*cont.*)
severe injuries
'black-box,' zone 2, 60
FDP tendons, 59, 60
finger and palmar trauma, 59
finger function, 59
surgical techniques, 59
zone 2C/2D, 59–60
surgery and rehabilitation, 45, 61
tendon sheath (*see* Tendon(s))
Flexor tendon adhesion
post-operative management
adequate analgesia, 57
dorsal blocking splint, 54
dynamic extension splinting, 57
early and controlled active motion, 55, 56
excellent finger mobility, 57–58
fingers extension, 55
formation of adhesions, 54
inter-phalangeal joints, 57
Kleinert traction, 55
MCP joints, 56, 57
mobilisation, 54–55
passive extension exercises, 57
Phalen test, 56
PIP joints, 57
protective palmar bars, 57
proximal phalanx, 57
rehabilitation method, 57
repairs, fingers and plam, 56–57
rupture patients, 55–56
tendon healing, 54
ultrasound, 57
severe injuries, 59
Flexor tendon rehabilitation
post-operative management, 54, 56, 57
and surgery, 61
zone 2 'black-box,' 60
Flexor tendon rupture, 54, 55, 57, 64
Flexor tenosynovitis
bursal infections, 421
clinical, 419–420
definition, 419
outcome, 421
surgery, 420
Fracture(s)
articular (*see* Articular fractures)
carpal, 294
dislocation, 190–192, 227–229
distal phalanx
amputations of fingertip, 168–169
DIP joint dislocation, 168
intra-articular, 166–167
Jersey finger or FDP Tendon Avulsions, 167
of shaft, 166
of tuft, 165
distal radius (*see* Distal radius fractures)
distal ulna, 274–277
DRUJ (*see* Distal radioulnar joint (DRUJ))
extra articular, 270–272
insufficiency, 278–279
malunion, 291
metacarpo phalageal joint (*see* Metacarpophalangeal joint (MCPJ))
phalangeal, 291–293
proximal phalanx (*see* Proximal phalanx)
Salter Harris classification, 289, 290
Fractures, carpal bones
anatomy, 238
capitate, 245–246
greater-arc injuries, 238
hamate, 241–244
Kienböck's, 237
lunate, 238–239
mechanism, 237–238
non-scaphoid, 237
pisiform, 241
trapezium, 246–247
trapezoid, 247
triquetrum, 239–240
Free flaps, toe pulp and variations
advantages, 153
indications, 151
limitations, 153
outcomes, 153
technique and refinements, 151–153
Free medial plantar flap
advantages, 158
indications, 157
limitations, 158
outcomes, 158
technique and refinements, 157–158
Free posterior interosseus flap
advantages, 157
indications, 156
limitations, 157
outcomes, 157
technique and refinements, 156–158
Fungal infections
cutaneous
dermatophytosis (tinea manuum), 432
onychomycosis, 433
deep or systemic, 433
subcutaneous, 433

G

Greater-arc injuries, 238
Grouped-fascicular neurosynthesis, 31
Growth plate fracture, 289, 290
Gunshot wound, 36–37

H

Hamate fractures
anatomy, 241–242
diagnosis, 242–243
mechanism, 242–244
treatment, 243–244

Hand infections
acute infections
bite injures, 425–427
deep space infections, 421–423
felon, 418–419
herpetic whitlow, 417–418
Methicillin-resistant *S. aureus* (MRSA), 416
necrotising fasciitis, 427–429
osteomyelitis, 424–425
paronychia, 416–417
pyogenic flexor tenosynovitis, 419–421
septic arthritis, 423–424
atypical infections
fungal infections, 432–434
mycobacterial infections, 429–432
signs and symptoms, 415
Hansen's disease. *See* Leprosy
Hatchet flap
advantages, 135
indications, 135
limitations, 135
outcomes, 136
technique and refinements, 135, 136
Head metacarpal fractures
avulsion fractures, 223–224
coronal vertical fracture, 223, 224
countersunk screws, 223, 224
dorsal view, 223, 224
longitudinal curvilinear incision, 223
osteochondral damage, 223
thumb, 227
Herpetic whitlow
antiviral therapy, 417–418
type 1 or 2 herpes simplex virus, 417
Heterodigital flaps
cross finger flap, 138–140
distant flaps, 140
FDMCA/Foucher flap, 150–151
neurovascular island flaps (Littler/Buchler flaps), 149–150
Homodigital flaps
adipofascial turnover flap (Merle flap), 142
bilateral lateral VY flaps (Kutler and Segmuller flaps), 136–137
hatchet flap, 135–136
palmar advancement flaps (Moberg flap), 148, 149
reverse, 138, 139
switch flap, 148–149
triangular advancement island flaps, 137–138
VY advancement flaps, 135
Hyphae, 432
Hyponychium, 164
Hypothenar hammer syndrome, 12

I

Iatrogenic arterial injuries, 15–16
Imaging
distal radius fractures
AP distance, 267, 269
capitate and distal pole of scaphoid, 266
CT scans, 268, 270
dorsal and volar rims, 266, 268
3D reconstruction, 268
lunate fossa and sigmoid notch, 267
PA, 267–268
palmar cortex of the pisiform lies, 266
palmar inclination, 266, 267
plain radiographs, 266
radial height (RH), 266, 267
radial inclination, 266, 267
radial translation, 266, 267
standard and angled radiographs, 267, 269
standard radiographic angles, 266, 267
teardrop, 266, 268
ulnar variance (UV), 266, 267
of scaphoid
bone scintigraphy, 251
CT scans, 252
inter and intra observer reliability, 251
MRI, 252
PA, 251
undisplaced, 251
x-rays demonstrates, 251
Instability
definition, 311
fingers (*see* Finger instability)
thumb (*see* Thumb)
Insufficiency fractures, 278–279
Internal fixation
displaced condylar fractures, 206, 207
lag screws, 201
non-union, 210
occasional surgeon, 214
spiral fractures, 202
Interosseous membrane (IOM), 380
Inter phalangeal joint (IPJ) of thumb
bony architecture, 313
joint capsule, 313
Intra articular fractures
anatomical reconstruction, 274
anatomical reduction and stable internal fixation, 272
angularly stable implants, 273
bone grafting, 274
chondral injuries, 272
CT scanning, 272
desired screw, measurement, 273–274
distal radius, curved dorsal surface, 273
dorsal approach, 273
incongruity, 272
intermediate column, 273
metaphysis and diaphysis, 274
palmar surface, distal radius, 273
persistent pain, 272
randomized trial, 272
surgical approach, 273
volar shearing fractures, 274
IPJ. *See* Inter phalangeal joint (IPJ) of thumb

J
Jersey finger, 167, 168, 171
Joint dislocation
  CMC, 219, 220, 222, 228
  dorsal
    clinical features and diagnosis, 318
    epidemiology, 318
    treatment options, 318
  TMC, 229–230
  volar, 318–319
Joint injury, children
  DRUJ ligament injury, 298
  interphalangeal joint dislocations, 297
  ligament injuries in wrist, 298
  in thumb, 297–298

K
"Kleinert traction," 54
Kutler and Segmuller flaps, 136–137
K-wires, diaphyseal fractures
  bone sutures, 199
  buddy-taping, 200
  disadvantages, 199
  length and rotation, 199
  PIP joint, 199
  poor use, 199, 200
  single ended wire, 200
  soft tissue injury, 199, 200

L
Lag screws, diaphyseal fractures, 201
Leprosy *(Mycobacterium leprae),* 432
Ligamentous injuries. *See* Collateral ligament tear
Ligamentum subcruetum, 381
Littler/Buchler flaps
  advantages, 150
  indications, 149
  limitations, 150
  outcomes, 150
  technique and refinements, 149–150
Local flaps
  adipo fascial turnover flaps, 112, 114
  description, 105
  fascio-cutaneous flaps (*see* Fascio-cutaneous flaps)
LRINEC (Laboratory Risk Indicator for Necrotizing Fasciitis) Score, 428–429
Lunate fractures
  diagnosis, 239
  mechanism, 238
  treatment, 239
Lunula, 163
Lymphatic system, 4–5

M
Mallet finger
  Baseball, 76
  classification, 76–77
  clinical features and diagnosis, 314
  complications, 315
  diagnosis, 76
  Doyle's classification, 314
  epidemiology, 313–314
  extensor tendon insertion, 76
  fixation techniques, 170, 171
  fracture, 76
  obscure, 76
  outcome and lit review, 314–315
  splinting, joints, 77
  subluxed distal phalanx, 77
  surgical treatment, 314
  treatment, 77
  treatment options, 314, 315
Mallet thumb
  clinical features and diagnosis, 315–316
  complications, 317
  epidemiology, 315
  outcome and lit review, 316–317
  surgical treatment, 316
  treatment options, 316
Malunion
  angulation deformity, 232
  boxer's fracture, 232
  diaphyseal fractures, 198
  fifth metacarpal, 232
  phalangeal neck fracture, 206
  proximal phalanx, 210, 211
  pseudoclawing, 232
  rotational deformity, 232
  step/rotational osteotomy, 232
Mangled Extremity Severity Score (MESS), 397
MCPJ. *See* Metacarpophalangeal joint (MCPJ)
Merle flap
  advantages, 142
  indications, 142
  limitations, 142
  outcomes, 142
  technique and refinements, 142, 143
Metacarpal fractures
  base fractures, 227, 228
  bone loss, 231–232
  carpometacarpal fracture-dislocations, 227, 228
  clinical assessment
    dropped knuckle, 219
    inspection, 218
    palpation, 218
    rotational, angulation/shortening deformity, 218–219
    shortening of digit, 219
    tenodesis effect, 219
  complications, 231
  functional anatomy
    articulations, 218
    cadaveric study, 218
    CMC joint, 218
    deforming force, 217
    dorsal apex, 217
    intervening connections, 218

intrinsic muscles, 217
MCP joint, 218
thumb metacarpal, 218
volar cortex, 217
hardware issues, 231
head fractures, 223–224
indications, fixation, 222
infection, 231
investigations
Brewerton view, 220
CMC joint dislocations, 220, 222
CT scan, 221, 222
MCP joint, 220
Mehara and Bhan view, 221, 222
plain radiographs, 220
skyline view, 220, 221
irreducible/unstable, 217
malunion, 232
management, 222, 233
mechanisms of injury, 218
neck fractures, 224–225
nonoperative treatment, 222–223
operative treatment, 223
post-traumatic osteoarthritis, 231
rehabilitation, 230–231
shaft fractures, 225–226
stiffness, 231
thumb, 227–230
Metacarpophalangeal joint (MCPJ)
avulsion fractures, 211–213
of fingers
bony architecture, 349
clinical features and diagnosis, 355
complex MCP subluxation, 351–353
complications, 355–356
dorsal dislocations, 350
epidemiology, 354–355
extensor retinacular system, 350
extrinsic joint support, 350
joint capsule, 349–350
lateral dislocations, 354–356
outcome and lit review, 355
sagittal band disruptions, 356–357
simple MCP subluxation, 351
surgical treatment, 355
treatment options, 355
volar dislocations, 353–354
metacarpal head, 213–215
of thumb
bony architecture, 335–336
dorsal dislocations, 339–342
extrinsic joint support, 337
joint capsule, 336–337
radial collateral ligament tear, 347–349
UCL tear, 342–346
volar dislocations, 337–339
Metaphyseal fractures, proximal phalanx
angulation, 204
dorsal angulation, 204
extensor influence, 204
flexor influence, 204
intramedullary K-wire fixation, 204–205
intrinsic muscles, 204
lumbrical tendons passes palmer, 204
metacarpophalangeal joint, 204
middle aged and elderly, 204
Micro vascular free flap transfers
anatomy and elevation techniques, 119
cosmetic implications, 118
description, 118
free innervated muscle flaps
gastrocnemius muscle, 125–127
gracilis muscle, 126, 128
latissimus dorsi flap, 125
myo-cutaneous flaps, 124–126
skin and fasciocutaneous free flaps, 119–124
and team work, 118
Microvascular toenail transfer
advantages, 142
indications, 142
limitations, 142
outcomes, 142
technique and refinements, 142
Middle phalangeal fractures
dorsal fracture subluxation, 182–183, 186–187
pilon fracture, 183, 184, 187–188
soft tissue injuries, 188
volar plate bony avulsion, 182
Motor neuron
location, 24
protection, 27–28
Mycobacterial infections
cutaneous tuberculosis, 430
leprosy *(Mycobacterium leprae),* 432
mycobacterium tuberculosis, 429–430
non-tuberculous, 431–432
tuberculous arthritis, 431
tuberculous osteomyelitis, 430–431
tuberculous tenosynovitis, 430
Mycobacterium tuberculosis (TB), 429–430
Myo-cutaneous flaps
latissimus dorsi flap, 124–125
non innervated muscle free flaps, 125, 126

N

Nail avulsions
fixation, 170, 171
removal, 169–170
replacing, 170
Nailbed defects
microvascular toenail transfer, 142
turnover adipofascial flap, 140–142
Nail bed injury
deformities, 289
laceration of sterile matrix, 288–289
Nail bed lacerations, 164, 170
Neck fractures, proximal phalanx
dorsal periosteum, 205
fixation, 205, 206

Neck fractures, proximal phalanx (*cont.*)
K-wire, 205
mal-union, 205–206
and subcondylar, 205
Neck metacarpal fractures
boxer's fracture, 224–225
immobilisation, 224
intramedullary wiring, 224, 225
Kirschner wires, 224, 225
locking plates, 224
and shaft fractures, 224
ulnar gutter cast, 224
Necrotising fasciitis
cellulitis, 427
finger test, 428
LRINEC Score, 428–429
mortality rates, 429
polymicrobial, 428
treatment modalities, 428–429
Negative pressure wound therapy (NPWT), 99
Nerve conduits, 39–40
Nerve fascicle, 24
Nerve fibre, 24, 31
Nerve gap management
conduits, 39–40
donor nerves, 38
grafting (*see* Nerve graft)
Nerve graft
allografts, 39
principles, 37–38
reconstruction, 42
surgical technique, 38–39
vascularised, 39
Nerve injury
assessment
general, 28
history, 28
motor function, 30
sensory function, 28–30
water immersion test, 31
blood supply and surgical anatomy, 25
classifications, 302
closed injury, 302
development, neurophysiology, 23
lacerated nerve, 303
management, nerve gap (*see* Nerve gap management)
micro-anatomy
axon, 24
epineurium, 25
fascicles collection, 24, 25
neuron, 24
peripheral nerve, 24
peripheral, 303
peripheral nerve (*see* Peripheral nerve)
proximal, 304
regeneration and recovery, 301–304
repair (*see* Nerve repair)
transfer (*see* Nerve transfer)
Nerve repair
outcomes, 42
recovery
bleeding, 32
epineural, 31
meticulous microsurgical techniques, 31
neurosynthesis, 31
postoperative motor and sensory re-education, 32
primary, 31
timing, 32
tissue, 31
surgical technique
anaesthesia and pneumatic tourniquet, 33
classification, 35–36
delayed exploration, 32
epineural, 33
fascicular, 33–34
gunshot wound, 36–37
immediate/primary, 32, 33
motor and sensory return, 34
neurapraxia, 36
post-operative care and complications, 34–35
traction injury, 35
wound closure, 33
Nerve transfer
clinical application, 40–41
electromyography, 41, 42
end to side neurosynthesis, 41
exploration, 41
grafting, 40
investigations, 41
motor, 40
principles, 40
proximal peripheral nerve injuries, 40
restore function, 40
Neurapraxia, 36
Neuropathic pain
epidemiology, 450
interventional procedures, 452
patient information and education, 451
pharmacotherapy
first line treatment, 451
second line treatment, 451
third line treatment, 451
physical and vocational rehabilitation, 452
signs, 451
symptoms, 451
Nociception
central, 438
peripheral, 438
Nociceptive pain
central nociception, 438
peripheral nociception, 438
Non-tuberculous mycobacteria (NTM), 431–432
Non-union
avulsion fractures, 211–213
proximal phalanx, 210, 211
Numerical rating scale (NRS), 441

O

Open–dorsal approach, 255–256
Open fractures, 400

Open–palmar approach, 255
Opiates, 444
Opioids, 444
Osteomyelitis
  acute or chronic, 424
  of fourth metacarpal head, 425
  iatrogenic, 424
  infecting organisms, 424
  intramedullary infection, 425
  localised, 425
  Stage IV, 425
  stages, classification, 425
  superficial, 425
  treatment, 425
  tuberculous, 430–431
Osteotomy
  de-rotational, 210, 211
  healed mal-union, 208
  metacarpal, 210
  metaphyseal fractures, 204
  site, 210
  timing, 210
Outcomes, PIP joint
  bone injuries, 189
  head of proximal phalanx, 188
  soft tissue injuries, 188

P
Pain
  acute (*see* Acute pain)
  character, 440–441
  chronic (*see* Chronic pain)
  intensity
    NRS, 441
    VAS, 441
    VRS, 441
  neuropathic, 438
  nociceptive
    central nociception, 438
    peripheral nociception, 438
  pre-emptive and preventive analgesia, 439–440
Pain management
  acute pain
    local anaesthetic blockade, 447–448
    patient information and education, 442
    pharmacotherapy, 442
    ultrasound visualisation, 448–449
  chronic pain, 449
  CRPS
    interventional procedures, 454
    patient information and education, 453
    pharmacotherapy, 453
    physical and vocational rehabilitation, 454
    psychological intervention, 454
    surgery, 454
  neuropathic pain
    interventional procedures, 452
    patient information and education, 451
    pharmacotherapy, 451–452
    physical and vocational rehabilitation, 452
  post amputation pain
    non-pharmacological, 450
    pharmacotherapy, 450
    prevention, 450
Palmar advancement flaps (Moberg flap)
  advantages, 148
  indications, 148
  limitations, 148
  outcomes, 148
  technique and refinements, 148, 149
Paronychia
  chronic, 417
  infection or abscess, 416
  release, 417
  "run-around abscess," 416
Patient-Rated Wrist Evaluation (PRWE) scores, 265
Pedicled groin flap
  ASIS, 115, 116
  medial length, 116, 119–120
  neonate with extensive defect, 116, 118
  SCIA, 114–116
Perforator flap, 107
Peripheral nerve
  denervated Schwann cells, 27
  lacerations, 23
  management, 23
  morphology, 26–27
  physiology, 25–26
  protection, motor neurons, 27
  sensory neurons, 27–28
Phalangeal fractures
  distal, 291–292
  middle and proximal, 292–293
Phantom limb pain, 450
Pilon fracture, 183, 184, 187–188
PIPJ. *See* Proximal interphalangeal joint (PIPJ)
Pisiform fractures
  anatomy, 241
  diagnosis, 241
  mechanism, 241
  treatment, 241
Post amputation pain
  epidemiology, 449–450
  non-pharmacological, 450
  pharmacotherapy
    antineuropathic agents, 450
    calcitonin, 450
    ketamine, 450
    opioid analgesics, 450
    simple (non opioid) analgesics, 450
  prevention, 450
Posterior interosseous island fascio-cutaneous flap (PIIF), 109–112
Postoperative pain. *See* Acute pain
Pretrochlear bursa, 321
Preventive analgesia, 439–440
Primary flexor tendon repair
  A2 and A4 pulleys, 50
  advantages, 50

Primary flexor tendon repair (*cont.*)
Bruner incision, 61
cost, 52
cuts
finger extension, 49, 62–63
finger flexion, 49, 63–64
early mobilisation, 50
FPL tendon, 52, 53
post-operative management, 50, 64
rupture rates, 51–52
skin
incision, 49, 50
marking adjacent to wound, 61–62
splint for early active motion, 52
suture technique, 50–51, 64
timing, 49
tourniquet control, surgery, 49, 61
'triple-Tsuge' technique, 52–53
Primary idiopathic disease, 16
Primary reconstruction, 407–408
Propeller flaps, 111
Proximal interphalangeal joint (PIPJ)
aetiology
bony anatomy, 176, 177
concave joint surfaces, 176
convex joint surfaces, 176
dorsal dislocation, proximal phalanx, 176
dorsal subluxation, 176
mechanism of injury, 175
side and volar views, 175, 176
bone injuries, 189
bony architecture and angles in hand, 319–320
clinical presentation, 176–178
collateral ligament ruptures, 324–327
complications, 189
CT scan, 178
description, 175
dorsal dislocations
classification, 321–322
clinical features and diagnosis, 322
complications, 324
epidemiology and pathomechanics, 321
outcomes, 324, 325
surgical treatment, 324
treatment options, 322, 323
type I and II Injuries, 322–323
type III Injuries, 323
of fingers
bony architecture, 320
injuries to the PIPJ, 321
joint capsule, 320–321
middle phalanx injuries (*see* Middle phalangeal fractures)
open dorsal capsular injuries, 327–335
palpation, 177
phalanx (*see* Proximal phalangeal fractures)
prognosis, 189
radiograph, 178
soft tissue injuries, 178–181
stiffness, 176
swelling, 176–177
volar dislocation
clinical features and diagnosis, 327
complications, 327
epidemiology and pathomechanics, 327
surgical treatment, 327
treatment options, 327
volar split laceration, 177
Proximal phalangeal fractures
extra-articular fracture
bone, 181
surgical treatment, 183–184
head, 188
intra-articular fractures
bone, 181–182
surgical treatment, 183–186
Proximal phalanx
assessment
computerised radiology, 197
crush injury, 196
malrotation, 197
radiographs, 197
spiral fracture, 196–197
transverse fractures, 196
catastrophic and irretrievable, 195, 196
comminuted fractures, 209–210
condylar fractures, 206–209
conservative treatment, 195
description, 195
diaphyseal fractures, 197–202
fixations, 195
good/perfect results, 195
internal fixation, 214
malunion, 210, 211
metaphyseal fractures, 204–205
neck fractures, 205–206
non-union, 210
spiral fractures, 202, 203
surgical time, 215
transverse fractures, 202–204
union, 210
Proximal pole fractures, 254–255, 257

Q
Quaba flap
advantages, 145
indications, 144
limitations, 145
outcomes, 145
technique and refinements, 144–145

R
Radial collateral ligament tear
clinical features and diagnosis, 348
complications, 349
epidemiology, 347
outcome and lit review, 349
pathology, 348
surgical treatment, 348–349
treatment options, 348

Radical debridement, 397–400
Random pattern flaps, 102, 104
Raynaud's disease, 16–17
Recurrent or chronic exertional compartment syndrome, 10
Rehabilitation
  acute carpal instability
    closed reduction, 374
    immobilizing wrist, 373–374
    isolated lunotriquetral dissociation, 377
    isolated scapholunate dissociation, 376–377
    open reduction and repair technique, 374–376
  complex upper limb trauma, 410
  CRPS, 454
  distal phalanx, 169–171
  extensor tendon injuries, 84, 85
  flexor tendon, 45, 61
  metacarpal fractures, 230–231
Reinnervation, nerve, 27, 34, 38, 41
Replantation, fingertip, 289–290
Revascularization, injured extremity
  embolectomy, role, 401
  of proximal injuries, 400–401
  quality of debridement, 401
  reperfusion injury, 400
  vein grafting, 401
  viability of limb, 400
Reverse Bennett's fracture, 360
Reverse homodigital flap
  advantages, 138
  indication, 138
  limitations, 138
  outcomes, 138, 139
  technique and refinements, 138
Reverse perilunate instability, 368
Rolando's fracture, 229, 233
Rotational deformity, 218–219, 227, 232
"Run-around abscess," 416
Rutherford classification, lower extremity, 13

S

Sagittal band (SB) disruptions
  clinical features and diagnosis, 356
  complications, 357
  epidemiology, 356
  outcome and lit review, 357
  rupture, 78–79
  surgical treatment, 356–357
  treatment options, 356
Salvage, 376–377
Scaphoid fracture(s)
  acute (*see* Acute scaphoid fracture)
  Herbert classification, 250
  Russe classification, 250
Sensory function
  areas, 28–29
  digital nerves, 29
  distribution, sensory supply, 29
  hand, 28
  motor function, 30
  moving touch, 28
  proximal/brachial plexus injuries, 29–30
  testing, nerve repair, 28
  vibration, 28
Septic arthritis
  arthroscopic debridement, 423, 424
  of DIPJ, sinus formation and osteomyelitis, 423
  H-flap and retraction, extensor tendon, 424
  S. Aureus and Streptococcal infections, 423
  treatment, 423
Shaft metacarpal fractures
  early mobilisation, 225
  functional brace, 225
  intramedullary fixation, 226
  nailing, 226
  open reduction, 226
  plaster slab/immediate mobilisation, 225
  plating, 226
  surgical techniques, 225–226
  thumb, 227
Short arc motion protocol, 83
Skeletal fixation
  bone loss, 404–406
  bone shortening and fixation, 403–404
  bone viability, assessment, 403
  debridement, 402
  internal fixation, 401–402
  stable, 401
Skin
  and fasciocutaneous free flaps
    anterior thigh flap, 120–122
    dorsal circumflex scapular artery axis, 123, 124
    free groin flap, 119–121
    lateral arm fascio-cutaneous flap, 121, 123
    medial plantar flap, 123
    radial forearm flap, 120
    temporal fascia free flap, 124
    tensor fascia lata flap, 123–124
  trauma
    conservative resection, 94
    degloved skin and subcutaneous tissue, 93
    dermis bleeds, 93
    full thickness gloving injury, 94, 95
    SSG/full thickness skin graft, 93–94
    vacuum assisted dressings (VAC), 94
    vascularised or contaminated skin, 94
Skin cover
  avulsed hand amputation, 96–98
  bone fixation, 96
  choice of reconstruction, 96
  compound defect, skin and ulnar nerve loss, 98, 100–101
  concept, 96
  cosmesis, 98, 102
  debridement and proper repair, 96
  forearm revascularization, 96, 99–100
  guide/tool kit, 96
  management, compound fractures, 96
  pedicled forearm flap, 98

Skin cover (*cont.*)
quality and durability, 98
skin loss and extensor tendon loss, 98, 101
split skin graft donor site, 98, 102
SSG, 98
tendon and nerve grafts, 98
wound closure techniques, 98–104
Skin grafts, non-flap, 133
Small free flaps
artificial dermis, 156
DASH score, 156
digits, 155
disadvantages, 156
first webspace free flap/dorsalis pedis free flap, 158–159
free medial plantar flap, 157–158
free posterior interosseus flap, 156–158
functional surfaces of digits, 155
pain and cosmetic deformity, 156
toe pulp transfer, 155
venous flow through flap, 156, 157
webspace reconstruction, 155
Soft tissue cover
comparative reviews
in distal thumb reconstruction, 154
of fingertip reconstruction, 145–147
flap (*see* Flap(s))
non-comparative case series
in distal thumb reconstruction, 154
of fingertip reconstruction, 146, 147
non-flap, 133–134
thumb (*see* Thumb)
Soft tissue injuries
collateral ligament injuries, 178–181
middle phalanx, 188
volar plate, 178–181
Space of Poirier, 368
Spiral fractures, proximal phalanx
collateral damage, 202
extensor and periosteum, 202
intra-medullary K-wire, 202
lateral approach, 202, 203
ulnar approach, 202
Sports hand injuries, 313, 321, 342, 355
Staged reconstruction, 407–408
Stener lesion, 344, 345, 348, 355
Stiffness (digital), 319, 321, 326, 330, 333, 342, 353
Subluxation, MCPJ of fingers
complex
clinical features and diagnosis, 351–353
complications, 353
outcome and lit review, 353
surgical treatment, 352–354
treatment options, 351
simple, 351
Subungual haematoma, 164–165, 169
Surgery
condylar fractures, 208
CRPS, 454
flexor tendon, 45, 61
PIP joint (*see* Proximal interphalangeal joint (PIPJ))
for scaphoid fracture
open–dorsal approach, 255–256
open–palmar approach, 255
percutaneous, 256
vasospastic injuries, 17–18
Swanson's classification, 134
Switch flap
advantages, 148
indications, 148
limitations, 148, 149
outcomes, 148–149
technique and refinements, 148, 149

T
Tendon(s)
flexor (*see* Flexor tendon)
injury, 299–300
sheath management
A2 and A4 pulleys, 53–54
closure, 53
free movement repair, 54
haemostasis, wound, 54
plastic surgery, 53
Tenosynovitis
flexor (*see* Flexor tenosynovitis)
tuberculous, 430
Thromboangitis obliterans, 19
Thrombosis
Allen's test, 14
conditions, 13–14
graft, 14
hypothenar hammer syndrome, 13
occlusive, 14
symptoms, 13
treatment, 14
Thumb
acute instability
CMCJ, 361–363
collateral ligament injury, 317–318
IPJ, 313
joint dislocation, 318–319
mallet thumb, 315–317
MCPJ, 335–349
PIPJ, 319–330
algorithm, soft-tissue, 155
classification, 148
free flaps, 151–153
local flaps
distant pedicled flaps, 151
heterodigital flaps, 149–151
homodigital flaps, 148–149
metacarpal fractures
carpometacarpal fracture-dislocations, 229
description, 227
extra-articular base fractures, 227–228
head fractures, 227
intra-articular base fractures, 229

shaft fractures, 227
TMC joint dislocations, 229–230
outcomes, 154–155
Tinea infections, 432
Tourniquet, 169
Traction injury, 35
Transverse fractures, proximal phalanx
configuration, 202
description, 202
dorsal approach, 203–204
lister loop technique, 202–203
Trapeziometacarpal (TMC) joint dislocations
cadaveric study, 229–230
description, 229, 230
diagnosis, 230
open and closed reduction, 230
pronation and extension after reduction, 230
Trapezium fractures
complications, 246–247
diagnosis, 246
incidence, 246
mechanism, 246
treatment, 246
Trapezoid fractures, 247
Trauma
assessment, 89
compartmental compression
carpal tunnel/Guyon's canal, 92
fasciotomies, 92
forearm, 91–92
intra-compartmental pressures, 91
muscles of forearm and hand, 91
pain, 91
wait and watch approach, 91
escharotomy, 92
limb injury, 90
mechanism of injury, 90
plastic and vascular surgical skills, 89–90
vascularity, 90–91
wound contamination, 91
wound debridment, tissue viability and degloving
bone, 95
crushed forearm before and after, 92, 93
muscle, 95
skin, 93–95
sub-cutaneous tissue, 92–94
vessels, 95
Triangular fibrocartilage complex (TFCC), 380–381
Triquetrum fractures
complications, 240
diagnosis, 240
incidence, 239–240
treatment, 240
Tuberculosis (TB)
cutaneous, 430
mycobacterium, 429–430
Turnover adipofascial flap
advantages, 142
indications, 140
limitations, 141
outcomes, 142
technique and refinements, 140–141

U

UCL. *See* Ulnar collateral ligament (UCL) tear
Ulna, distal fractures
classification, 274–275
distal ulnar fractures and symptomatic non-unions, 277
DRUJ congruity or stability, 276
DRUJ stability, 275, 276
forearm fractures, 276–277
pathoanatomy of TFCC attachments, 275–276
styloid, 274
ulnar styloid, 277
weight bearing function, 274, 275
Ulnar collateral ligament (UCL) tear
clinical features and diagnosis, 343–345
complications, 346
epidemiology, 342
pathology, 342–343
surgical treatment, 345–347
treatment options, 345

V

Vacuum assisted therapy (VAC) therapy, 99
Variable angle, 281
Vascular disorders, upper limb
clinical presentation, 5
clinical tests, 5–6
Doppler signal, 6
investigations, 6–7
and neck, 5
palpation, 5
patient history, 5
symptoms, 5
Vascular injury
closed injuries, upper limb arteries, 304–305
management, 305
open trauma, 304
Vascularised nerve grafts, 39
Vasospastic injuries
arteriovenous fistula, 18
cessation, 16
diagnostic testing, 17
disease
primary idiopathic, 16
Raynaud's, 16–17
management, 17
physical examination, 17
regulation, 17
surgery, 17–18
symptoms and signs, 17
therapy, 17
vascular constriction, distal extremities, 16
vasomotor homoeostasis, 17
Venkatswami flap
advantages, 138
indications, 137

Venkatswami flap (*cont.*)
limitations, 138
outcomes, 138
technique and refinements, 137–138
Venous flow through flap
advantages, 156
indications, 156
limitations, 156
outcomes, 156
technique and refinements, 156, 157
Verbal rating scale (VRS), 441
Visual analogue scale (VAS), 441
Volar finger defects, 145
Volar oblique (or volar facing), 134
Volar plate injury(ies)
bony avulsion, 182–183
and collateral ligament
bony avulsion, 178, 179
boutonniere injuries, 179
farm environment, 180–181
hyperextension injury, 178
outcome, 181
PIP joint dislocation, 179–180
radiographs, bony boutonniere injuries, 179, 180
staphylococci and streptococci, 180
steroid injection, 178
suspicions, 179
dorsal dislocations
clinical features and diagnosis, 339–342
complications, 342, 343
epidemiology, 339, 340
outcome and lit review, 341–342
surgical treatment, 341
treatment options, 340–341
VY advancement flaps
advantages, 135
indications, 135
limitations, 135
outcomes, 135
technique and refinements, 135

W
Wallerian degeneration, 25
Wound closure techniques
collagen substitutes, 102
full thickness skin grafts, 102
meshed skin grafts, 102–104
natural healing with dressings, 99
neonate, 102, 103
primary and delayed primary wound closure, 98–99
SSG, 102–104
VAC therapy, 99, 102
vacuum therapy, 102
Wound excision, 398
Wrist fracture(s)
arthroscopy, 256
associated injuries, 277–278
and axial loading, 249
bone quality, 262
component of multiple injuries, 262
joint effusion/haemarthrosis, 251
movement, 251
outcome, 261
radial deviation and extending, 250
skeletally immature, 261