Ian A. Trail
Andrew N. M. Fleming
Editors

Disorders of the Hand

Volume 2: Hand Reconstruction and Nerve Compression

Springer

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Volume 2: Hand Reconstruction and Nerve Compression

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Preface

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

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

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

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

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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

Part I Hand Reconstruction

1 Nerve Reconstruction 3
Julia K. Terzis and Petros Konofaos

2 Tendon Transfers. 33
Michael Solomons

3 Extensor Tendon Reconstruction. 57
Simon Ball and Rupert Eckersley

4 Flexor Tendon Reconstruction. 75
L. Paul van Minnen and Thybout M. Moojen

5 Malunion/Nonunion of the Digits 97
Carlos Irisarri

6 Scaphoid Non Union 115
Hermann Krimmer

7 Distal Radius Malunion 129
Francisco del Piñal

8 Local Vascular Bone Graft Reconstruction 153
Louisa N. Banks and Ian A. Trail

9 Chronic Instability of the Fingers and Thumb. 161
Carlos Heras-Palou

10 Chronic Carpal Instability 179
Marc Garcia-Elias

11 Chronic Distal Radio-ulna Instability 195
Marc Bransby-Zachary and Ian A. Trail

Part II Nerve Compression

12 Carpal Tunnel Syndrome 217
Ian S.H. McNab and Sarah Tucker

13 Nerve Compression – Median Nerve Entrapment in the Forearm 231
B.J.R. Sluijter and M.J.P.F. Ritt

14 Nerve Compression: Ulnar Nerve of the Elbow 243
Prasad Sawardeker, Katie E. Kindt, and Mark E. Baratz

15 Ulnar Tunnel Syndrome 267
Michael J. Brody and Randipsingh R. Bindra

16 Compressive Neuropathies of the Radial Nerve 283
Bradley C. Carofino, Allen T. Bishop, and Alexander Y. Shin

Index 297

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Part I

Hand Reconstruction

Nerve Reconstruction

1

Julia K. Terzis and Petros Konofaos

Keywords

Peripheral Nerve Injuries • Nerve Reconstruction • Principles of nerve repair • End-to-end repair • End-to-side-neurrorhaphy • Nerve grafting • Vascularized nerve grafting • Brachial plexus injuries • Avulsion plexopathies • Nerve Transfers

Introduction

Until the late eighteenth century it was believed that peripheral nerves did not regenerate after injury. Introduction of microsurgical techniques [1] in peripheral nerve surgery and the establishment of the principle of tension free repair [2] allowed inspired surgeons such as Narakas, Millesi, Allieu, Brunelli, Terzis, Doi, Gu, and others to suggest several new approaches to nerve reconstruction.

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Many factors influence the success of nerve repair and reconstruction. The age of the patient, the timing of nerve repair, the level of injury, the extent of the zone of injury, the technical skill of the surgeon and the method of repair contribute to the functional outcome after nerve injury. The basic tenets of nerve repair continue to hold true, including an accurate preoperative assessment, properly timed and executed exploration, meticulous nerve repair and intensive postoperative re-education [3].

As soon as nerve injury occurs, its target muscles begin to undergo atrophy and lose their motor end plates. Expedient diagnosis and testing is the best means of maximizing functional return. An adequate and properly timed treatment of peripheral nerve injuries is crucial to achieve a reasonable satisfying clinical outcome, although a complete nerve injury always will lead to varying degrees of permanent dysfunction in adults.

The aim of this chapter is to review the principles and techniques of nerve reconstruction and to discuss the options of repair including direct repair, nerve grafts, end-to-side neurorrhaphy

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and nerve transfers following nerve injuries in the upper extremity.

Background – Aetiology

The hand has been called an extension of the brain, and the sensory and motor performance of the hand is based on adequate function of components in the peripheral as well as the central nervous system. From a hand surgery perspective, poor functional outcomes after peripheral nerve lesions represent a frustrating problem.

Injuries to peripheral nerves are common in all forms of upper extremity trauma but management of them remains a challenge. Common causes include lacerations, fractures, dislocations, ligamentous tears, crush and amputation injuries. Injuries are most often caused by domestic or industrial accidents or interpersonal violence. Nerve injuries range from nerve compression lesions, like carpal tunnel syndrome, up to severe rupture and avulsion of spinal nerve roots of the brachial plexus (BP). Males suffer traumatic nerve injuries at a ratio of 2.2:1 compared with females [4]. The typical patient who sustains a nerve laceration is a male in his late teens or early twenties.

As a protective instinct, the arm, forearm and hand are frequently outstretched during injury. The upper extremity therefore often absorbs the initial impact, with the dominant arm involved slightly more frequently. The most frequently injured nerves are the radial nerves of the index finger, the ulnar digital nerves of the small finger, and the median and ulnar nerves at the wrist level [5].

As far as BP injuries are concerned, high-velocity motor vehicle accidents account for the majority of the cases; most studies report that motorcycle accidents are responsible approximately twice as often as automobile accidents. Nerve injuries in these cases are from traction and compression, with traction accounting for 95 % of injuries. Other common causes include (in different percentages according to different studies) industrial accidents, pedestrian vehicle accidents, snowmobile accidents, gunshot wounds, and other penetrating injuries [6].

Only around 3 % of hand injuries include injury to peripheral nerve trunks. Even a minor injury to a finger causing a digital nerve injury (incidence 6.2/100,000 inhabitants/year) may induce dysfunction of the hand. The consequences of a median or ulnar nerve injury in the forearm are even more wide-ranging for the patient. The injury does not only cause problems in the patient's professional life but leisure activities are also severely impaired.

The overall incidence of BP injuries in multitrauma patients secondary to motor vehicle accidents ranges from 0.67 to 1.3 % [7]. This number increases to 4.2 % for victims of motorcycle accidents. This difference can easily be explained by the increased forces applied to the BP of the unprotected body during a high velocity motorcycle accident.

Presentation

Pathophysiology

Following peripheral nerve injury, morphologic and metabolic changes occur. Within the first few hours to days, morphologic changes occur in the corresponding neurons, including swelling of the cell body, displacement of the nucleus to the periphery, and disappearance of basophilic material from the cytoplasm, a phenomenon termed chromatolysis.

Within 2–3 days of injury, edema forms in the axonal stumps and the distal stump undergoes Wallerian degeneration. This degenerative process is called Wallerian degeneration after Augustus Waller, who first characterized morphological changes in the distal stump of sectioned frog glossopharyngeal and hypoglossal nerves 160 years ago [8]. During Wallerian degeneration, Schwann cells from the distal stump proliferate, help inflammatory infiltrating cells to eliminate debris, and upregulate the synthesis of trophic (factors which support neuronal survival and axonal growth) and tropic (factors which influence the growth direction of the regenerating axons) factors. The Schwann cells, close to the site of transection, go through the same type of changes as the Schwann cells in the distal nerve segment.

After 3–6 weeks, endoneurial tubes are left behind that consist of basement membranes lined with Schwann cells which proliferate and organize into columns, guiding the regenerating axonal sprouts within the basement membranes to their targets. In the gap between the proximal and distal nerve segment an inflammatory response occurs and a fibrin matrix, filled with macrophages, is formed. Schwann cells can migrate from both ends where the migration of such cells takes part in concert with the outgrowing axons. Metabolic changes within the neuronal cell body involve switching the machinery normally set up to transmit nerve impulses to manufacturing structural components needed for reconstruction and repair of the damaged nerve.

End organs also undergo changes after nerve injury. Complete atrophy occurs within 2–6 weeks of denervation. Fibrosis occurs in motor fibers at 1–2 years and fragmentation and disintegration occur by 2 years. It is generally agreed that functional recovery is diminished if the nerve does not reach the motor end-plate by 12 months. Sensory end-organs are less sensitive to denervation than motor end-organs. It has been shown that recovery of protective sensibility is possible even after many years from nerve injury [9] but that the degree of functional sensation decreases the longer the delay in nerve repair.

Classification of Nerve Injury

In 1941, Cohen introduced a classification to describe nerve injuries which was later popularized by Seddon [10]. According to this, there are three distinct clinical entities for a dysfunctional nerve: neurapraxia, axonotmesis or neurotmesis.

Neurapraxia, refers to a localized conduction block, is a comparatively mild injury, with motor and sensory loss but no evidence of Wallerian degeneration. The nerve distally conducts normally. Tinel's sign (a tingling sensation perceived distally when percussion is carried out over the injury site of a nerve which indicates involvement or in a partial lesion the commencement of regeneration as the nerve attempts to heal) [11]. The underlying mechanism is attributed to focal demyelination or ischemia. Recovery may occur within hours, days, weeks or up to a few months.

In axonotmesis the axons are ruptured, but the epineurium and perineurium remain intact. It is commonly seen after crush injuries. Wallerian degeneration does occur distal to the injury, but regeneration from the proximal stump is still possible. Functional recovery depends on the severity of the lesion and the degree of internal disorganization in the injured nerve as well as its distance to the end organ.

Neurotmesis describes the situation in which the entire nerve trunk is completely ruptured and axonal continuity can not be restored. Sharp injuries, some traction injuries or injection of noxious drugs are the most common causes. Prognosis for spontaneous recovery is extremely poor without surgical intervention.

In 1951, Sunderland [12] expanded upon Seddon's classification system by defining five distinct degrees of nerve injury. Sunderland's 3rd and 4th degree injuries were included as extensions of axonotmesis and neurotmesis respectively.

First degree injury (neurapraxia) is a localized conduction block with preservation of the nerves'anatomical continuity. Although recovery is complete, the time required varies from days to 3 months.

In second degree injury (axonotmesis) the endoneurium and the perineurium remain intact. A Tinel's sign is present. Wallerian degeneration occurs distal to the site of injury. Nerve recovery may be complete.

Third degree injury involves endoneurial scarring and disorganization within the fascicles. The endoneurial tube is disrupted, resulting in erroneous alignment of the regenerating fibers. An advancing Tinel's sign indicates the level of regeneration, but the degree of recovery will not be complete.

In fourth degree injury the nerve is in continuity, but regeneration does not occur across scar block. A Tinel's sign is found at the level of the injury, but does not advance further. It is commonly caused by severe stretch, traction, crush, cautery injury or nerve injection. Surgical intervention is necessary.

In fifth degree injury there is severance of the nerve trunk. Recovery is not possible without surgical intervention. This lesion is associated with penetrating trauma.

Preoperative Investigation and Diagnosis

The formulation of a diagnosis, treatment plan, and prognosis can be largely accomplished by means of a careful and detailed history and physical examination. The timing of the injury will help guide treatment recommendations, which the mechanism gives clues about the severity of the lesion.

The examination of passive range of motion of all joints of the affected extremity should be done and recorded before examination of active range of motion. All the upper extremity muscles have to be tested and compared to corresponding ones on the contralateral normal side. The grip and pinch muscle strength are measured using a Preston dynamometer set on intermediate position. The sensory evaluation should include the supraclavicular area, the arm, the forearm, and the hand. Color and trophic changes of the arm should be observed. For evaluation of sensibility in the hand, static and moving two-point discrimination (needs to take place with the patient sitting across from the examiner and having the eyes closed), Semmes-Weinstein monofilament pressure testing or von Frey cutaneous pressure threshold testing, testing for perception of high- and low-frequency vibration, and ninhydrin testing should be performed.

A detailed history of pain, its onset, duration, quality, sharpness, and radiation is routinely recorded. The results are recorded on a BP chart (Fig. 1.1) which includes all muscle groups of the

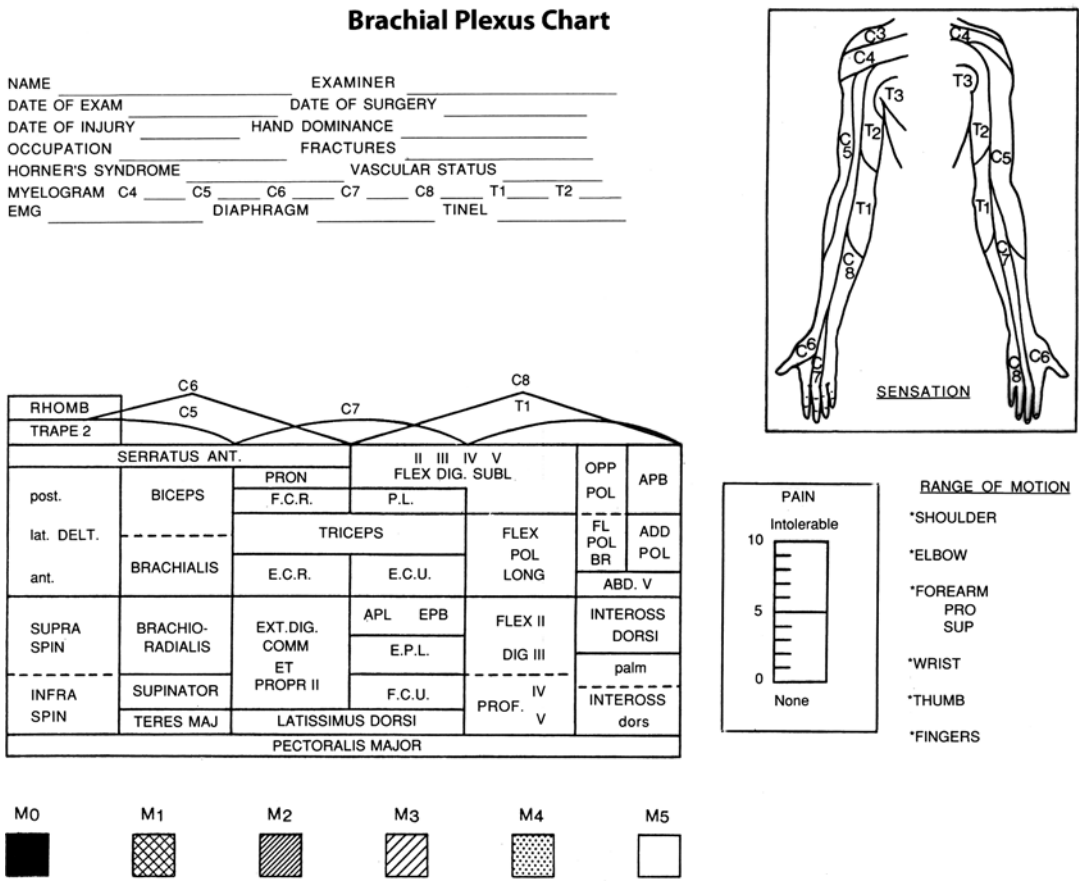


Fig. 1.1 Preoperative brachial plexus chart

upper extremity, sensory mapping, and pain level. This is important not only for the initial visit, but also to document and follow clinical recovery after repair.

The British Medical Research Council grading scale is used by most physicians. This system has been further modified by Terzis [13] with intermediate grades of (+) and (−).

In cases of BP injuries, the presence of Horner's sign is a strong indicator of avulsion of the C8 and T1 roots. Moreover, the absence of a Tinel's sign in the supraclavicular area is a strong indicator of root avulsion and is a bad prognostic sign because it indicates lack of intraplexus donors for reconstruction. On the other hand, a positive Tinel's sign is a strong indicator of roots connectivity with the spinal cord.

The initial electrodiagnostic evaluation of the upper extremity should include needle electromyography and nerve conduction studies. Axonal discontinuity results not only in predictable pathologic features but also in time-related electrical changes that parallel the pathophysiology of denervation. Wallerian degeneration results in the emergence of spontaneous electrical discharges for at least 3 weeks after the injury. Therefore, a needle electromyogram should be postponed for at least that long and preferably carried out at 6 weeks.

The lamina test is performed in cases of adult BP injuries. Tiny volleys of electrical stimulation are applied at the level of each foramen on each exiting root to determine whether the patient perceives the area of the dermatome innervated by this root. A positive response would be strong evidence against avulsion.

Depending on the mechanism of injury and the location of the nerve lesion, radiologic imaging may be necessary to confirm or support a diagnosis of a nerve injury. In cases of BP injuries, imaging studies (such as myelography, CT myelography, and magnetic resonance imaging) are used in order to detect abnormalities of the nerve roots (such as traumatic pseudomeningocele, deformity of nerve root sleeves, dural scar, and nerve root avulsion). A combination of myelography with computed tomography of the cervical spine is used to identify root avulsions (Fig. 1.2). In case of previous vascular injury and subsequent reconstruction, angiography should be employed to investigate the blood supply of the extremity and to identify any vascular compromise (Fig. 1.3).

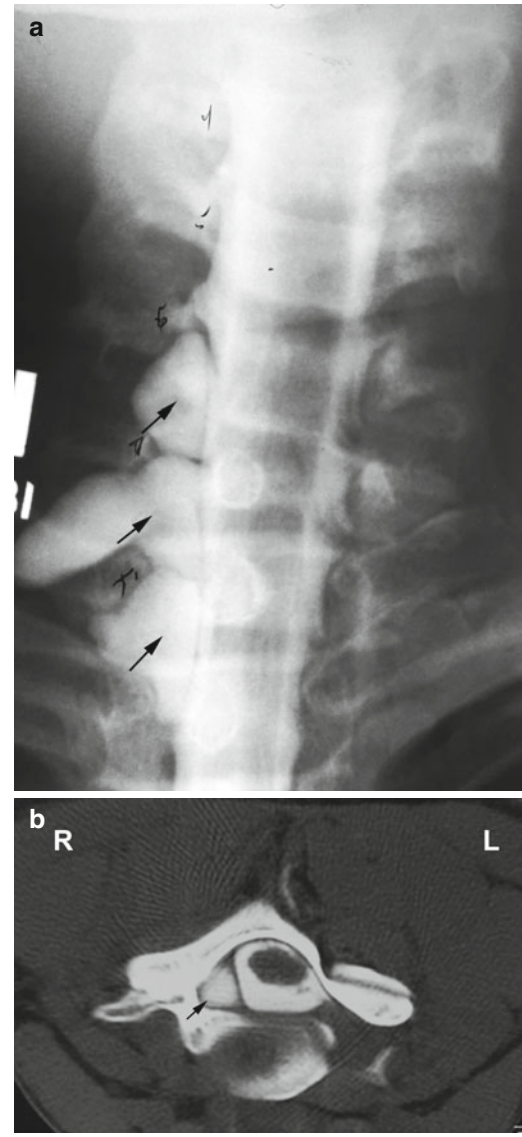


Fig. 1.2 CT Myelography showing root avulsion. (**a**) Myelography of the Cervical spine in a patient with multiple root avulsions (*arrows*). (**b**) Example of CT myelography in a patient with severe right brachial plexus injury. Note avulsed root on the right (*arrow*)

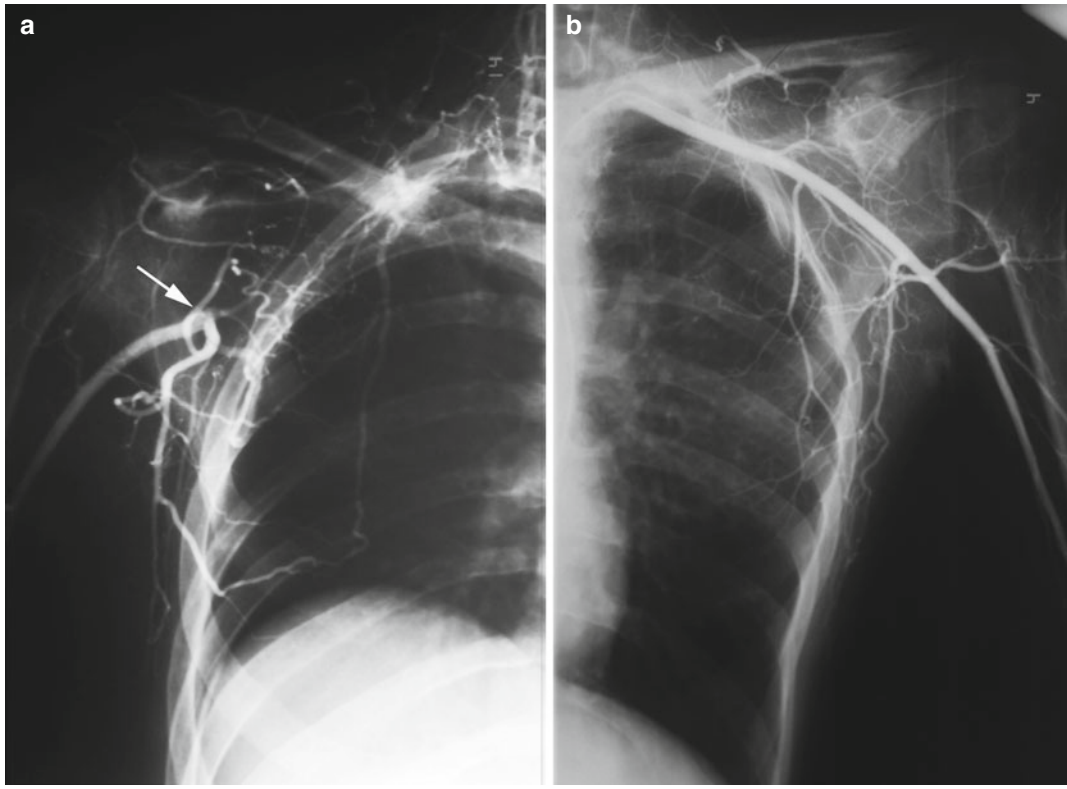


Fig. 1.3 Angiography of upper extremity in cases of vascular injury. (**a**) Angiography of right upper extremity. Note interruption of (*R*) subclavian artery (*arrow*). Axillary artery receives flow from collateral vessels. (**b**) Normal angiography of left upper extremity

Treatment Options

Principles of Nerve Repair

The basic principles of nerve repair include a sequence of eight basic principles that represent the basis of the microsurgical management of the nerve injured patient [14]:

1. Preoperative assessment of motor and sensory function
2. Adequate debridement of the proximal and distal nerve stumps in order to allow nerve regeneration to proceed across the repair site
3. Utilization of microsurgical techniques
4. Tension-free repair
5. When a tension-free repair is not possible, use of other techniques for nerve repair; nerve grafts, end-to-side nerve repair or nerve transfers
6. Primary repair; when this is not possible, delay repair for approximately 3 weeks when the 'zone of injury' is clarified
7. Utilization of a nerve repair technique that allows early protected range of motion to permit nerve gliding
8. Occupational and physical therapy in order to maximize the clinical outcome

Timing of Nerve Repair

A primary nerve repair is defined as reconstruction shortly after the injury. Secondary repair is defined as occurring at a later period after injury. Several investigators have reported that nerve repair is better when performed within 6 weeks of injury and several studies have shown primary repair to be superior to secondary repair as long as the tissue bed is adequate [15, 16].

In general, nerve injuries associated with open wounds require early exploration except from gunshot wounds, which are more appropriate to be treated as closed or blunt trauma. In crush nerve lesions or injuries associated with

significant soft tissue damage it can be difficult to estimate the extent of the zone of injury. In these cases, a delayed repair, after 3 weeks, is indicated, when the zone of injury becomes better demarcated and the extent of scar tissue can be easily defined.

In closed or blunt trauma, initial management is expectant with close observation. If complete recovery is not observed within 6 weeks, electrodiagnostic studies should be obtained for baseline evaluation. If at 12 weeks complete recovery has not occurred, repeat electrodiagnostic studies should take place. Presence of increase of motor units potentials in electromyography is an indicator that spontaneous reinnervation most likely will follow. Lack of signs of reinnervation (clinical or electrical) at 12 weeks post injury requires surgical exploration.

BP injuries are worth specific consideration regarding the timing of exploration and reconstruction. Such injuries require extra care since BP injuries usually come with other associated injuries including fractures, vascular injuries and associated soft-tissue injury. Although exploration of the BP injury may need to be performed with a slight delay, the modern management of BP injuries is early aggressive microsurgical reconstruction [17].

Techniques of Nerve Repair

In general, nerve exploration and repair should be performed under high magnification of the operating microscope. Exploration always takes place proximal and distal to the lesion site until normal nerve to inspection and palpation is encountered. If the history and physical examination is suspicious of double level injury then the entire length of the nerve needs to be explored. The ideal scenario for nerve repair is end-to-end coaptation of the nerve stumps.

The procedure of repairing a nerve trunk can be divided into four steps. After the zone of injury is defined, the nerve endings are cut back to healthy fascicles. Then, the nerve ends are approximated keeping in mind the importance of considering the length of the gap and possible tension at the coaptation site. If additional nerve length is required, releasing constricting fascia, dividing adventitia attachments, dissecting any tethering bands, transposing nerves (e.g. ulnar at elbow) and flexing neighboring joints (e.g. wrist for median and ulnar lesions in Zone 5) will mobilize the nerve further. Tensionless repairs have demonstrated superior results. Exceeding 10 % of the resting length of the peripheral nerve has been shown to decrease blood flow to the nerve by 50 % [18]. Tension is assessed intraoperatively to determine the need for grafting. A good rule of thumb is that if nerve ends can be approximated with 8-0 sutures, then grafting is not required.

The next step is the correctly aligned coaptation of the nerve ends. Last step is the maintenance of nerve repair with microsutures (9-0 or 10-0 nylon) which are inserted into the epineurium. Placement of the sutures should avoid malrotation of the nerve ends.

Epineurial repair has been shown to have similar functional results to group fascicular repair in smaller, more distal nerves [19]. Group fascicular repair is preferred in larger nerves where motor and sensory fasicles can be accurately matched (most notably the ulnar nerve below the elbow). The cross-sectional appearance of the proximal and distal stumps should be carefully inspected under high magnification prior to proceeding with the nerve repair.

The accuracy of nerve apposition at the repair site influences the functional restoration. Presently, anatomic axon-to-axon reconnection and normal restoration of function after significant nerve injury remain an unobtainable goal. Electrophysiologically-aided motor- and sensory- fascicle differentiation has been an important tool that facilitates our ability to depict the intraneural composition of sensory and motor bundles prior to nerve coaptation [20]. In 1976, Williams and Terzis [21] introduced single fascicular recordings as an intraoperative diagnostic tool for the management of peripheral nerve lesions in continuity which was a new method of sophisticated intraoperative differentiation between motor and sensory components.

Several histochemical methods have been developed to permit differentiation of motor and sensory fibers. The enzyme carbonic anhydrase

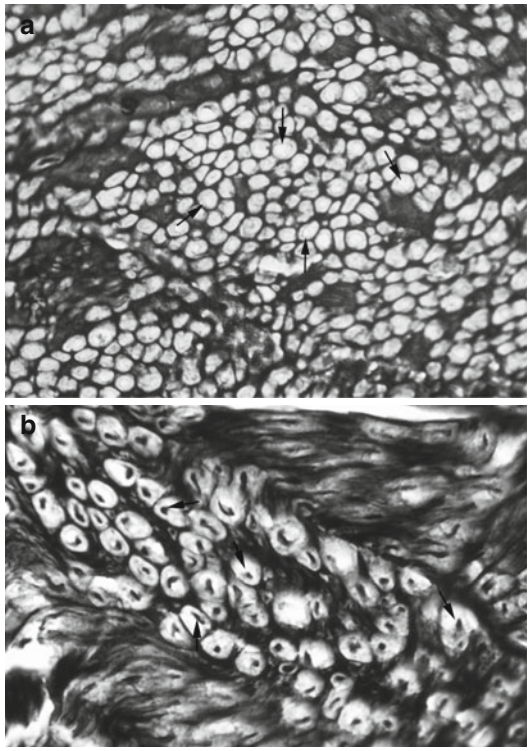


Fig. 1.4 Example of Carbonic Anhydrase staining. (**a**) Cross section of a motor fascicle. Note lack of axonal staining with the carbonic anhydrase (*arrows*). (**b**) Cross section of a sensory fascicle. Note dark staining of the axons (*arrows*)

can differentiate between motor and sensory fascicles of peripheral nerves [22] (Fig. 1.4). The application of this staining method to human peripheral nerve was first described by Riley and Lang in 1984 [22] and later modified for widespread clinical use by Carson and Terzis in 1985 [23]. Although it can provide a convenient method for identifying predominantly sensory versus motor fascicles in cut ends of peripheral nerves, its use depends on the surgeon's experience, available operating time and existence of an experienced laboratory in nerve histochemistry. Acetylcholinesterase histochemistry was also used in conjunction with peripheral nerve surgery, this enzyme in contrast to carbonic anhydrase, is present only in motor fibers [24].

End-to-End-Repair

The surgeon should be familiar with the various techniques available and tailor them to the situation, taking into account which nerve is injured and the level of the injury in the upper extremity. The basic choices include epineurial repair, group fascicular repair, fascicular repair or a combination of those techniques. The goal is to achieve tension free coaptation and proper alignment.

In the epineurial repair, coaptation is achieved by single epineurial stitches in the epineurium along the circumference of the nerve. A perfect superficial alignment can be achieved using epineurial vessels as landmarks, but the internal orientation of fascicular bundles and individual fascicles may not be correct. This method is indicated when one or only few fascicles are injured and is appropriate for distal nerve repairs (digital nerves).

In group fascicular repair, fascicular groups are coapted with single sutures in the perineurium or perifascicular connective tissue which surrounds groups of fascicles. Prior to coaptation, the fascicular groups need to be identified and matched together. In large nerves with multiple fascicles, nerve regeneration can be enhanced by use of this technique.

In fascicular repair, coaptation of individual fascicles is achieved by 10-0 or 11-0 microsutures in the internal epineurium surrounding individual fascicles. This type of repair is not feasible unless it can be performed with minimal tension.

End-to-Side Nerve Repair (Fig. 1.5)

The idea of end-to-side nerve repair was popularized by Viterbo et al. in 1992 [25] after its introduction a century ago [26]. This technique allows for additional muscle reinnervation with minimal detriment to donor-nerve function [25]. Using this technique a neurorrhaphy is created between the proximal end of an injured nerve and the side of an uninjured donor nerve by simple microsurgical attachment at the site of a window (epineurial and/or perineurial window).

The efficacy of end-to-side neurorrhaphy has been established in several rat models. Noah et al. [27] suggested that more axons went through the coaptation site when a perineurial window or partial neurectomy was created in the donor-nerve prior to coaptation vs leaving the perineurium or epineurium intact. Okajima et al.

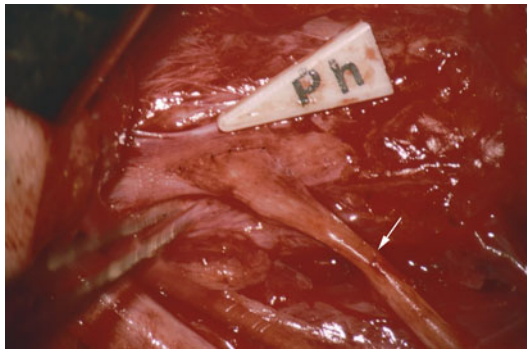


Fig. 1.5 Example of end-to-side nerve repair. Example of an end-to-side neurorrhaphy in an obstetrical brachial plexus case. An epineurial and perineurial window has been made on the phrenic nerve. An interposition nerve graft (*arrow*) is coapted by end-to-side repair at the site of the window. The nerve graft is targeted to neurotize the musculocutaneous nerve (not shown). Because an end-to-side coaptation was used there is no downgrading of the function of the ipsilateral diaphragm

[28] studied the early regenerative response after end-to-side neurorrhaphy and were able to identify increased nodal sprouting proximal to the perineurial window and/or partial neurectomy groups vs the intact epineurium group.

In clinical practice, Terzis [29] used end-to-side neurorrhaphy extensively in order to minimize morbidity from the various extraplexus donors. Thus, only the number of donor fibers needed are taken, such as in partial phrenic or partial hypoglossal transfers, which are used in combination with an end-to-side coaptation via an interposition nerve graft especially in cases of facial paralysis and obstetrical BP reconstruction.

Nerve Grafting (Fig. 1.6)

When tension-free repair is not possible, a suitable alternative must be pursued. The surgical technique employed in these alternatives is similar, whether it be a nerve graft or nerve transfer.

Nerve grafting has long been considered the 'gold standard' for repair of irreducible nerve gaps. The choice of autogenous graft is dependent on several factors: the size of the nerve gap, location of proposed nerve repair, and associated donor-site morbidity.

Before grafting, the proximal and distal nerve stumps must be prepared to normal tissue outside of the zone of injury. In cases of polyfascicular nerve stumps, interfascicular dissection is preferred in order to prepare corresponding fascicular groups. The intraneural topography of both nerve stumps is obtained by means of intraoperative electrodiagnostic studies and carbonic anhydrase histochemistry.

Then, the defect size is measured and the nerve grafts are harvested. The nerve grafts are then tailored so that they bridge corresponding fascicular groups. The proximal end of each graft is coapted to the proximal fascicular group and its distal end to the corresponding distal bundles.

Selection of the graft donors is limited by the availability of donor nerves and the functional and aesthetic deficits created by their harvest. According to Sunderland and Roy [30] the ideal donor-nerve should possess the following characteristics:

1. the sensory deficit should occur in a non-critical area of the body
2. the donor-nerve should possess long, unbranched segments
3. the donor-nerve should easily be accessible and reliably located
4. the donor-nerve should be of overall diameter and possess large fascicles with little interfascicular connective tissue and few interfascicular connections

The commonly used donor-nerves available for grafting are typically the sural nerve, the saphenous nerve, the medial brachial cutaneous nerve and the lateral antebrachial cutaneous nerve.

Vascularized Nerve Grafts (Fig. 1.7)

The first vascularized nerve graft in the upper extremity was a pedicled nerve graft in 1945 by St. Clair Strange for reconstruction of large nerve defect: the ulnar nerve was transferred in two stages to reconstruct the median nerve [31]. Taylor et al. [32] used the superficial radial nerve as a vascularized nerve graft, to repair a large defect of a median nerve.

In 1984, Breidenbach and Terzis [33] defined the blood supply of peripheral nerves that could be used for microvascular transfer and introduced a classification of the blood supply of nerves

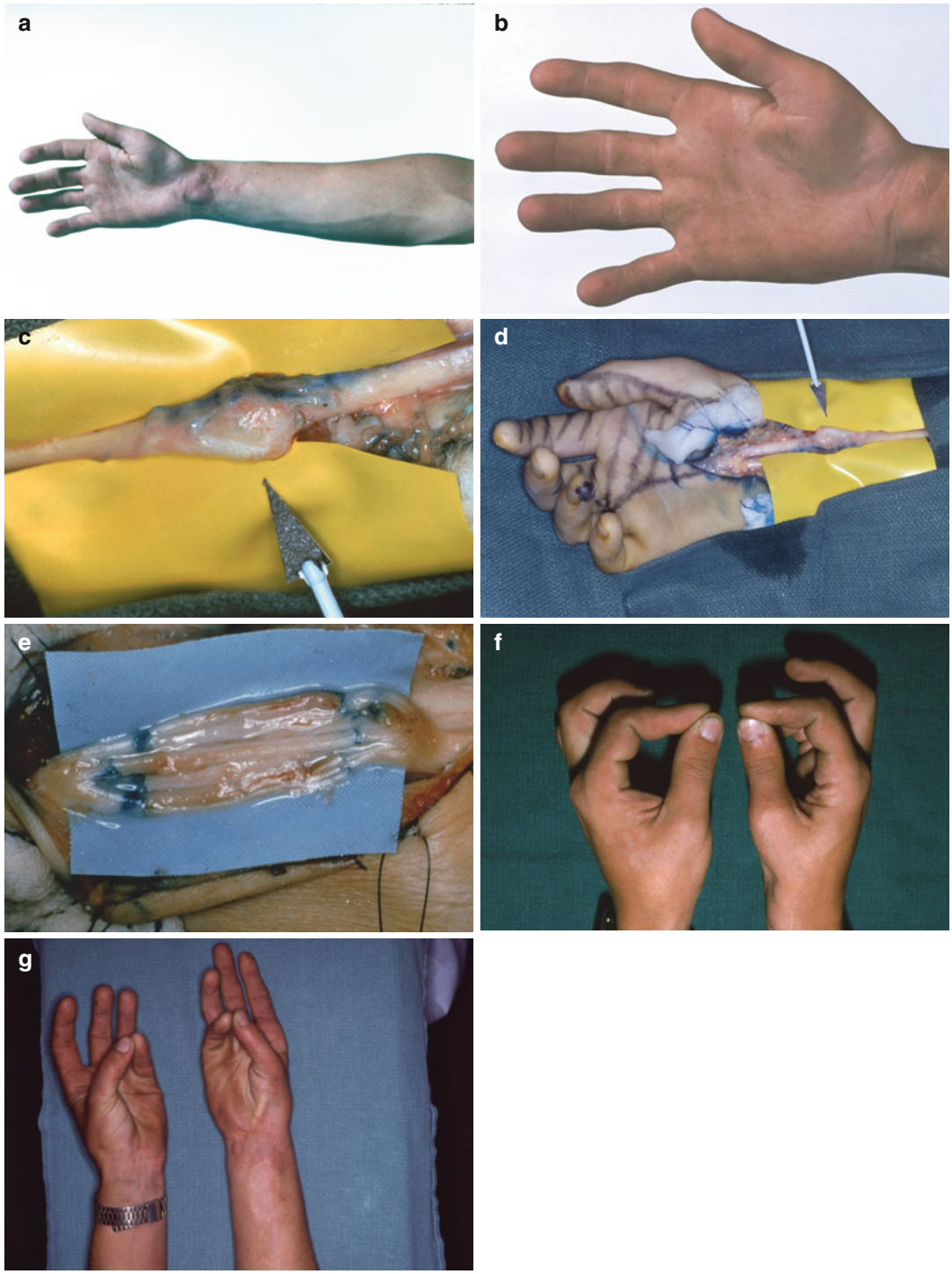


Fig. 1.6 Example of a case treated with interposition nerve grafting. A 19 year old boy was involved in an accident in which he sustained a glass laceration of the volar aspect of his right dominant wrist. He presented 18 months later to our Center with complete anesthesia of the thumb, index and radial side of the middle finger and had no thumb opposition (**a**, **b**). On exploration, a large median nerve neuroma was present (**c**, **d**). The neuroma was excised and the defect was reconstructed with five interposition sural nerve grafts (**e**). Eight months later, he also had opponensplasty which involved transfer of the sublimis tendon from the ring finger to the thumb to augment opposition. Upon follow-up the patient showed adequate pinch (**f**) and strong thumb opposition (**g**). Sensory return to the radial side of his hand has been satisfactory, enabling him to return to his previous work

based on the number of dominant vascular pedicles.

The clinical indication for a vascularized nerve graft is a scarred recipient bed that will not support a nonvascularized nerve graft. In cases of long gaps, vascularized nerve grafts can be placed in association with nonvascularized nerve grafts to cover the cross-sectional area of the injured nerve. The obvious advantage of this technique is the ability to provide immediate intraneural perfusion in a poorly vascularized bed and to reconstruct large nerve defects.

The use of vascularized nerve grafts is particularly important in BP surgery. In cases of avulsion of the C8 and T1 roots, the ulnar nerve should be used as a vascularized nerve graft for ipsilateral plexus reconstruction or as a cross-chest nerve graft from the contralateral C7 root for neurotization of the denervated upper extremity [34] (Fig. 1.8).

Breidenbach and Terzis [35] first reported that the ulnar nerve can be transferred in its total length on the superior ulnar collateral vascular pedicle (Fig. 1.9). Terzis subsequently reported a series of 151 vascularized ulnar nerve grafts for posttraumatic BP palsy patients [34]. According to this study, pedicled or free vascularized ulnar nerve grafts achieved superior results compared to those obtained with conventional nerve grafts.

Technique

Using this technique, the ulnar nerve with its supplying vascular pedicle is transferred as a pedicle or free vascularized nerve to bridge several nerve defects. The vascular pedicle is anastomosed to an artery and a vein of the recipient site and subsequently the nerve coaptations take place. The vascularized ulnar nerve graft is folded into segments maintaining their vascular connections according to the technique proposed

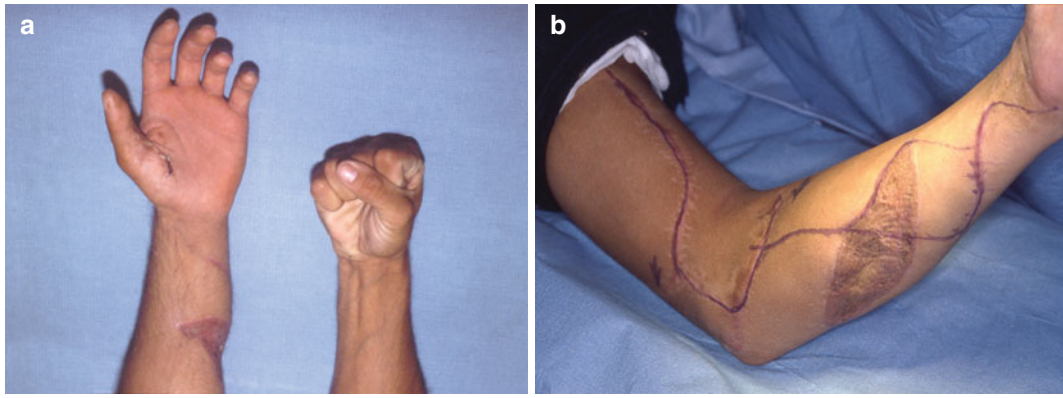
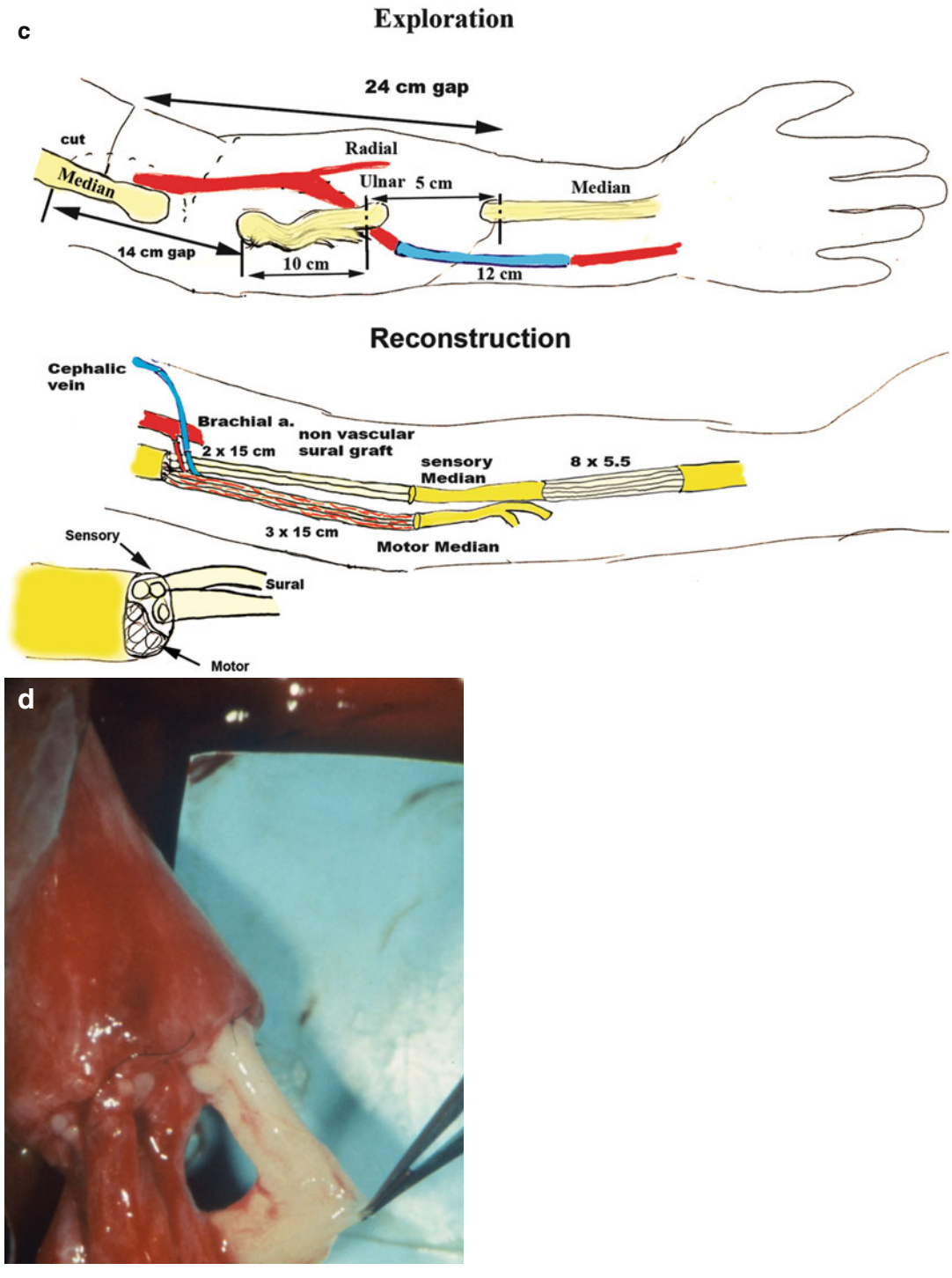


Fig. 1.7 Example of a case with Vascularized Nerve Grafts. This is a 23 year old male who was involved in a boating accident in which the propeller of a motor boat ran over his left arm. He was taken emergently to a local hospital where he was noted to have severe neurovascular injuries as well as tissue loss of the left forearm. He received elsewhere emergency revascularization of his left extremity with the use of saphenous vein grafts. He also had multiple levels of nerve injuries of the left ulnar and median nerve. Preoperative view of the patient (**a**, **b**). Three months later, he underwent reconstruction of his left median nerve which was transected at four levels (**c** above). The sensory part of the superficial and deep peroneal nerves based on their common vascular supply was harvested and used to reconstruct the motor portion of the median nerve (3 × 15 cm, one deep and two superficial peroneal nerve grafts). Nonvascularized sural nerve grafts were used to reconstruct the sensory portion of the median nerve (2 cables × 15 cm proximally and 8 cables × 5.5 cm distally: **c** below). Close-up of the proximal coaptation: vascularized nerve grafts on the left, nonvascularized sural nerve grafts on the right (**d**). Seven months after the injury he underwent reconstruction of the left ulnar nerve utilizing vascularized saphenous nerve graft (1 cable × 30 cm) for the motor portion of the ulnar nerve and sural nerve graft for the sensory component of the ulnar nerve (**e**). Four years postoperatively, we can see very good results. Powerful finger flexion, thumb opposition, and intrinsic function (**f–i**). He can easily pick up a can of soda (**j**) and has never had any morbidity in the donor extremity (**k**) (Requested permission from: Terzis and Kostopoulos [67])



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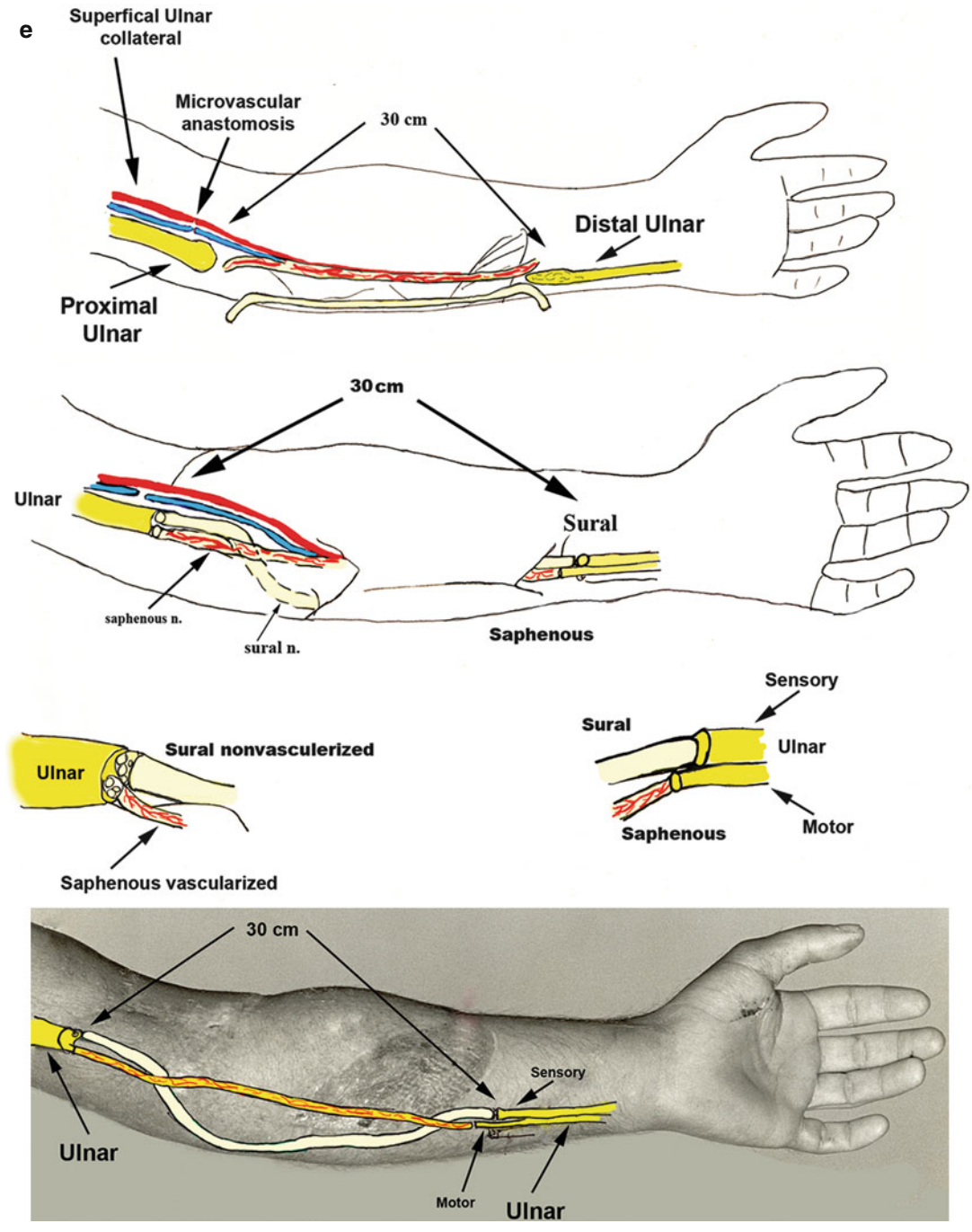

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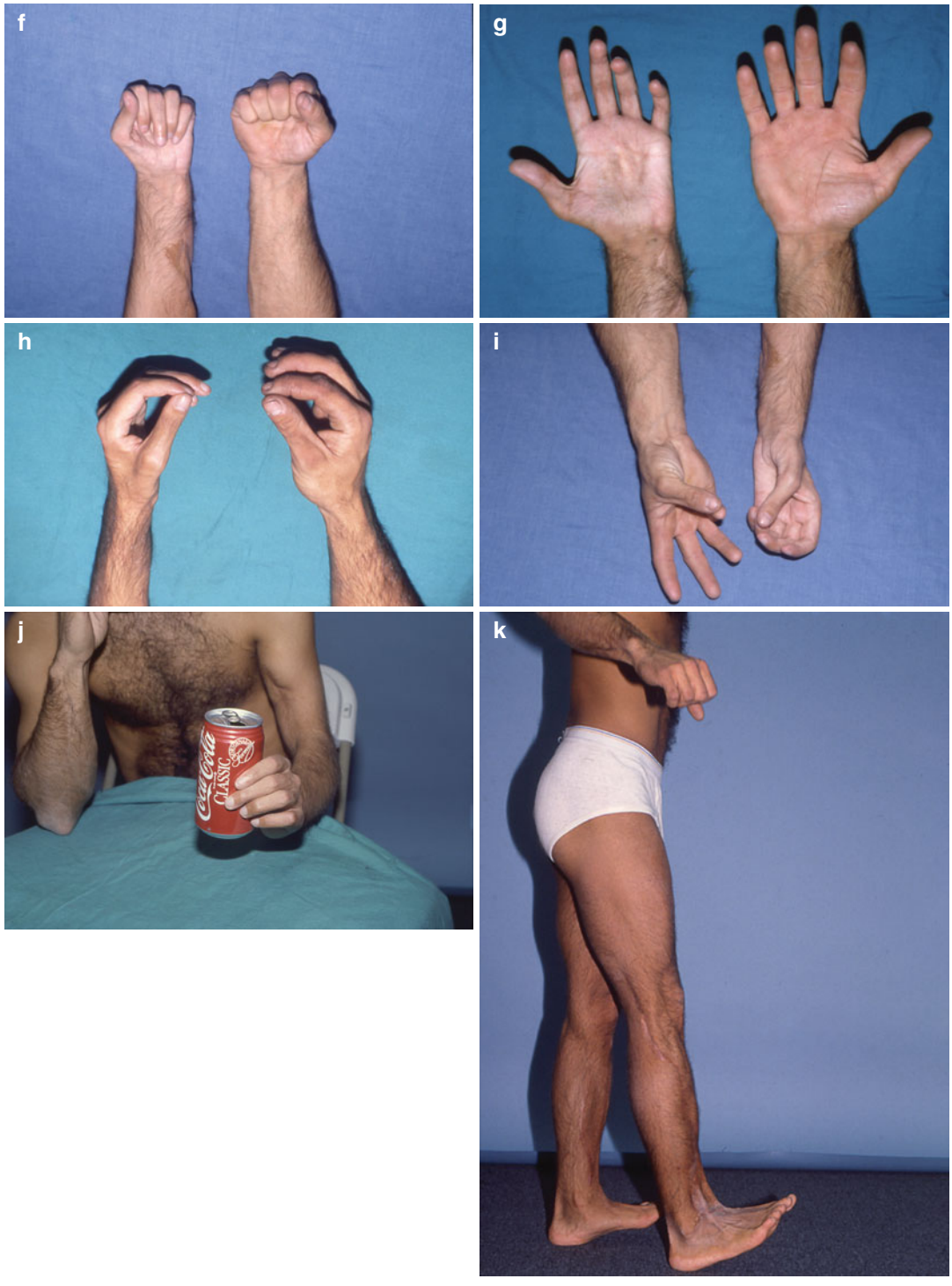

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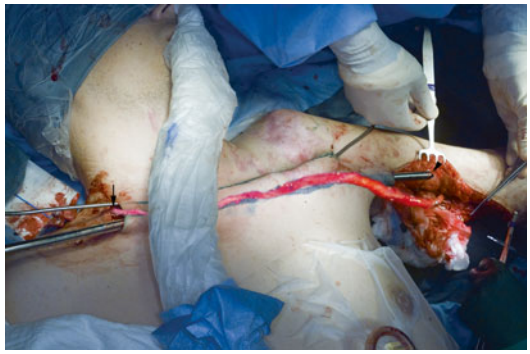

Fig. 1.8 Example of a cross chest vascularized ulnar nerve graft. Cross chest vascularized ulnar nerve graft prior to tunneling. The proximal ulnar will be coapted to the anterior division of the right C7 root. The distal ulnar nerve will be coapted to the median nerve of the left paralyzed extremity. *Arrow* points to the metal "passer" that will be used to transfer the nerve across the chest

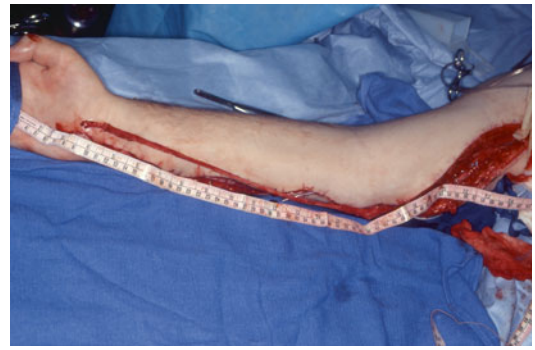

Fig. 1.9 Example of ulnar nerve harvested as a VNG next to the arm. Exploration of the right vascularized ulnar nerve graft prior to microvascular transfer. The entire length of the nerve receives its blood supply from the superior ulnar collateral vascular pedicle. Terzis' method for the use of the free vascularized ulnar nerve for ipsilateral intraplexus reconstruction. The epineurium is transected longitudinally without compromising the longitudinal epineurial blood supply and the fascicles are transected transversely. The blood supply is maintained through the folded epineurium

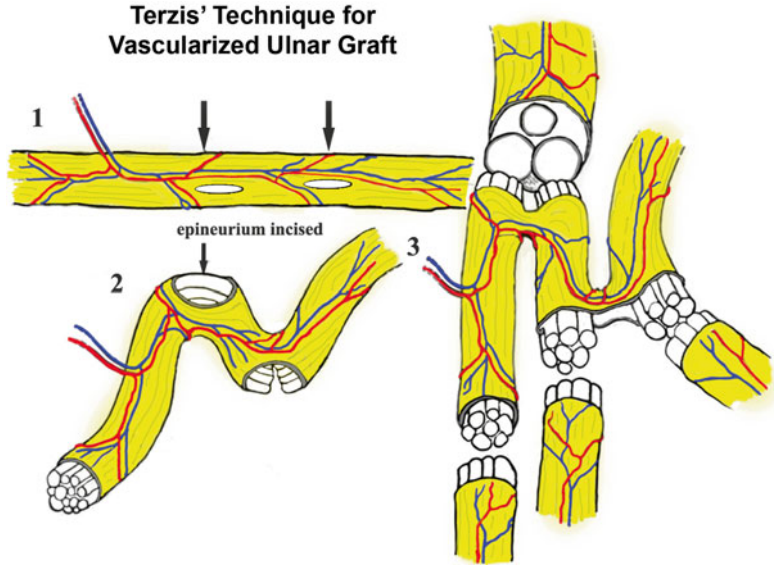

Fig. 1.10 Terzis' technique of using the vascularized ulnar nerve for ipsilateral BP reconstruction. Terzis' technique of folding the vascularized ulnar nerve graft into segments while maintaining its blood supply by preserving the integrity of epineurial vessels

by Terzis and Kostopoulos [34] (Fig. 1.10). In this situation, the longitudinal blood supply of the epineurium of the ulnar nerve is preserved while the intraneural contents are transected to address the bridging nerve defects, thus maintaining excellent blood supply throughout the vascularized ulnar nerve graft. In more distal lesions, vascularized fascia can be used to improve the blood supply of the underlying bed by enveloping the nerve reconstruction (Fig. 1.11).

Ulnar Nerve

Cases of global plexopathy with avulsion of the lower roots and rupture of the upper roots provide the best indication for using the ipsilateral ulnar as a vascularized graft for BP reconstruction. The ulnar nerve can be harvested on the superior ulnar

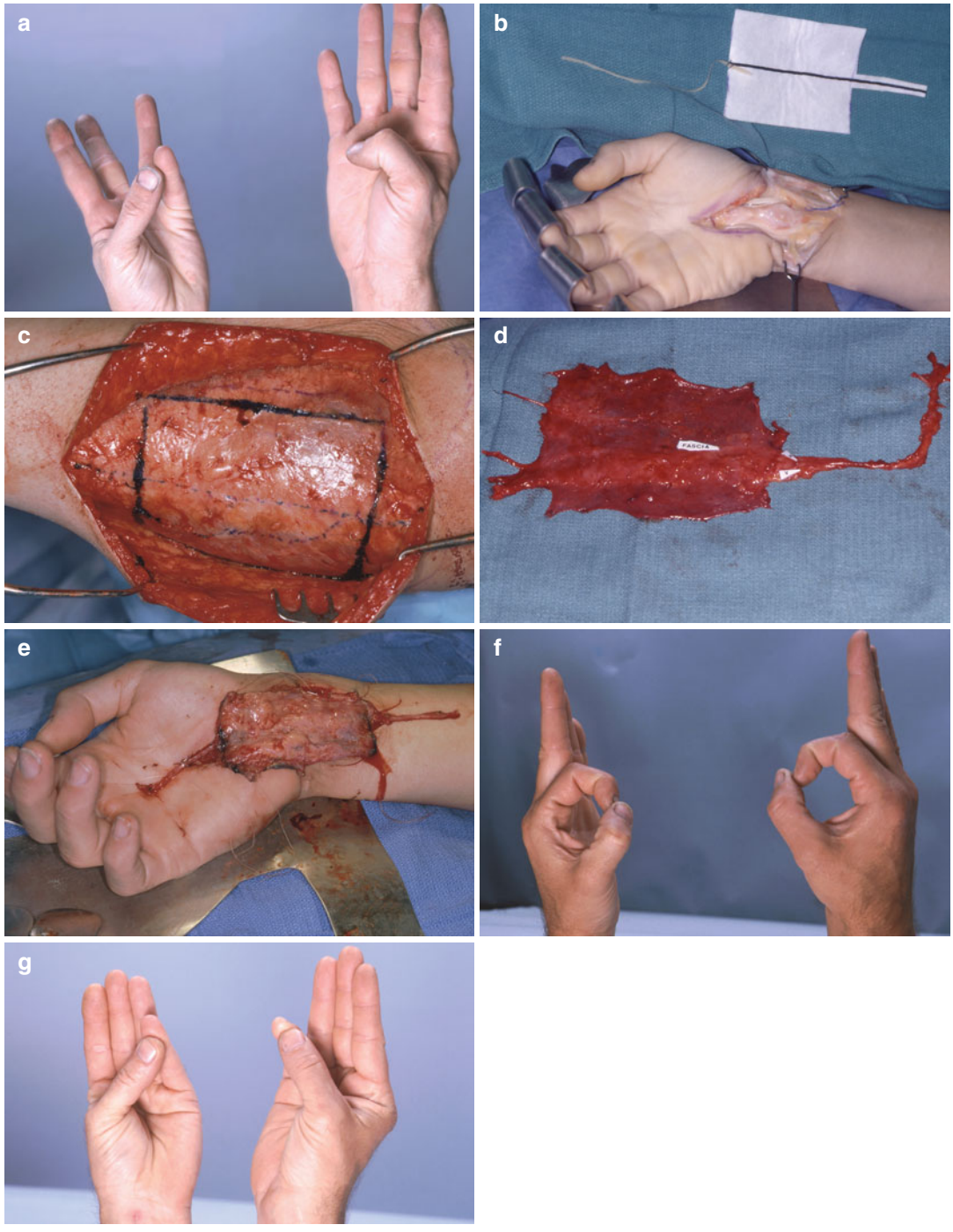

Fig. 1.11 Example of a vascularized fascia to improve the blood supply of nerve grafting in an unfavorable recipient bed. (**a**, **b**) Patient with right carpal tunnel syndrome and pain secondary to severe crush injury of the right distal forearm and hand. Note lack of opposition of the right thumb (**a**). Upon exploration a large neuroma in continuity of the median nerve was apparent (**b**). Extensive microneurolysis under high magnification of the operating microscope took place along with the transfer of a vascularized posterior calf fascia to envelop the nerve at the wrist. (**c**) The vascularized posterior calf fascia has been outlined in the non-dominant lower extremity. (**d**) The vascularized fascia flap after harvesting. (**e**) The vascularized fascia on the right wrist prior to microvascular anastomoses. (**f**, **g**) On the last follow-up, note excellent pinch and opposition. In addition, the patient is pain free and has returned full time to his job as a jeweler

collateral vascular pedicle. The superior ulnar collateral artery is sufficient to maintain the blood supply for the total length of the ulnar nerve (Fig. 1.9).

If used for ipsilateral BP reconstruction, the nerve is transected in the appropriate segments to bridge the nerve defects always preserving the epineurial blood supply (Fig. 1.10).

If used for neurotization of the median nerve from the contralateral C7 (cC7) root then the nerve is harvested as a free vascularized cross-chest graft (Fig. 1.8) and the superior ulnar collateral vascular pedicle is anastomosed to the transverse cervical vessels of the unaffected side prior to nerve coaptations of the proximal ulnar end with the anterior division of the cC7. Subsequently, the distal part of the ulnar nerve is coapted to the median nerve on the affected side.

Sural Nerve

Vascularized sural nerve graft for extremity nerve reconstruction should be used as a free vascularized nerve graft based on the sural artery, if available, or with an arterialized saphenous vein that is transferred in conjunction with the sural nerve (Fig. 1.12).

Saphenous Nerve

For BP injuries, the indications are the same as use of vascularized ulnar. Moreover, it can be used for more distal injuries when multiple major nerves are injured or the nerve gaps are too long.

Clinical Pearl – Ideal Donor Nerve

the sensory deficit should occur in a non-critical area of the body

the donor-nerve should possess long, unbranched segments

the donor-nerve should easily be accessible and reliably located

the donor-nerve should be of overall diameter and possess large fascicles with little interfascicular connective tissue and few interfascicular connections

Nerve Transfers

A nerve transfer recruits redundant nerve fascicles from a donor nerve to innervate critical motor or sensory nerves close to their target end-organs. Traditionally nerve transfers in the upper extremity have been used for BP injuries where there are limited proximal intraplexus motor donors. However, nerve transfers lately are starting to be used for a variety of peripheral nerve injuries.

According to Dvali et al. [36] the indications for nerve transfers in the upper extremity are:

1. BP avulsion injuries
2. Proximal nerve injuries which require a long distance for regeneration
3. Major limb trauma with associated loss of nerve tissue
4. In patients with long denervation time or in older patients
5. Avoidance of re-exploring an area of previous injury because of potential damage to critical structures

The ideal motor donor nerve should carry a large number of axons and be near to the denervated target. Moreover, it is preferred for a motor donor nerve to have limited donor-site morbidity and to innervate synergistic muscles.

According to Rosenfield et al. [16] the advantages of nerve transfers are:

1. There is preservation of muscle structure due to the fact that reinnervation must be made prior to 18 months to avoid irreversible fibrosis
2. There is no need for nerve grafting in order to bridge the nerve gap and thus avoidance of its potential complications
3. Potential mismatching is avoided, as nerves with dedicated function are selected as donors (eg. the common digital nerve from the fourth webspace to the first webspace to provides sensation to the ulnar side of the thumb and radial side of the index finger).

Lesions in Continuity

Intraoperative Diagnosis and Treatment

The exact level, type, and extent of the nerve lesion can be accurately determined only during surgical

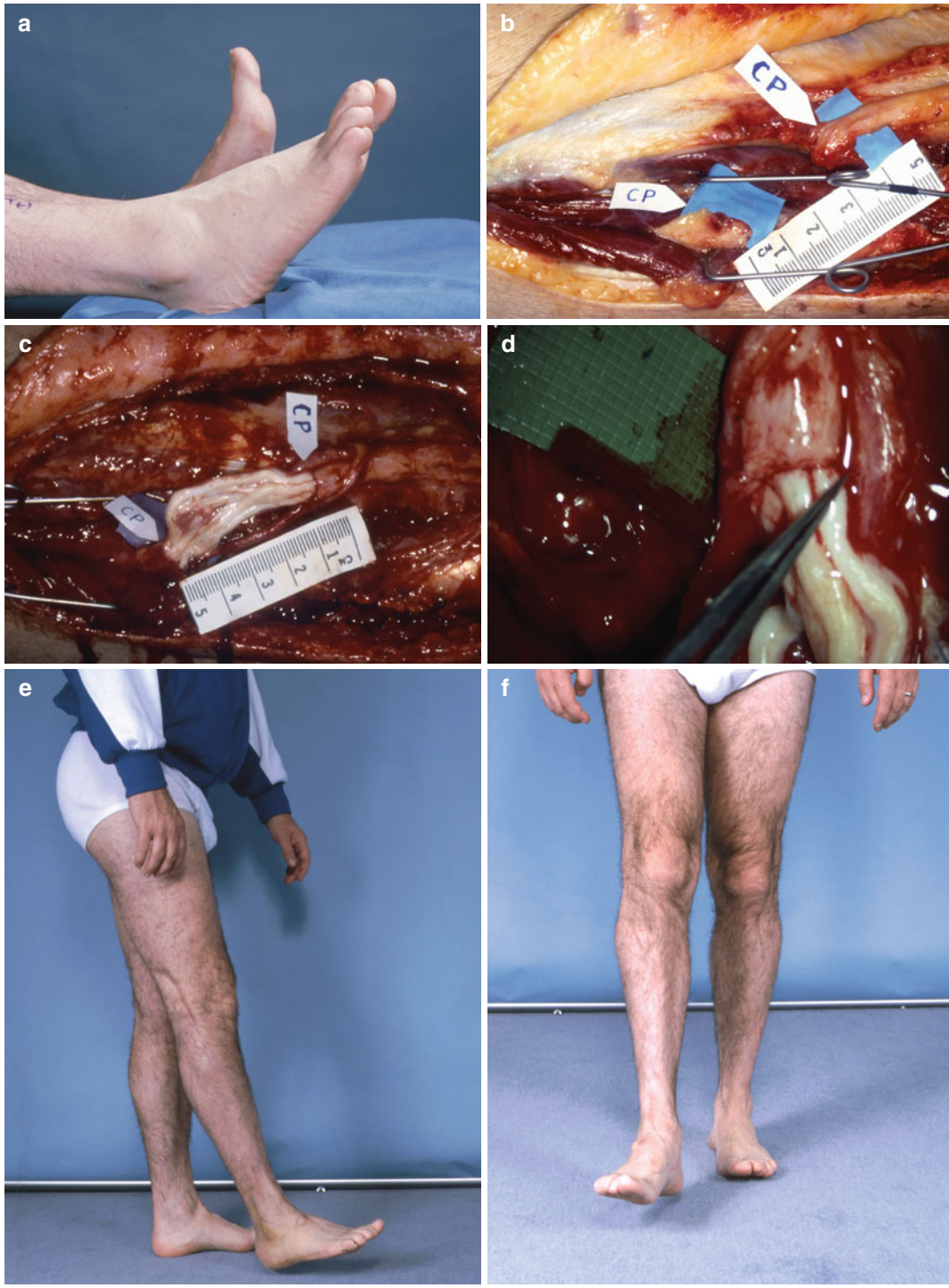

Fig. 1.12 Use of ipsilateral sural as a vascularized nerve graft for lower extremity nerve repair reconstruction. (**a**–**f**) A 41 year old male, who suffered a propeller injury and sustained a laceration at his right popliteal fossa. He presented to our Center with a right foot drop (**a**). Intraoperative view of the severed peroneal nerve stumps and the created five centimeter defect (**b**). Intraoperative view of the common peroneal nerve reconstruction using a combination of vascularized and nonvascularized sural nerve grafts (**c**, **d**). Patient at his last follow-up demonstrates excellent dorsiflexion of his right foot and is walking without a splint (**e**, **f**)

Clinical Pearls – Indications for Nerve Transfer

BP avulsion injuries

Proximal nerve injuries which require a long distance for regeneration

Major limb trauma with associated loss of nerve tissue

In patients with long denervation time or in older patients

Avoidance of re-exploring an area of previous injury because of potential damage to critical structures

exploration. Intraoperative electrophysiologic recordings are extremely important for providing direct evidence of the extent of neural injury. Intraoperative monitoring can be useful to facilitate the decision to repair, graft, or resect nerve tissue. However, while it does not substitute for preoperative electrodiagnostics, it is helpful to monitor nerve function, guide dissection, and identify neural from scar tissue [37].

Stimulating and recording electrodes are placed at least 5 cm apart on the nerve proximal and distal to the lesion respectively. A ground electrode is placed at the wound's edges. Then, compound action potential (CAP) are recorded across the injury site and displayed by a computerized device and disclose the degree of conduction across the tested nerve. The compound action potential will appear only if the nerve is functioning [20].

If a CAP is present, extensive neurolysis with or without interfascicular dissection takes place. If the nerve, following release of the constricting epineurium, still 'feels' hard to palpation, interfascicular neurolysis is performed. Intraoperative bulging of individual fascicles on release is a good prognostic sign, as it signifies that functional restoration will occur. Lack of bulging following interfascicular neurolysis is a poor indicator for recovery.

If a CAP is present but it is diminished in height and width and the nerve is in continuity but hard to palpation and after epineuriotomy and interfascicular dissection it is apparent that the perineurial integrity is lost in some of the fascicles but it is present in others, then under magnification, the involved fascicles should be resected and grafted, while the others, with perineurial integrity, should be preserved [21].

In cases where no CAP is recorded or there is no clinical evidence of sensory and motor function on preoperative assessment, interposition nerve grafts are used to reconstruct the injured nerve. In early cases (≤3 months) or secondary to sharp laceration injuries without appreciable loss of neural tissue, after proximal and distal mobilization of the nerve stumps an end-to-end epineurial repair is performed.

In cases of BP injuries, the presence of sensory action potentials and normal conduction velocities in a flail and anesthetic extremity implies root avulsion. Furthermore, intraoperative electrodiagnostic studies are useful in order to verify a suspected avulsion of a root or to determine whether resection of a neuroma and interposition nerve grafting should be performed.

An additional preoperative electrophysiological study for the investigation of patients with BP injury is the Lamina test, which was introduced by Liberson and Terzis [38] in 1987.

During this test, small volleys of electrical stimulation are applied on each exiting root to determine whether the patient perceives the area of the dermatome innervated by this root. A positive response would be strong evidence against avulsion.

Furthermore, the response of the vital signs of the patient to the resection of a neuroma can also indicate connectivity of the corresponding root to the spinal cord. If the vital signs of a lightly anesthetized patient rise suddenly during resection of the neuroma, this indicates that the root is in continuity with the spinal cord and not avulsed [17].

Terzis et al. [39] developed an intraoperative assessment measurement tool of the severity of a BP injury and labeled it 'Severity Score'. Each root, if intact, is given five points. A normal BP severity score equals 25. A globally avulsed BP has a score of 0. The lower the severity score, the worse the prognosis.

Avulsion of the BP roots always carries the worst prognosis and makes functional restoration in the paralysed upper extremity much more

challenging. A variety of extraplexus donors should be recruited in such cases to reconstruct the distal plexus elements [17].

One or Two Root Avulsions

(a) If the C5 and/or C6 are avulsed, reconstruction of the shoulder and elbow function can be achieved by means of:
   1. Distal spinal accessory nerve transfer to the suprascapular nerve
   2. If the three lower roots (C7, C8 and T1) are intact, intraplexus donors (ipsilateral C7, branch to the long head of the triceps) are used for biceps and deltoid neurotization. If the three lower roots are also ruptured, three intercostal nerves can be used for direct biceps neurotization.

(b) If the lower two roots (C8 and T1) are avulsed, C8 and T1 roots should be neurotized from C5, C6, or C7 in infants only. In adults, hand function is unattainable and thus the ulnar nerve can be harvested as a free or pedicled vascularized nerve graft in order to neurotize the musculocutaneous, axillary, median, and/or radial nerves [34, 40].

Three Roots Avulsion

(a) When the three upper roots (C5, C6, and C7) are avulsed, reconstruction is as follows:
   1. Distal spinal accessory nerve transfer to reconstruct the suprascapular nerve
   2. Intercostal nerves transfer for reconstruction of the axillary and the nerve to triceps. Fascicles of the ipsilateral ulnar nerve can be used for musculocutaneous nerve neurotization (Oberlin's transfer) [17, 41].

(b) If C7, C8, and T1 roots are avulsed, the force of this injury may affect the upper roots (C5, C6) as well, which may be ruptured. In such cases, the distal part of the accessory nerve is transferred to the suprascapular nerve whereas the ipsilateral ulnar nerve is used as a vascularized free or pedicled nerve graft to connect (in an end-to-end fashion) C5 and C6 roots with musculocutaneous, median, axillary, and/or radial nerves

Four Roots Avulsion

In BP injuries with four roots avulsion (C6, C7, C8, and T1), reconstruction is as follows:
   1. If the C5 root is well-developed (i.e. the BP is prefixed), the same reconstructive plan is used as with three root avulsions. If C5 is small, it is usually dedicated to neurotization of the musculocutaneous nerve via sural nerve grafts
   2. Distal spinal accessory nerve neurotises the suprascapular nerve
   3. Intercostals nerves are used for neurotization of axillary and triceps nerves
   4. Selective contralateral C7 root transfer [42, 43] is used as follows: The anterior division is coapted to a vascularized ulnar nerve graft for neurotization of the median nerve on the affected side while the posterior division is coapted to two saphenous cross-chest grafts which are 'banked' for future free muscles for hand reconstruction.

Global Avulsion

In case of global avulsion plexopathy, all reconstructions are carried out from extraplexus donors as follows:
   1. Transfer of the distal part of the accessory nerve to the suprascapular nerve
   2. Intercostals nerves are used for neurotization of axillary and musculocutaneous nerves
   3. Selective cC7 root transfer is used as follows: The anterior division is coapted to a cross-chest vascularized ulnar nerve graft for median nerve neurotization while the posterior division is coapted to two-cross-chest saphenous nerve grafts for triceps neurotization and as a 'banked' nerve for future free muscle for finger extension.

Surgical Techniques

Intraplexus Donors

The use of intraplexus motor donors is always preferred over extraplexus motor donors.

Intraplexus donors have a greater number of axons than the extraplexus donors and, when available, there is less need of postoperative reeducation. Generally, outcomes are superior if proximal healthy roots are available for neurotization of distal targets.

C5 Root Transfer

This is the strongest motor donor, and if there is no avulsion, the proximal part of the ruptured root can be used as a motor donor for multiple neurotizations with interposition nerve grafts. In normal conditions, the C5 root contains about 16,000 myelinated axons. If C5 is ruptured, but the proximal stump is in continuity with the spinal cord, with simultaneous lower root avulsions, then during the initial BP reconstruction, the ipsilateral ulnar nerve is used as a free or pedicled vascularized graft to reconstruct the musculocutaneous, the median, and, on occasion, the radial nerve.

Technique

The patient is placed in the supine position, with the head turned away from the operative side. A curved incision along the posterior border of the sternocleidomastoid muscle is carried out and the supraclavicular plexus is identified between the anterior and the middle scalenes muscles. The phrenic nerve is identified and stimulated in order to assess its integrity. Then, dissection proceeds posteriorly and the C5, C6 roots are identified.

Care should be taken to isolate the C5 root and preserve it, except in cases in which it is ruptured; in these cases, if the distal stump cannot be identified, the proximal stump should be used as a donor for neurotization procedures.

C7 Root Transfer

The selective ipsilateral or contralateral C7 root transfer for neurotization of high priority targets in BP reconstruction has been introduced by Terzis since 1991 [42, 43]. Due to extensive overlap among the nerve fibers derived from the upper and lower plexus, no single muscle of the upper extremity is solely innervated by the C7 nerve root [44]. The procedure involves extensive intraoperative mapping of the intact C7 root. Subsequently, the selective use of the anterior division fibers are targeted for contralateral flexor target neurotization while the posterior division motor fibers are destined for reinnervation of extensor targets in the contralateral paretic extremity.

When the upper plexus roots (C5 and C6) are avulsed from the spinal cord, but C7 root is preserved, reconstruction of shoulder and elbow function can be achieved by using the ipsilateral C7 root for higher priority targets. In cases with global BP avulsion, the contralateral C7 root is used as a motor donor.

Technique

The C7 root in the intact BP is explored to the level of its divisions. After longitudinal epineuriotomy and using intraoperative electrical stimulation, each bundle within each division of the C7 is mapped [42, 43]. The intraoperative mapping of the components of each C7 division is a mandatory step. Bundles that supply wrist extensors are preserved and never sacrificed. Bundles supplying the pectoralis major in the anterior division and the latissimus dorsi and triceps muscles in the posterior division are isolated with vessel loops. The former are used as motor donors for contralateral flexors and the latter for contralateral extensors.

Extraplexus Nerve Donors

In BP root avulsion injuries, a variety of extraplexus donors are recruited to reconstruct the distal plexus components. Extraplexus donor nerves for distal target neurotization include the cervical plexus motors, the spinal accessory nerve, intercostal nerves, the phrenic nerve, or the cC7 root.

Spinal Accessory Nerve Transfer

The spinal accessory nerve (XI) is a pure motor nerve, which innervates the sternocleidomastoid and trapezius muscles. Proximity of the XI to the suprascapular nerve allows direct approximation. Among other extraplexus motor donors, the

distal XI has an advantage because it is a pure motor nerve with functional characteristics similar to those of the suprascapular nerve and the neurotization can take place directly without a graft. In order to minimize trapezius muscle denervation, the nerve is transected distally after it gives off two proximal branches. At this level, the XI contains about 1,300–1,600 myelinated nerve fibers [13].

Technique

With the patient's neck turned away from the affected side, a curvilinear neck incision is made along the posterior border of the sternocleidomastoid muscle. The XI nerve can be found as it emerges along the lateral border of the sternocleidomastoid muscle, cranial to the C4 spinal nerve. The nerve stimulator confirms its identity. The transverse cervical vessels comprise a landmark for detection of the spinal accessory nerve on the anterior surface of the trapezius muscle. After these vessels are identified, a nerve stimulator is used to identify the distal part of the XI, which can be found at a mean distance of 5.2 cm from the midpoint of the clavicle along the anterior border of the trapezius.

The suprascapular nerve can be found by palpation at the level of the suprascapular notch. After identification of the suprascapular nerve, a longitudinal epineurotomy is routinely carried out with the diamond knife to decompress it in a proximodistal fashion to the level of the scapular notch.

The terminal branches of the XI are divided deep posteromedially and moved to the supraclavicular fossa and directly coapted to the suprascapular nerve. In the adult plexopathy patient the results from suprascapular nerve neurotization are significantly better if the transfer is direct without an interposition nerve graft [13].

Intercostals Nerve Transfer

Yeoman and Seddon [45] first described intercostal nerve (ICNs) transfer for BP reconstruction. ICNs are the ventral primary rami of spinal nerves T2–T11. ICNs from T7–T11 supply the muscles and skin of the anterior abdominal wall, and theoretically carry a higher number of motor axons than the upper intercostal nerves. An ICN contains less than 1,200–1,300 myelinated fibers, of which only 40 % are motor fibers. ICNs are satisfactory donors for a variety of transfers; for neurotization of musculocutaneous nerve at least three ICNs need to be used [39].

Technique

Exposure of the ICNs is achieved by elevating the periosteum of the corresponding rib. After exposure, stimulation of the motor branches takes place and the nerve is dissected proximodistally up to the level of the costochondral junction, and posteriorly to the posterior axillary line. Once all the ICNs are prepared, they are passed through a subcutaneous tunnel to the ipsilateral axilla and coapted in an end-to-end fashion to the nerve supplying the target muscle. ICNs can not be used for neurotization of both triceps and biceps as crippling co-contraction will result in the adult which should be avoided.

Phrenic Nerve Transfer

The phrenic nerve originates from the C4 and C5 roots. The phrenic nerve has mainly been used for musculocutaneous nerve neurotization. At our center, phrenic nerve neurotization has been used when the nerve is ruptured and the distal part cannot be found. In obstetrical BP palsies the phrenic nerve is used in an end-to-side manner through a perineurial window so there is no downgrading of ipsilateral function of the diaphragm [46] (Fig. 1.5).

Phrenic nerve contains about 1,300–1,600 myelinated nerve fibers. Before the phrenic nerve is considered for transfer, diaphragm and pulmonary function must be evaluated. Moreover, the entire phrenic nerve should rarely be sacrificed in a patient who has concomitant intercostal nerve harvesting, thus harvesting phrenic motor axons should be done through an end-to-side coaptation.

Technique

Following a supraclavicular approach the phrenic nerve is easily identified, lying on the anterior surface of the anterior scalenus muscle. Under

high magnification, perineurial windows are performed with partial neurotomies. Interposition nerve grafts are brought in the operating field and these are coapted with the phrenic nerve in an end-to-side fashion.

Ulnar-to-Musculocutaneous Nerve Transfer

Oberlin et al. [41] transfer of one or more ulnar nerve fascicles that were destined for the flexor carpi ulnaris to the biceps branch of the musculocutaneous nerve. This is performed to restore elbow flexion in patients who have an irreparable upper trunk injury or avulsion, and an intact lower trunk. Careful selection of ulnar nerve fascicles using intraoperative nerve stimulation enables one to perform this transfer without a donor motor deficit. The main advantage of this technique is the rapid motor recovery time because the transfer is performed so close to the target muscle without using an interposition nerve graft.

Technique

A longitudinal 10 cm incision is performed on the anteromedial aspect of the arm, starting 10 cm caudal to the acromion. The musculocutaneous nerve is identified between the biceps and the coracobrachialis muscles, followed distally to identify the nerve to the biceps. The ulnar nerve is approached at the same level and is identified by means of electrical stimulation.

The branches to the biceps are traced proximally where they usually coalesce into a single motor branch within the parent musculocutaneous nerve, and then transected. The distal part of the branch to the biceps is then rotated medially toward the previously dissected ulnar nerve. The intraoperative mapping of the components of the ulnar nerve is a mandatory step. Bundles supplying the flexor carpi ulnaris are isolated with vessel loops. These are used as motor donors for the musculocutaneous neurotization.

The chosen fascicles are separated from the rest of the ulnar nerve over a distance of 2 cm and are divided distally. The fascicles are then turned laterally and superiorly and are sutured to the nerve to the biceps under an operating microscope.

Rehabilitation

It is advisable that a nerve repair, a nerve reconstruction by interposition nerve grafts or nerve transfers are protected by immobilization, which lasts up to 6 weeks depending on the location of the nerve injury and the type of the nerve repair that was performed. Immediately, after completion of the nerve repair, a custom-made brace is applied to the patient. This brace keeps the arm abducted 45° in anterior flexion and with the elbow flexed (Fig. 1.13). The brace is removed after 6 weeks, and then a sling is applied on the patient's operated extremity for 4 weeks. This custom-made brace is applied for either brachial plexus reconstruction or

Fig. 1.13 Photo of Patient with brace (arm in anterior flexion and elbow flexed). Example of a custom made brace, applied to the patient after the nerve reconstruction

nerve repairs to the arm or forearm. In cases of digital nerve reconstruction a hand splint in the position of function (20° degrees of wrist extension, metacarpophalangeal (MP) joints at 90° degrees and intephalangeal (IP) joints in extension) is applied for 6 weeks in order to immobilize the fingers and thus preserve the nerve coaptations.

After immobilization, rehabilitation is initiated to achieve full passive and active range of motion. The rehabilitation goals in the early postoperative period are to gain full passive range of motion and to avert joint stiffness. Physical therapy with passive range of motion is started with the removal of the brace. Local application of ultrasound and massage and slow pulse stimulation are initiated at 6 weeks.

The slow pulse stimulation will be ongoing for at least a period of 2 years in cases of BP reconstruction (Fig. 1.14) or a shorter period after isolated peripheral nerve repair. The elongation of the outgrowing axons is followed by the advancing Tinel's sign, which is recorded in each follow-up visit.

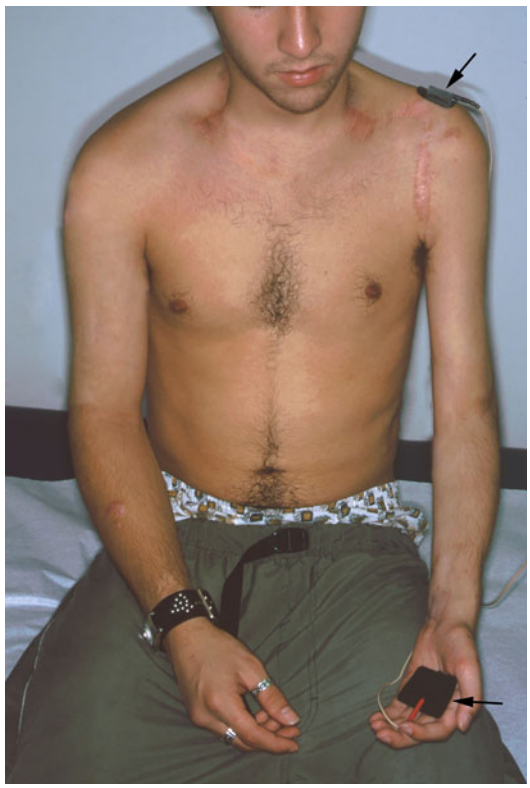

Fig. 1.14 Photo of a Patient fitted with a slow pulse stimulator. Example of a patient undergoing slow pulse stimulation. Note one "pad" is placed on the shoulder and the second on the palm (*arrows*)

Later-stage rehabilitation is focused on motor and/or sensory re-education. An effective relearning process is probably highly influenced by the motivation and compliance of the individual patient. In addition, time is spent to counsel the patient to return to his or hers previous occupation in a part-time basis or back to school during the lengthy period of nerve regeneration and functional restoration.

Outcomes

The interpretation of results of peripheral nerve and BP reconstruction has always been difficult. This is due to several reasons, including the lack of standardization and staging owing to varying degrees of nerve injury which sometimes may involve multiple levels and the lack of consensus as to the best reconstructive approach in patients with middle and high level injuries.

Several factors, such as the patient's age, the etiology and level of the lesion, associated injuries, denervation time, the length of the nerve defect, the type of repair and the surgeon's experience can influence the prognosis following nerve repair. There is no evidence which correlates smoking history with nerve recovery. Application of the principles of nerve repair (magnification, minimal tension, meticulous atraumatic technique, and experienced surgeon) can enhance the chances for a successful result.

The type of nerve repair depends on the nature of the lesion. The use of intraoperative CAPs as an adjunct in the surgical reconstruction of nerve lesions is useful in determining if there are any conducting fascicles in lesions in continuity [20, 21].

As far as BP reconstruction is concerned, overall results from a clinical series of 204 operated cases by the senior author (JKT), including 112 cases with multiple avulsions, demonstrated that intraplexus donors consistently yielded the strongest contractile force, regardless of the

muscle target [39]. In this series [39], good or excellent results were obtained in 75 % of the suprascapular nerve reconstructions, 40 % of the deltoid reconstructions and 48 % of biceps restorations. Sedel [47] suggested that even when only two roots are available for reconstruction, a good functional outcome can be expected.

The results obtained by neurotizations using unavulsed C5 and C6 roots are far superior to those achieved when utilizing extraplexus donors like the accessory or intercostal nerves. Allieu et al. [48] reported a 66 % success rate for restoring elbow flexion following neurotization by intraplexus donors (C5 or C6).

Oberlin et al. [41] described the transfer of the branch to the flexor carpi ulnaris to the motor branch of the biceps muscle without a donor motor deficit. His series using this technique showed 85 % good results of M3 or better biceps strength. Mackinnon et al. [49] reported M4 or better strength of the elbow in 6 patients who had direct transfers of motor fascicles from both the ulnar and median nerves to biceps and brachialis, respectively.

The medial pectoral nerve can also be transferred to the musculocutaneous or the axillary nerves [50]. Functional recovery for the biceps has been reported in 80–85 % of cases, and the first evidence of reinnervation occurs between 6 and 8 months [51]. The nerve to the long head of triceps can be used for deltoid muscle restoration [52, 53]. The nerve of the long head of the triceps is synergistic to the target muscle; it is a pure motor nerve with many axons and a size that matches the axillary nerve. Leechavengvongs et al. [52] reported M4 recovery was achieved in 100 % for shoulder abduction and 85 % for external rotation after nerve to long head of triceps transfer for deltoid neurotization.

Selective C7 root transfer is a reasonable donor for multiple target neurotization because it can be coated with two to four cross-chest nerve grafts [43]. The senior author (JKT) introduced the selective cC7 technique for multiple target neurotizations. Moreover, this technique can provide motor fiber for future muscle transplantation because other donors, such as intercostals, are usually consumed for shoulder and elbow reconstruction. The postoperative morbidity of the donor limb after selective cC7 technique is limited.

In a series of 56 adult patients with severe BP injuries in whom the selective cC7 technique was utilized [42, 43], 71 % of the patients experienced numbness in the median nerve area which by 6 months was indiscernible. Motor deficit was not observed. Moreover, motor recovery reached a level of M3+ or greater in 20 % of cases for deltoid, 52 % for biceps, 24 % for triceps, 34 % for wrist and finger flexors, and 20 % for wrist and finger extensors. In addition, sensory recovery of S2 or greater was achieved in 76 % of patients [42, 43].

On the other hand, Gu et al. [54], who utilized the entire cC7, reported a series of 32 patients in whom cC7 transfer was directed to musculocutaneous, median and radial nerves. Functional recovery reached M3 or greater in 80 % of patients for the biceps, in 66 % for the wrist and finger extensors, and in 50 %% for finger flexors, and S3 sensory return or greater in 12 patients (85.7 %) after median nerve neurotization [54].

It appears that some extraplexus donors give consistently superior results when used with specific targets [55]. Songcharoen et al. [56] described 80 % motor recovery ($M \geq 3$), obtaining 60° of shoulder abduction and 45° of shoulder flexion in the transfer of 577 spinal accessory nerves to suprascapular nerve. At our center, the distal accessory nerve is routinely used to reconstruct the suprascapular nerve either by direct end-to-end coaptation or by interposition nerve grafting [13]. Outcomes were good or excellent in 79 % of the patients for the supraspinatus muscle and in 55 % for the infraspinatus muscle [13].

ICNs continue to be a standard approach in the reconstruction of severe plexus lesions, especially avulsions [55]. The most common recipient nerves are the musculocutaneous and/or branches of the posterior cord. The literature reports that 65–72 % of patients obtain M3 or greater biceps recovery rates following intercostal to musculocutaneous nerve transfers [56, 57]. At our center, a total of 718 intercostal nerves were used to neurotize different targets in adult post-traumatic patients. Lower intercostal nerves (T7, T8, T9, and T10), yielded better results in

our series than upper intercostals (T3, T4, T5, and T6) and for this reason are mostly used for musculocutaneous, triceps, and axillary nerve neurotization. T4 and T5 intercostals are used more often for neurotization of the thoracodorsal and long thoracic nerves.

As far as peripheral nerve reconstruction of the upper extremity, alternative methods of reconstruction must be considered in nerve injuries with significant nerve gaps or tension, which include nerve grafting, nerve transfers and end-to-side nerve repair. Autologous nerve grafts have proven to be a popular and reliable method for the reconstruction of peripheral nerve defects [58]. Most often, the sural nerve is used as a donor graft for peripheral nerve reconstruction.

Time from initial injury to nerve grafting is often a determinant of functional outcome [59]. Vascularized nerve grafts are a valuable tool for certain indications; scarred recipient site or poor vascularity of the whole area where the nerve is to be repaired or when long defects need to be bridged.

Injuries to the ulnar nerve are the most frequent, occurring either in isolation or in association with the median nerve [60]. Kim et al. [61] reported the largest series of ulnar nerve lesions in 2003 and stated that surgical results were generally better for lesions in continuity with positive compound action potential recordings than for discontinuous lesions. Among 181 patients, favorable results were seen in 92 % of patients who underwent neurolysis, 72 % of patients with suture repair (primary and secondary), and 67 % of patients who received graft repair.

The senior author (JKT) reported her experience with ulnar nerve reconstruction in a series of 44 patients [62]. According to this, good and excellent motor results were seen in 92 % of patients who underwent neurolysis, 60 % of patients who received secondary suture repair and in 63 % of patients with graft repair.

The radial nerve is the most frequently injured nerve in the upper extremity especially in patients with multiple injuries [63]. Shergill et al. [64] and Kim et al. [65] reported the largest series of radial nerve lesions. Shergill et al. [64], reviewed 260 patients with radial nerve injuries and reported that 30 % had good results, 28 % fair and 42 % of the repairs had failed. Kim et al. [65] reported a series of 260 RN injuries in 2001. Among 180 patients, favorable results were observed in 91 % of patients who underwent primary suture repair, in 83 % of patients who underwent secondary suture repair, in 80 % of patients who received graft repair, and in 98 % of patients that had neurolysis. Our Center reported a series of 35 patients with various radial nerve lesions in 2011 [15]. Good and excellent results were seen in 100 % of patients who underwent neurolysis, in 88.88 % of patients who received suture repair, and in 57.14 % of patients with graft repair.

The use of end-to-side repair in the clinical setting for motor recovery remains controversial [14]. Currently, motor reconstruction in the absence of available proximal nerve is best handled by nerve transfers [66]. Motor neuron regeneration through end-to-side repairs is optimized by deliberate injury of donor nerve axons.

Complications of Treatment

Any kind of complication that occurs after major surgery can also occur after nerve reconstruction. Moreover, there are complications related to the type of nerve repair, which may include:

In an end-to-end neurrorhaphy the most common complications are: (1) Fibrosis in the repair site from sutures placement or inadequate recipient bed, and (2) Misdirection of the nerve fibers due to improper alignment of the nerve stumps.

As far as nerve grafting is concerned, the most common complications are: (1) Sensory loss and scarring in the donor site; (2) Neuroma formation in the proximal stump of the donor-nerve, and (3) Failure of the graft to survive if the repair took place in a vascularly compromised bed.

In nerve transfers complications may include: (1) Loss of function from donor nerve site and (2) Donor muscle atrophy. In end-to-side neurorrhaphy complications include: (1) Donor axonal injury and (2) Donor muscle atrophy.

Conclusions

Advances in the field of peripheral nerve surgery have increased our understanding of the complex molecular and cellular events surrounding nerve injury and repair. There are several factors that influence recovery following a nerve injury: time elapsed, patient age, mechanism of injury, proximity of the lesion to distal targets, and associated soft tissue or vascular injuries. All these factors must be carefully considered in order to optimize the operative approach used in each patient. Prompt repair of nerve injuries leads to improved outcomes by allowing for earlier distal motor end plate and sensory receptor reinnervation.

The ultimate goal of any peripheral nerve reconstruction is the restoration of function as promptly and completely as possible, while minimizing comorbidities. If end-to-end repair is not possible, several options for repair include interpositional nerve grafting, nerve transfers and end-to-side neurorrhaphy. Selection of each technique depends on the surgeon's experience and individual nerve injury characteristics.

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Tendon Transfers

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Michael Solomons

Keywords
Tendon Transfers • Hand tendon transfers • Ulnar nerve transfers • Ulnar nerve reconstruction • Median nerve transfers • Median nerve reconstruction • Radial nerve transfers • Radial nerve reconstruction • Wrist drop • Jones Transfer • Opposition transfer • Zancolli Lasso • Claw hand • Riordan

Introduction

The art of hand surgery is appreciated when normal anatomical structures are brought in to substitute damaged non-functional tissue. Examples include vascularized bone grafts, flap surgery and tendon transfers. The exquisite functioning of the hand is characterised by mobility and sensibility. Motion is achieved by the contractile action of functioning musculo-tendinous units on mobile joints. When a specific function has been lost due to failure of the nerve, the muscle or the tendon, impairment is inevitable. Unless the damaged structures can be repaired and a satisfactory outcome achieved, tendon transfers become the mainstay of reconstruction and rehabilitation to a near normal premorbid status.

In borrowing from a functioning musculotendinous unit to replace that which is non-functioning, we acknowledge the collective experience of those who practised before us. One hundred and fifty years ago, Nicoladeni first reported on a successful tendon transfer in the foot. With the impetus being the polio epidemic, and the means-to-achieve being the vast improvements in anti-sepsis, safe anaesthesia and better equipment, many thousands of tendon transfers were performed, peaking in the mid 1900's. Many of the eponymous transfers we perform today date from this period. (Brand, Boyes, Omer, Jones, Bunnel, etc.)

Prior to performing transfers, the practising surgeon needs to be familiar with the essential fundamental principles governing these procedures. Thereafter a surgical strategy can be formulated.

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General Principles of Tendon Transfers (See Table 2.1)

Supple Joints

Applying an active motor to a passively mobile joint is the fundamental tenet of a tendon transfer. No amount of transferred power will mobilize a stiff joint. Extensive physiotherapy, the judicious use of splints and corrective surgery are often needed to correct stiffness or contracture prior to tendon transfer procedures. Once a plateau or end point has been reached, then the definitive transfer is performed, understanding that the active range of motion will, at best, match the passive range of motion.

Tissue Equilibrium

The elastic energy inherent in the transfer should be maximally applied to the passively mobile joint that the transfer is designed to move. Any residual oedema, firm scar or joint stiffness will result in an increased resistance to the muscle force and therefore, a compromised result. This optimal tissue condition is termed "tissue equilibrium". Reducing post operative oedema and scar formation is equally important.

Gentle dissection through a fatty subcutaneous bed offers the best pathway for a transferred tendon. Curved incisions should be used to place tendon junctures beneath flaps rather than beneath incisions [3].

Adequate Donor Strength

The transferred musculotendinous unit must have enough strength to perform its desired function. While there has been excellent basic science work on muscle strengths by the masters (Brand, Boyes), these tables are often not practical and easily usable [1, 2]. Form follows function and therefore one can assume that tendons of similar cross sectional size have similar strength.

A transferred muscle should be of near normal strength, as Omer has shown that it will be lose one grade from 5 to 4 or from 4 to 3 [3]. Surgeons are often advised to beware a muscle that has recovered strength following denervation or paralysis. These muscles might appear strong enough but often suffer fatigability.

Adequate Donor Excursion

The transferred musculotendinous unit should have an excursion similar to that of the tendon it is replacing. Surgeons are advised to commit the following excursion values to memory. Wrist flexors and extensors – 3 cm, finger extensors (including EPL) – 5 cm and finger flexors – 7 cm (3, 5, 7 rule) [2] (See Table 2.2).

Tendons of similar excursion are often not available. Extensive mobilization of the fascial attachment of the muscle can increase the excursion. A good example is the FCU to EDC transfer. A 3 cm excursion can be increased to the desired 5 cm by dissecting the muscle free from its broad attachment to the ulna. Furthermore, the body can adapt to excursion differences by utilizing the tenodesis effect. By flexing the wrist, the effective excursion on the transferred FCU is increased, thereby facilitating finger extension. The fact that wrist flexion (FCU) and finger extension are in-phase or synergistic also adds to the success of the transfer (see section "Synergism").

Table 2.1 General principles of tendon transfers

1. Supple joints before transfer.
2. Tissue equilibrium
3. Adequate strength (donor)
4. Adequate excursion (donor)
5. Expendable donor
6. Straight line of pull
7. Synergism
8. One tendon – One function

Table 2.2 Tendon excursion

Tendon	Excursion (cm)
Wrist flexors	3
Wrist extensors	3
Finger extensors	5
Finger flexors	7

Expendable Donor

It goes without saying that every donor muscle to be transferred should either be expendable or, after careful clinical assessment, shown to be less important than the recipient muscle targeted. Fortunately, there are two wrist flexors and three wrist extensors which means that function remains following the use of one of these tendons for transfer.

Straight Line of Pull

In order to minimize the energy loss of the transfer, the transfer tendon should assume as straight a line as possible from the donor motor to the recipient. Excessive use of a redirectional pulley should be avoided where possible.

Synergism

This is a very important concept to understand. In the normal balance of the hand, as the wrist flexes, so the fingers extend and as the wrist extends, the fingers flex into a fist. Transfers that take advantage of this co-contractile synergism are destined to be superior to those that are out of phase.

A classic example of this is the wrist flexor (FCU or FCR) to EDC transfer in high radial nerve palsy. Any attempt to extend the fingers is associated with a reflex of spontaneous tendency to flex the wrist which thereby augments the transfer. In so doing, the brain does not have to relearn the action centrally.

Another way to understand this point is the notion that if the active transfer does not work or gets stuck down in scar tissue, then the transfer will still function as a tenodesis affect.

One Tendon – One Function

A transferred tendon should not be split into two separate insertion points. It would be extremely difficult to set up the tension in both limbs of the transfer so that two actions will be performed simultaneously. Secondly, to expect one tendon transfer to perform a function at two or more contiguous joints is subjecting that transfer to increased risk of failure. An example is expecting a transferred wrist flexor to extend both the wrist and the fingers.

Table 2.3 Surgical algorithm

1. What is lost?
2. What is available?
3. Is the hand suitable for a tendon transfer?
4. Is the patient suitable for a tendon transfer?

Surgical Algorithm (Table 2.3)

What Is Lost?

Careful clinical assessment will reveal which joint functions need to be reconstructive. It is more important to identify the functional deficit rather than a list of all the muscles that are not actively contracting.

What Is Available?

Often, the algorithm ends here. If no motors are available for transfer, then no reconstructive options are available. If multiple donors are available, then the principles as discussed above will guide the preferred and correct choice.

Is the Hand Suitable for a Tendon Transfer?

Once a donor muscle has been selected, the next critical assessment is to decide whether the patient and/or the patient's hand are suitable for a tendon transfer. In order for the transferred muscle to perform a locomotor function, the joint that is mobilized by the donor muscle must be supple and capable of an easy passive range of motion. Surgery should be delayed until physiotherapy has restored full passive mobility.

The second critical criterion to assess in the hand is the sensibility to the anatomical structure to be reanimated. A tendon transfer to a poorly

sensate hand will often result in a suboptimal clinical and functional outcome. This is because the brain will tend to exclude these areas from functional activity, and hence movement because of their reduced sensory feedback.

Is the Patient Suitable for a Tendon Transfer?

Tendon transfer surgery can be technically challenging and careful attention must be applied to the post operative rehabilitation. Young children and patients with impaired cognitive functioning will struggle with a complicated post operative physiotherapy regimen. Cerebral palsy patients are a unique group that can be difficult to assess. They often have many combined factors mitigating against tendon transfers. These might include spasticity, poor sensibility, altered cognitive functioning and failure of the developing brain to incorporate the use of the affected limb.

Radial Nerve Palsy

Anatomy

The radial nerve is the terminal branch of the posterior cord of the brachial plexus. It passes in a posterior direction around the humerus where it runs between the medial and lateral heads of triceps in the spiral groove. At this level it is closely applied to the bone and, as such, is liable to injury when the humerus shaft is fractured. The three heads of triceps are innervated proximally in the upper arm and this muscle is usually not affected in the more common causes of radial nerve palsy. At the mid-humerus level the radial nerve is immediately posterior to the bone and at the level of the junction between the middle and distal thirds of the humerus; the nerve lies immediately lateral to the bone. An understanding of the relevant surface anatomy of the radial nerve is important when planning surgical incisions or inserting external fixation pins.

Just proximal to the cubital fossa of the elbow the nerve lies between biceps and brachioradialis muscles. At this level it gives off branches to Extensor Carpi Radialis Longus and Brachioradialis (BR). The nerve now splits into the superficial branch and the Posterior Interosseous Nerve (PIN). The superficial branch is a purely sensory nerve which lies on the deep surface of BR until the distal one third of the forearm. The PIN passes lateral to the proximal radius between the two heads of supinator muscle which it supplies. After it leaves the supinator muscle the PIN splits into its terminal branches supplying all the extensor muscles.

Assessment

In high radial nerve palsy there is loss of all extensor function below the elbow (BR, ECRL, ECRB, EDC, EIP plus EDQ, EPL, APL, EPB) (See Table 2.4). This should be differentiated from posterior interosseous nerve palsy or low radial nerve palsy where BR & ECRL are preserved. These patients present with weak but intact wrist extension in marked radial deviation (Fig. 2.1).

High Radial Nerve Palsy

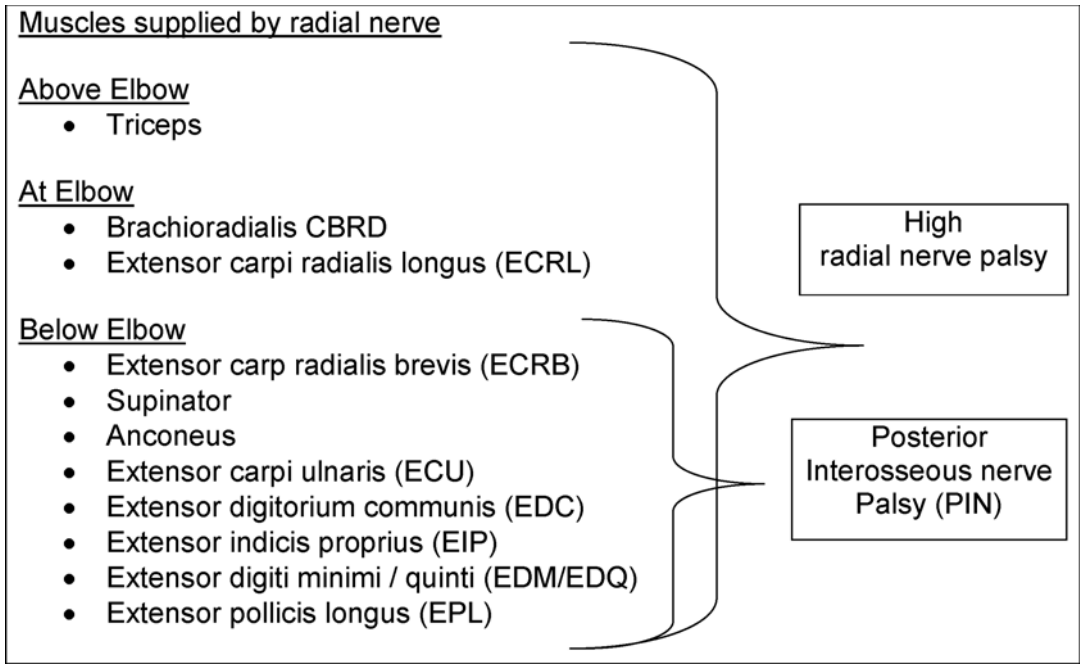
These patients present with three distinct clinical deficits.

- Loss of wrist extension
- Loss of thumb extension
- Loss of finger extension

Most authors would agree that the preferred technique to restore wrist extension is by transferring the pronator teres. This reliable transfer is complicated only by the short donor tendon of pronator teres which needs to be elongated by a strip of periosteum at the time of harvest. In the unlikely scenario where the pronator teres cannot be used, then options include utilizing the FDS tendon from the middle and/or ring finger or performing a single FCU transfer to reconstruct wrist extension, finger extension and thumb extension [4].

Loss of function in EPL, APL and EPB results in an inability to retropulse the thumb out of the palm. The poorly positioned thumb now interferes with cylindrical grasp and the ability to

Table 2.4 Muscles supplied by radial nerve



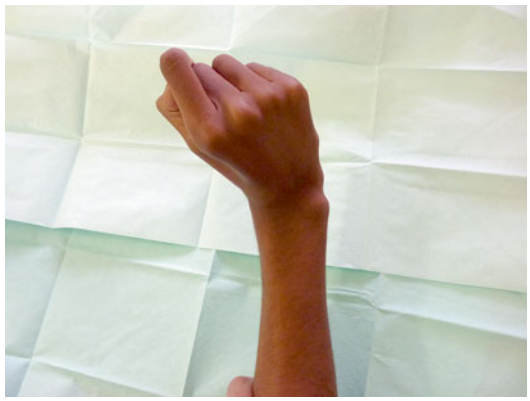


Fig. 2.1 Marked radial deviation on attempted wrist extension due to intact BR and ECRL in PIN palsy

release larger objects. As in the case of wrist extension transfers, reconstruction of thumb extension is mostly concurred by various authors. The EPL is rerouted out of the 3rd compartment and anastomosed to the palmaris longus. This transfer usually results in a satisfactory ability to clear the thumb out of the plane of the palm. In patients who do not have palmaris longus (12 %), the best option is to include EPL with the tendon transfer to the finger extensors as a mass action transfer. Wrist flexion will result in simultaneous metacarpophalangeal joint extension and extension/retropulsion of the thumb.

The most controversial aspect of radial nerve palsy reconstruction revolves around the restoration of finger extension. The use of a wrist flexor (FCU or FCR) to achieve finger extension, results in a transfer that is both strong enough as well as being in-phase with the normal tenodesis effect of the hand. Boyes advocated the use of finger flexors (FDS 4) to motor EDC but few clinicians make use of this technique today [5, 6].

Jones initially described the use of FCU as a motor [7] but many, including Brand [8], feel strongly that FCU is critical for the normal kinematics of the wrist as it is the main motor for flexion–ulna deviation in the "dart throwers" or hammering motion. Despite these concerns, Raskin and Shaw Wiglis et al. failed to show any loss of wrist flexion strength or function after FCU harvest [9].

Many authors are of the opinion that the FCR transfer (Brand) is easier, quicker and associated

with less intra-operative dissection. Mobilization of FCU requires a substantial incision and dissection because FCU is a unipennate muscle with strong fascial attachments to the ulna periosteum along its entire length. In the rare event, where the wrist flexor is expected to power wrist, finger and thumb extension, then FCU would be a reasonable donor to sacrifice.

Goal	Transfer
1. Wrist extension	PT to ECRB
2. Finger extension	FCR to EDC (Brand) Or FCU to EDC (Jones) Or FDS to EDC (Boyes)
3. Thumb extension	PL to EPL (rerouted) Or if no PL (12 %) then FCR/FCU to EDC + EPL Or FDS to EPL

Low Radial Nerve Palsy

These patients present with intact wrist extension through the maintained functioning of ECRL. Because of this, they do however tend to extend into radial deviation which is made worse by the lack of tone in ECU. To take away the remaining ulna deviator in the form of FCU would exacerbate the radial deviation acutely. For this reason, where there is debate over the choice of wrist flexor to power finger extension in high radial nerve palsy, in posterior interosseous nerve palsy there is NO underlying debate. Only FCR should be utilized. Thought should be given to rerouting the transfer around the ulna border of the forearm to balance the radial deviation moment at the wrist.

Surgical Technique (Fig. 2.2)

The following surgical technique describes the FCR set of tendon transfers. An S-shaped curvy linear incision is made on the volar radial aspect of the mid forearm approximately 8 cm long. The midpoint of this incision is at the insertion of pronator teres and is situated over the radial artery and the volar border of brachioradialis. This utilitarian incision will be utilized to dissect PT, ECRB, FCR and PL.

As per the standard Henry approach, the deep fascia is incised at the medial edge of BR. The radial artery and vein lie immediately deep to the fascia at this point and will be retracted in an ulna direction. The oblique fibres of PT are identified coursing from the medial epicondyle to the mid volar radius. The distal/ulna and proximal/radial edges of the muscle and tendon are identified and dissected off the adjacent epimysium.

The PT tendon and a 2 cm strip of periosteum are elevated off the radius by sharp dissection in a proximal to distal direction. Gentle traction of the tendon will allow substantial mobilization of the pronator teres muscle. Eventually, the deep head blends into the common flexor muscles making further dissection hazardous. A 3 cm excursion of the PT is confirmed and is considered adequate.

A separate transverse incision is made over the volar wrist crease. Care is taken to identify and protect the palmar cutaneous branch of the median nerve. The PL is separated from its dense fascial attachments and transected distally. The FCR tendon is identified and mobilized by passing a tendon hook around it and delivering a loop of tendon out of the wound. By flexing the wrist and tensioning the tendon, the FCR can be transected as far distally as possible. These tendons are delivered into the 1st incision and the 2nd incision (transverse wrist) is closed with a subcuticular suture. As this incision is sutured in the flexion crease, it heals with a pleasing cosmetic result.

Through the same utilitarian incision, the volar skin flap is elevated to expose the tendons of PL and FCR. The dorsoradial flap is elevated and with the help of passive pronation, the tendons of ECRL, ECRB and BR are identified. Care must be taken to preserve the superficial branch of the radial nerve lying initially on the deep aspect of brachioradialis and then passing distally between the BR tendon volarly and the combined tendons of ECRL and B dorsally. Even though the radial nerve innervated muscles might not be functioning, there might be some sensation passing through the radial sensory nerve and for this reason, it needs to be protected. These two tendons often run in the same fascial sheath and must be carefully separated. Remember that the ECRL lies lateral to the ECRB on its way to the base of index finger metacarpal.

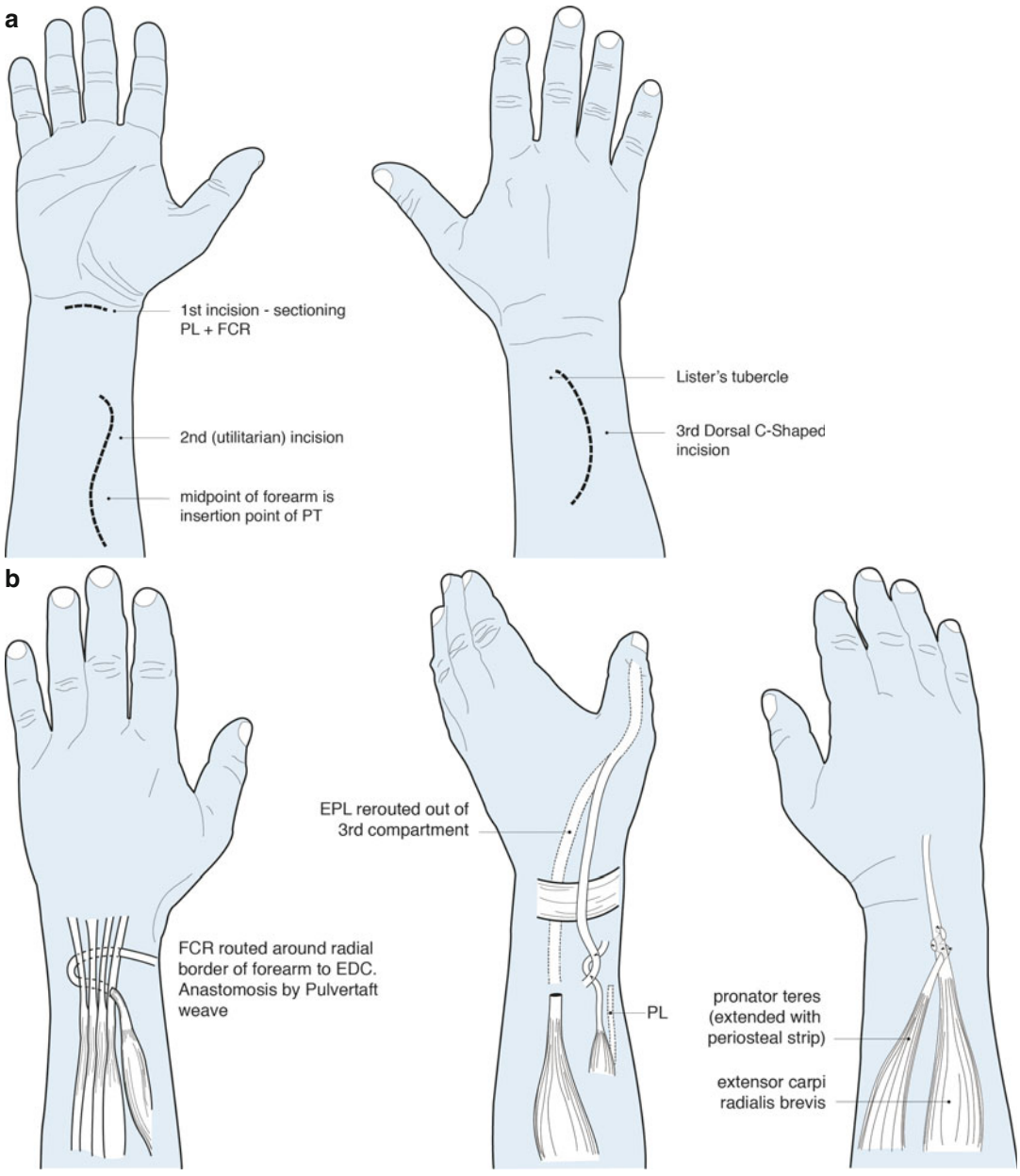


Fig. 2.2 Line drawing of radial nerve tendon transfer. (**a**) Incisions as discussed in text. (**b**) First the FCR is transferred around the radial border of the forearm and anastomosed to the EDC tendons by a Pulvertaft weave just distal to the musculotendinous junction. Then the PL is transferred to the rerouted EPL. Finally the PT is transferred to the ECRB

Pearl
ECR**L** is **L**ateral

The third incision is C-shaped and curved longitudinally, starting at Lister's tubercle with its apex over the interosseous membrane. An 8 cm incision will easily suffice. Full thickness skin

flaps are elevated with complete exposure of the extensor retinaculum. A large window is made in the proximal aspect of the retinaculum and this is flapped in an ulna direction. The EPL tendon is identified and transected proximally. The retinaculum is opened over the EPL distally to deliver the cut proximal end out of the 3rd dorsal compartment. This tendon is now rerouted over the 2nd compartment in preparation for transfer.

A subcutaneous dissection is performed from the 1st incision to the 3rd and the FCR and PL tendons are passed through. The 4th compartment consisting of the EDC tendons together with the EIP is mobilized and tractioned to confirm there are no adhesions limiting excursion and to confirm that all the MP joints are extending. Occasionally the little finger lags and the EDM (5th compartment) may need to be incorporated into the transfer. Now the transfers are performed.

The PL is sutured to the rerouted EPL and FCR is sutured to the EDC tendons. Various anastomoses can be performed including the Pulvertaft weave [10], the spiral technique [11], the Lasso technique and the side to side suture [12]. What is more important is the tension of the transfer and the bulk of the repair site. There have been many descriptions of methods of setting the tension in these transfers. There are two issues that need to be addressed. Firstly, the MP joints should achieve passive extension as the wrist is brought into flexion (the tenodesis effect) and secondly, in wrist extension, the fingers should be easily able to achieve a full fist. Failure to address the later point can seriously compromise flexion causing a larger functional impairment than a loss of extension.

The technique I prefer is to set the tension with the wrist in 20° extension and the MP joints in 0° extension. With this static position held, the tendons are tractioned gently to a maximum length and then the FCR is released approximately 1 cm. Two or more horizontal mattress sutures are placed and the tenodesis effect is checked. Any laxity or tightness can be corrected. The PL to EPL transfer often has a tendency to lag post operatively and for this reason a conscious effort is made to tension this transfer slightly tighter with the wrist in 45° of dorsiflexion. The 3rd incision can now be closed with subcuticular absorbable sutures.

The final aspect of the procedure involves suturing the pronator teres to ECRB. This component of the overall transfer is performed last to allow intraoperative checking of the tenodesis effect to assess the correct tension of the first two transfers. ECRB is mobilized and tractioned fully until the wrist extends to 60°. A tight weave between the tendon of PT and ECRB is performed. When released, the wrist should not drop below a 30° extended position. If it does, then the transfer should be tightened. The incision is now closed.

A volar below elbow plaster slab is applied with the wrist 60° extended and the MP joints in 0° extension. The IP joints are left free and the thumb is held with a volar plaster of Paris slab in a position of maximum retropulsion. After 3 weeks, the plaster is removed and the wrist supported in a volar splint. This splint is removed every 2 h to allow only active flexion of the wrist and fingers. Once flexion of the fingers into the palm is achieved (usually 1 or 2 weeks), a lively extensor splint is worn for a further 3 weeks. This splint should also be removed 2 hourly for wrist flexion and extension exercises to facilitate the tenodesis affect.

Timing of Radial Nerve Tendon Transfers

Radial nerve palsy complicates a fracture of the humeral diaphysis in approximately 10 % of cases. Other well described causes include Saturday night palsy, gunshot injuries and sharp penetrating injuries. The management of the radial nerve with an associated humerus shaft fracture is controversial. Between 70 and 90 % of these injuries will recover spontaneously and therefore primary exploration per se is not indicated.

The one exception is an open fracture of the humerus where the incidence of a transected nerve is greater than 50 % [13]. The rest will be treated expectantly. If after 6 months, there has

been no recovery then intervention is required. Most would advocate exploration and grafting. It needs to be clearly explained to the patient that, while the results nerve grafting are good [14], the recovery can be extremely prolonged (6–9 months on average).

Following a radial nerve tendon transfer procedure, the patient can reliably expect extensor function to the upper limb to be reconstructed. Return to work and avocational activities can be anticipated by 3 months.

Pearl: Outcome in Adults and in Children

Primary nerve repair is better than tendon transfer in both.

Children: Nerve grafting has better results than tendon transfer.

Adults: Nerve grafting has similar results to tendon transfer (but much longer recovery time).

It is therefore our unit's policy to perform nerve grafting in all children and adolescents but consider tendon transfer in all cases who fail to recover expectantly after 6 months, who do not recover extensor function 1 year after primary nerve repair and where a nerve gap is encountered in a delayed presentation following a penetrating injury. Furthermore, we do not routinely perform an interim transfer to augment wrist extension while waiting for the nerve repair or graft to recover. More than 80 % of these patients will recover wrist extension starting at about 3–4 months. A simple wrist extension splint will suffice and avoid surgery in 80 % of cases.

Ulnar Nerve Palsy

Introduction

Ulnar nerve palsy results in a devastating deficit to the hand. Due to its subcutaneous position it is readily susceptible to a host of injurious causes such as penetrating trauma, gunshots, compound fractures and burns. Its predilection for involvement in leprosy has resulted in a massive number of reconstructive procedures having been performed in endemic areas. Fortunately, end stage cubital tunnel syndrome with dense ulna nerve deficit is extremely rare today.

Anatomy

After leaving the medial cord of the brachial plexus, the ulnar nerve gives off no branches in the upper arm. It penetrates the medial intermuscular septum from anterior to posterior before passing behind the medial epicondyle. It enters the forearm between the two heads of FCU and innervates the muscle in its proximal 5 cm. The nerve now lies with the ulna artery on the volar surface of the FDP and innervates the two medial FDP muscle bellies. It continues to travel on the deep surface of the FCU tendon all the way to the pisiform bone. Approximately 5 cm proximal to the canal it gives off a dorsal sensory branch which passes deep and dorsal to the FCU on its way to the dorsal aspect of the hand. The ulna nerve passes radial to the pisiform and ulnar to the hook of the hamate in Guyon's canal. In Guyon's canal, the nerve splits into a sensory branch supplying sensation to the little finger and the ulna half of the ring finger, and a motor branch which supplies the hypothenar muscles, all the interossei, the medial two lumbricals, the adductor pollicis brevis and the deep head of flexor pollicis brevis. Occasionally one sees patients with median nerve palsy who have maintained opposition. This implies an anomalous supply of opponens pollicis by the ulna nerve. Often described, but rarely visualized, anastamoses between the median and the ulna nerve include the Martin-Gruber [15] and the Riche-Cannieu. The Martin-Gruber anastomosis is a branch in the proximal forearm from the median nerve to the ulna nerve. A recent large cadaveric study [15] has identified the presence of this nerve branch in more that 20 % of people. Much less common is a more distal anastomosis from the ulnar to the median nerve in the hand. This is termed the Rich-Cannieu anastamosis and can explain thenar muscle preservation in the face of severe end stage carpal tunnel syndrome.

Assessment

Meticulous clinical examination of both motor and sensory systems will determine the level of the ulna nerve injury. High ulnar nerve palsy is associated with loss of sensation to both the volar and dorsal aspects of the ulnar side of the hand.

Low ulnar nerve palsy, distal to the origin of the dorsal sensory branch will present with volar sensory deficit only. Sensory loss to the ulnar side of the hand is critical and must be restored. Lack of protective sensibility commonly results in burns or lacerations when supporting oneself or leaning against noxious surfaces.

Pearl: Motor Supply to Hand and Forearm

The ulnar nerve supplies very little in the forearm and almost everything in the hand.

The median nerve supplies almost everything in the forearm and little in the hand.

The motor supply to the hand by the ulnar nerve is critical for balanced dexterity. This leads to the first obvious deficit with an ulnar nerve palsy- unbalanced flexion. Digital flexion is initiated by metacarpophalangeal (MPJ) joint flexion. This motion is controlled by the intrinsics. Thereafter, the long flexors take over and continue to flex the fingers all the way into the palm. This action of composite flexion allows us to wrap our hands around large objects for cylindrical grasp. It is interesting to note that the opposite is mirrored on the extensor surface. Interphalangeal extension is controlled by the intrinsics whereas MPJ extension is by means of the long extrinsic extensors.

Without intrinsic muscle activity the fingers can only flex by sequentially "rolling up" from the DIP joints to the PIP joints and finally the MP joints. This form of roll up grasp precludes the effective grasp of larger objects.

The second problem with the ulna nerve deficient hand is clawing (see Fig. 2.3). As mentioned above, the MPJ's are flexed by the intrinsics and extended by the extrinsics. Ulnar nerve palsy results in an unbalanced situation where the extrinsic extensors dominate and pull the MP joints into extension. This is exaggerated by an inability to extend the PIP joints (intrinsic action). The more the person tries to "extend" the fingers, so the action of the extrinsic extensors is increased and the clawing worsens. Usually only the ring and little fingers are affected due to the remaining function in the lumbrical muscles to the index and middle fingers (median nerve). In low ulnar nerve palsy, the long flexors to the little and ring fingers are functioning and thus increase the tendency to flex the interphalangeal joints and make the claw worse. In high palsy, the FDP to the little and ring fingers are not functional and therefore there is less clawing. This is called the ulnar paradox.

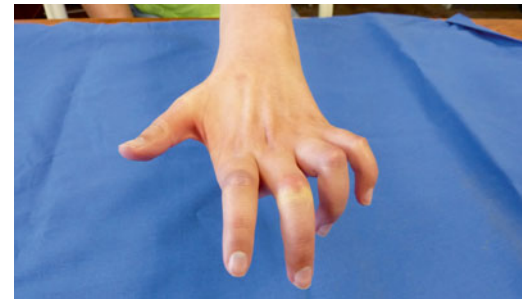


Fig. 2.3 Observe marked clawing of little and ring and lesser clawing of middle and index. Wasting of the 1st dorsal interosseous can also be seen

Ulnar Paradox

High ulnar nerve palsy: severe injury: mild claw

Low ulnar nerve palsy: less severe injury: severe claw

The third problem in the ulnar deficient hand is weakness of power grip. This is due to weakness of intrinsic flexion at the MP joints and is worsened by lack of long flexor function to the little and ring in high ulna nerve palsy. The absolute grip strength as measured on a dynamometer and the patient's need for power grip in the reconstructive algorithm needs to be assessed.

The fourth major problem with the ulnar nerve deficient hand is weakness of lateral key pinch between the pulp of the thumb and the lateral

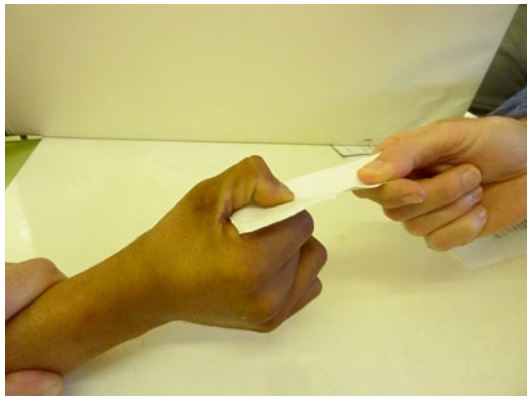


Fig. 2.4 Froment's sign: Observe marked flexion of IPJ on attempted pinch

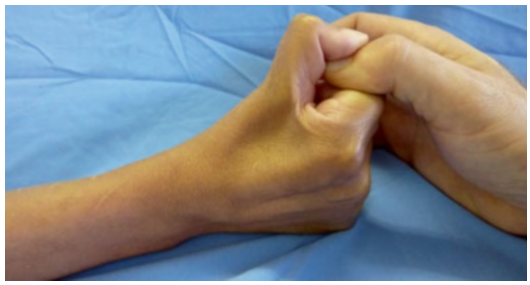


Fig. 2.5 Jeanne's sign: marked hyperextension of thumb MPJ on attempted pinch

aspect of the index finger. This action is a composite of thumb adduction and index abduction. Thumb adduction is powered by abductor pollicis and flexor pollicis brevis (superficial head median nerve supply, deep head ulna nerve supply). Index abduction is powered by the first dorsal interosseous. Some patients have extremely marked weakness of pinch and compensate in various ways. The common method is to hyperflex the interphalangeal joint (IPJ) of the thumb. This posture is termed Froment's sign (see Fig. 2.4). The IPJ of the thumb flexes to incorporate flexor pollicis longus into the pinch action and at the same time the flexed IP joint causes a subtle realignment of EPL which increases its adduction direction of pull. Jeanne's sign occurs when the thumb MP joint collapses into hyperextension due to the weakness of flexor pollicis brevis (see Fig. 2.5). FPB normally flexes the MP joint of the thumb and Jeanne's sign probably occurs when both heads of FPB are supplied by the ulnar nerve.

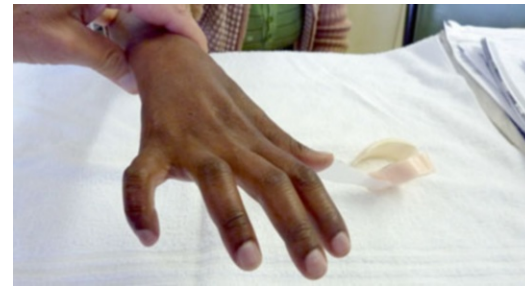


Fig. 2.6 Wartenberg's sign: marked abduction of little finger at MPJ. Note also the ulnar claw deformity

Wartenberg's sign describes the abduction deformity of the little finger at the MP joint due to unopposed extensor digiti minimi activity (see Fig. 2.6). EDM attaches from an ulnar direction causing ulnar deviation.

Management Algorithm

Of the four problems mentioned above, the first two will present with the same symptom complex, namely clawing of the fingers. This then leaves three pathological functions to be assessed. All patients with high or low ulnar nerve palsy need to be evaluated looking specifically at these three major clinical deficits.

1. clawing
2. weakness of grip
3. weakness of pinch

Elements of all three are present if looked for. The treating physician assisted by a hand therapist should create a treatment hierarchy based on the patient's vocational and avocational requirements. Grip and pinch strength testing as well as objective hand testing should be assessed.

It is this author's opinion that clawing and weakness of pinch can be relatively satisfactorily addressed whereas reconstruction of power grip is difficult and unpredictable.

Clawing

Not every patient with an ulnar nerve deficient hand will present with symptomatic weakness of grip strength. Office workers, older patients and involvement of the non-dominant hand would be

examples of indications for correction of the claw deformity rather than intrinsic replacement procedures which are capable of increasing grip strength. Anti-claw procedures are technically less challenging than intrinsic replacement procedures.

The premise of the anti-claw procedure is the understanding that by blocking MPJ hyperextension the long extrinsic extensors (EDC) are capable of extending the interphalangeal joints. Normally, proximal motion of the extensor digitorum communis tendons causes traction on the sagittal hood overlying the metacarpophalangeal joint. The transverse fibres of the hood attach on the volar aspect to the flexor sheath and the volar plate. In the claw hand the extensor digitorum communis pulls the metacarpophalangeal joint into hyperextension until there is no further extension possible. Distal to this joint, the extensor has not moved proximally enough to apply tension to the central slip insertion at the dorsal aspect of the middle phalanx resulting in proximal interphalangeal joint flexion. Limiting metacarpophalangeal joint extension allows more proximal travel (excursion) of the extensor digitorum communis such that tension can be applied to the central slip insertion at the proximal interphalangeal joint. Now the proximal interphalangeal joint extends under the influence of the long extrinsic extensor. Eliciting this phenomenon is termed Bouviere's manoueuvre (see Fig. 2.7). It is mandatory to perform this test in every case of ulnar nerve clawing presenting for surgical reconstruction. In long standing cases, the central slip may have stretched out or the proximal interphalangeal joint may have developed a fixed flexion deformity. In both scenarios, the Bouviere's test will be negative i.e. the proximal interphalangeal joint will not extend despite blocking the metacarpophalangeal joint in flexion.

While awaiting surgery, the patient should wear a splint that blocks the metacarpophalangeal joint in extension. This splint is termed a knuckleduster splint (see fig. 2.8).

Surgery to correct a mobile claw deformity revolves around creating an internal block to hyperextension at the metacarpophalangeal joint. There are various ways of creating an internal block to extension. These can be divided into **static and dynamic**.

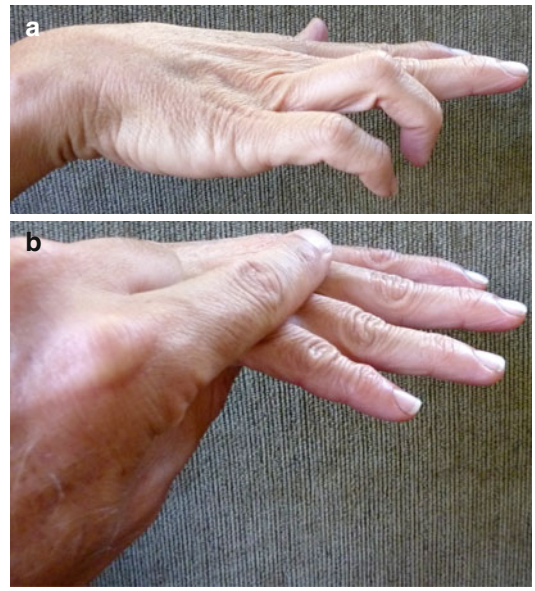


Fig. 2.7 (**a**) Clawing of little and ring. (**b**) By blocking MPJ hyperextension the PIPJ's can be extended by the long extensors – Bouviere's test

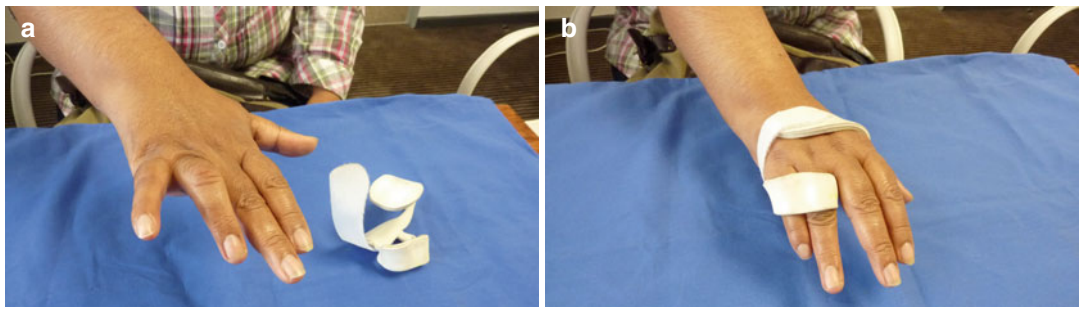


Fig. 2.8 Ulnar claw deformity (**a**) with and (**b**) without knuckle duster splint

Static operations include the use of the volar plate and a static tenodesis across the metacarpophalangeal joint. Zancolli described an elliptical excision of a portion of the volar plate of the metacarpophalangeal joint (Zancolli 1 procedure) [16]. The distally based flap was sutured to the volar neck of the metacarpal to create a block to extension (see Fig. 2.9a). Unfortunately, this tends to stretch out over time. Parkes used a tendon graft and sutured this from the deep transverse intermetacarpal ligament (DTIL), volar to the axis of the joint, through the lumbrical canals, to the lateral band [17] (see Fig. 2.9b). Bunnell suggested releasing all of the A1 pulley and part of the A2 pulley to allow bowstringing of the flexor tendons. By increasing the moment arm of the flexor tendon, flexion is augmented and extension weakened. A dorsal tenodesis procedure developed by Fowler [18] adds the influence of wrist position on the claw deformity. A graft is passed from the radial lateral band of each finger to be corrected, through the lumbrical canals, deep to the DTIL, through the intermetacarpal space to the extensor retinaculum. When the wrist is flexed, an active tenodesis effect occurs resulting in metacarpophalangeal joint flexion and interphalangeal joint extension (see Fig. 2.9c).

Various **dynamic procedures** have been described to achieve the above goals – namely increased metacarpophalangeal joint flexion and thereby facilitating interphalangeal joint extension. These all involve the use of the FDS tendons. What differs is the insertion. Originally described by Stiles, later modified by Bunnell and finally by Riordan [19], the FDS to the middle finger is transected distally and delivered into the palm, four strips are created. Each of these are passed through the lumbrical canals and inserted into the radial lateral band of each finger (see Fig. 2.9d). Once again, the idea is to create a flexion force at the MP joint and an extension force at the PIP joint. There are two problems with this transfer. The first involves sacrificing an important flexor in a patient who already has compromised grip strength. The second problem involves the undesirable result of developing a swan neck deformity when an extension force is added to the intrinsics at the PIP joint. To obviate against this, Burkhalter advised attaching the FDS to the proximal phalanx rather than the intrinsics [20] (see Fig. 2.9d).

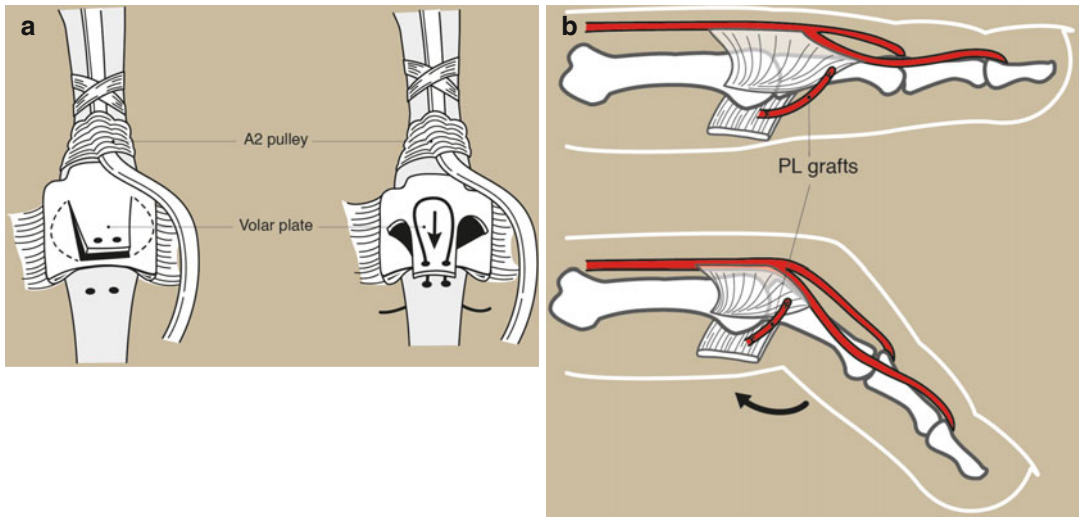


Fig. 2.9 (**a**–**e**) Line drawing of Anti-claw procedures. (**a**): Zancolli 1 – static capsulodesis. (**b**) Parkes procedure – static tenodesis with free tendon grafts. (**c**) Fowler operation – mostly static tenodesis with free tendon grafts anchored to extensor retinaculum. Has advantage of variable tension with wrist position (some dynamic component). (**d**) Dynamic – active motor is FDS middle. Variable attachment. Stiles, Bunnel, Riordan (Modified by Burkhalter). (**e**) Zancolli 29 – active motor is FDS. Around A1 or part A2 pulley

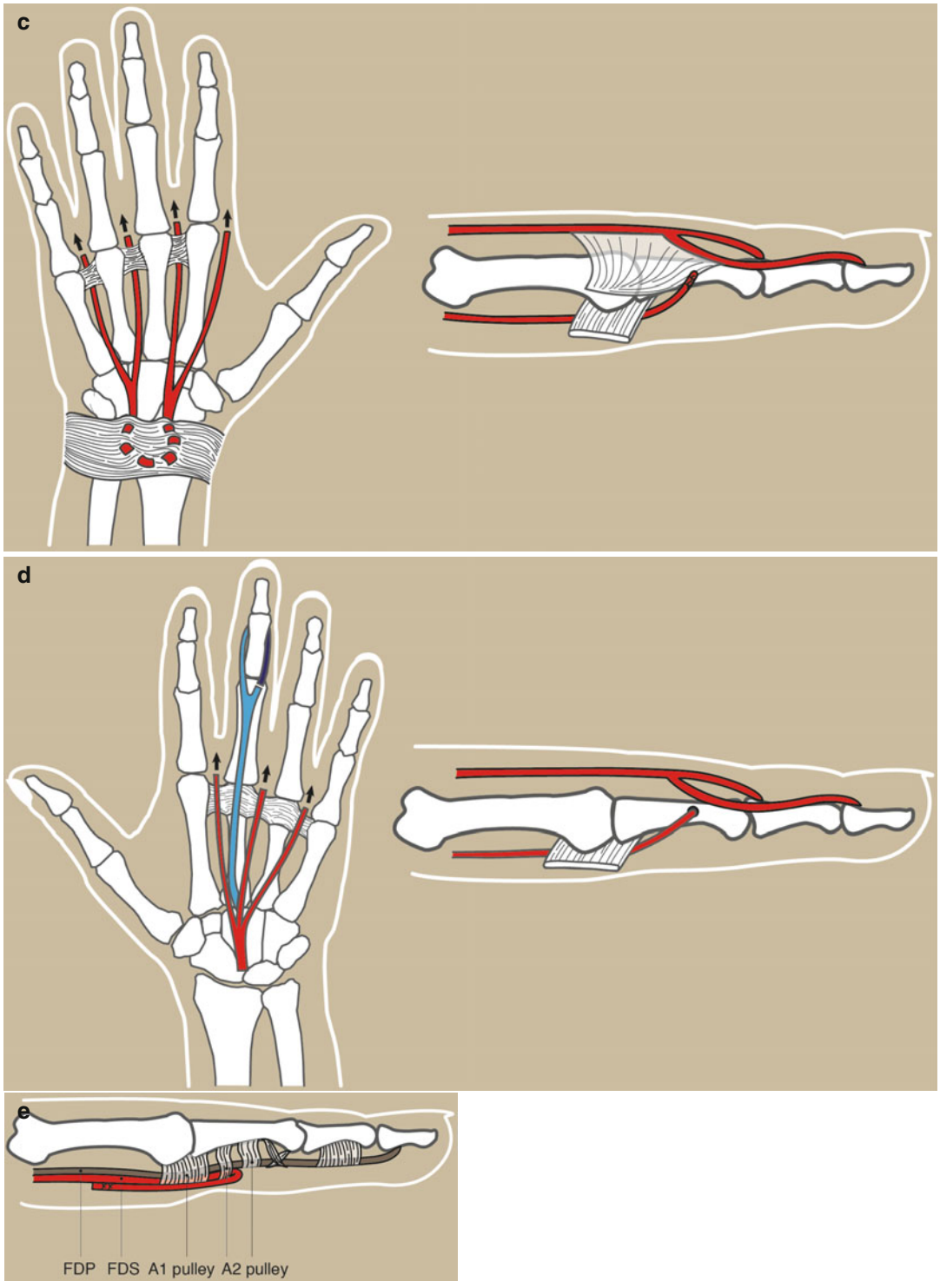


Fig. 2.9 (continued)

Zancolli describes the Zancolli Lasso procedure (Zancolli 2) where the FDS is passed deep to the A1 pulley and then brought out through the gap between A1 and A2 [21]. The concept is to create a flexion force at the MP joint. Omer modified this by suggesting that the FDS tendon could be brought out around the A2 pulley to increase this flexion moment (see Fig. 2.9e).

It is important to note that the static tenodeses and the FDS procedures do not increase the grip strength and secondly, harvesting the FDS might compromise an already weakened grip. To increase the grip strength, the intrinsic function needs to be "replaced". This can be done by using a wrist level motor for proximal phalanx power flexion.

Claw correction – creating a flexion moment at the MP joint
Static
Zancolli 1 MPJ volar plate capsulodesis
Parkes tendon graft from DTIL to lateral bands
Bunnel: Release A1 and part A2 causing bow stringing and increased flexion force
Fowler: Similar to Parkes but attached proximally to extensor retinaculum. Flexion moment is increase by flexing wrist.
Dynamic
Stiles, Bunnel, Riordan: Active FDS motor rerouted volar to DTIL and into lateral bands
Modified by Burkhalter: Insertion site into P1 to prevent swanneck
FDS around sheath pulleys and sutured back on itself – Zancolli 2 modified by Omer (Author's preferred technique).

Technique of Zancolli Lasso

An oblique incision is made in the proximal pulp of the middle finger. Both slips are carefully separated from the FDP tendon and sectioned. This incision is closed. The middle finger FDS is chosen as a motor because the FDP to this finger is functional as opposed to the FDP to the little and ring fingers. Sacrifice of the ring finger FDS would result in marked loss of flexion strength in this digit.

A transverse incision is made at the level of the distal palmar crease. The flexor sheaths of the middle, ring and little fingers are exposed and opened proximal to the A1 pulley. The FDS to the middle finger is tractioned and delivered proximally. It is split in two longitudinally. One slip is used for the ring finger and the other motors the little finger. Each slip is passed under the A1 pulley and under the proximal 2–4 mm of the A2 pulley. A transverse sheath incision is made at this level and the FDS slip delivered. It is sutured back on itself. The tension is set with the MPJ's flexed 60°. This is more than the original description of Zancolli but it is the author's opinion that the transfer is likely to stretch out over time. It is our policy to only correct the little and ring fingers rather than all the fingers as described by others.

Procedures to Increase Grip Strength

To increase MP joint flexion strength, a "new" muscle must be brought in from elsewhere. Available motors previously described include brachioradialis, ECRL, ERCB, and FCR. These motors need to be extended or elongated with free tendon grafts (palmaris longus, plantaris and the toe extensors). To further complicate matters, all the above transfers have been described with dorsal and volar passages. Dorsal transfers pass through the intermetacarpal spaces to the lumbrical canals and volar transfers pass through the carpal tunnel to the lumbrical canals. After passing volar to the DTIL, the tendons can be sutured either to the lateral bands, the flexor sheath or the proximal phalanx itself.

My preferred transfer is the ECRL (dorsal) four tailed procedure [22]. The palmaris longus is harvested and spit into four equal lengths. Two dissections are performed. A distal dissection on the radial side of each finger and on the ulnar side of the index to expose the lateral bands, and a proximal dissection to expose the ECRL tendon. The distal anastomoses are performed first.

The grafts are sutured to the lateral bands and passed through the lumbrical canals, volar to the DTIL and through the intermetacarpal space to exit dorsally. The grafts are tractioned to confirm flexion at the MP joints and extension at the IP joints. The graft ends are tagged and the distal incisions closed. ECRL is mobilized and the

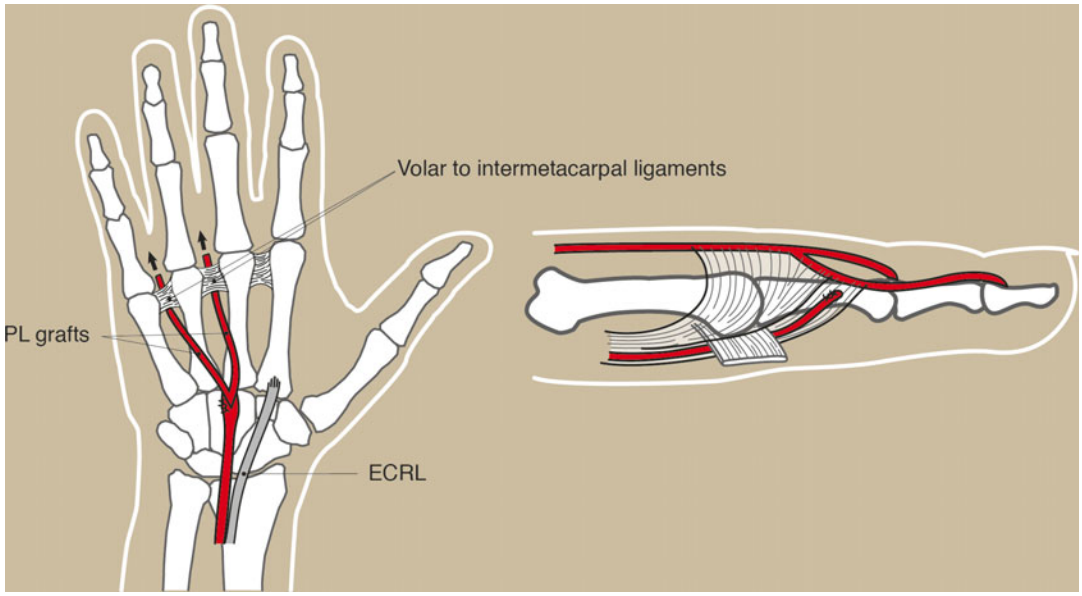
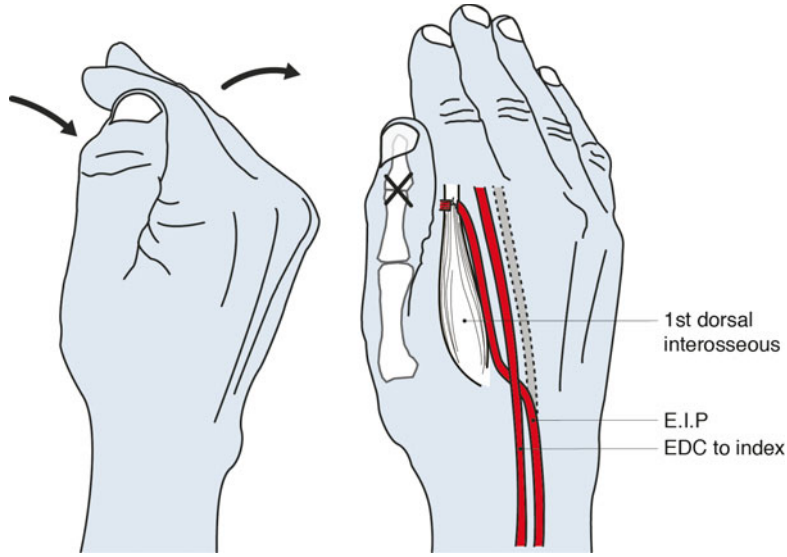


Fig. 2.10 Line drawing of ECRL four-tailed transfer for intrinsic replacement

Fig. 2.11 In ulnar nerve palsy, attempted pinch results in flexion at thumb IP joint and adduction of index finger at MP joint. Preferred pinch transfer – IPJ fusion and EIP to 1st dorsal interroseous



grafts are woven into the ECRL tendon. Tension is set with the wrist neutral and the grafts are pulled until the MP joints flex 40°. Post operatively, the wrist is held in extension and the MP joints in flexion to diminish the tension on the transfer. The problem with this transfer is that, while the wrist extensors and MP flexors are in phase, the dorsal pathway relies on wrist flexion to augment the strength of the transfer using the tenodesis effect. To correct this, Brand [23] performed some of these rerouting the ECRL through the carpal tunnel. This means that the ECRL contracts on attempted wrist extension and grip formation but is augmented by the tenodesis affect when the wrist is extended. The disadvantage of the volar approach is the passage of the tendons through the carpal tunnel resulting in possible carpal tunnel syndrome (see Fig. 2.10).

Procedures to Increase Pinch Strength

The ulnar nerve supplies all of the adductor pollicis, half of the flexor pollicis brevis and the 1st dorsal interosseous. Loss of thumb adduction and index abduction often, but not always results in marked weakness of pinch strength. Techniques to reconstruct pinch include a) transfers that increase thumb adduction, b) transfers that increase index abduction and c) thumb fusions.

Many transfers have been described to increase index finger abduction. The point of insertion is usually to the tendon of the first dorsal interosseous at the level of the base of the proximal phalanx. Motors that can be utilized without the need for an interposition graft include EIP and EPB. The use of a slip of APL as a motor needs to be extended with a free PL graft (Nevasier) [24]. Many authors would suggest that transfers to augment index abduction are seldom required. By stacking the fingers together, the tendency for the index finger to adduct can be blocked. Furthermore, these transfers are notoriously weak. I continue to use the EIP to first DI transfer and have been pleased with the results.

Recreating the line of pull is mandatory in thumb adduction transfers. If a wrist extensor is used then it must be lengthened with a free tendon graft through the 2nd or 3rd intermetacarpal space, across the palm to the adductor insertion. Alternatively, a ring or middle finger FDS is redirected by means of a palmar pulley across to the thumb MP joint. Others have used brachioradialis or EIP. A recent review by Fischer et al. [25] confirm that pinch strength is doubled from preoperatively and improves by between 40 and 73 % of normal.

Fusion of one of the thumb joints alone can improve grip strength by up to 2 kg. Thumb IP fusion prevents the Froment's sign and therefore increase the lever arm of the flexor pollicis longus. In some patients, with collapse of the MP joint into hyperextension (Jeane's sign), we prefer to fuse the thumb MP joint.

Many patients do not require a pinch transfer as they do not present with functionally limiting weakness of pinch. Correction of the claw deformity or reconstruction of integrated MP joint flexion takes priority. When required, we prefer a limited approach and perform an EIP to 1st dorsal interosseous transfer. This is combined with a fusion of the thumb IP joint. This simple technique reliably improves pinch strength from a pre-operative 2–3 kg to post operative pinch strength of 4–6 kg which is similar to the results achieved by more complex procedures. The lack of IP flexion will need to be discussed with the patient pre-operatively.

Procedures to increase pinch strength
Index abduction
EIP to first DI (author's preferred procedure)
APL plus PL to first DI
EPB to first DI
Thumb adduction
FDS 3/4 – palmar pulley
ECRB/ BR – palmaris longus extension
Thumb fusion
IP joint (authors preferred procedure)
MP joint

Ulnar Nerve Palsy – Closing Comments

In many parts of the world, ulnar nerve palsy remains a major cause of upper limb disability. The treating clinician is advised to understand the interactive forces governing the development of deformity and explore the myriad of treatment options available. A multiple disciplinary approach is absolutely mandatory to optimize outcomes. Unfortunately, whilst tendon transfers for ulnar palsy are technically pleasing, this remains a devastating hand injury for which patients will often transfer hand dominance or fail to return to premorbid activities and/or work. So critical are the intrinsic muscles to the fine and balanced dexterity of the hand that even a 75 % improvement will leave the patient somewhat compromised.

Median Nerve Palsy

Introduction

The median nerve is the "eye of the hand". Its massive cortical representation is testament to its

vital function in our daily world. Our sensory spectrum extends from being able to feel a single grain of sea sand, through huge temperature, texture and pressure differences. Loss of this critical 'organ' results in an extremely compromised hand and every effort must be made to restore this function. Fortunately the motor deficit is less debilitating and easier to reconstruct than ulnar nerve motor loss.

Anatomy

The median nerve enters the forearm medial to the biceps tendon where it runs with the brachial artery. The nerve passes between the two heads of pronator teres and supplies this muscle. It then passes deep to the FDS muscle to lie between FDS and FDP. The median nerve supplies all the FDS muscles. It gives off an anterior interosseous branch which supplies the FPL, FDP index and middle and finally the pronator quadratus. The main nerve continues to travel distally between FDP and FDP. Branches are given to FCR and PL. At the level of the distal forearm the nerve become more superficial and lies deep to and between the tendons of FCR and PL before it enters the carpal tunnel. At this stage it is mostly sensory to the thumb, index and middle and the radial half of the ring finger. A small motor branch supplies the thenar muscles including Opponens Pollicis, APB and part or whole of the FPB and the adductor pollicis.

Assessment

Median nerve palsy can be divided into high and low injuries. Low injuries occur at the wrist or distal forearm levels. At this level, the forearm extrinsic tendons have been innervated and this leaves only the intrinsics without motor supply. The median nerve innervated intrinsics include abductor pollicis brevis, opponens pollicis, the superficial head of flexor pollicis brevis and the medial two lumbricals. The clinically significant loss is to opposition. Opposition is a composite movement made up by palmar abduction and pronation followed by MPJ and IPJ flexion. In true opposition, the thumb can be brought opposite the other fingers such that the fingernails are in the same plane. Pseudo-opposition, uses FPB to bring the thumb to the other fingers but the fingernails end up perpendicular to each other (Fig. 2.12). Remember that a substantial percentage of patients with a low median nerve palsy will retain true opposition due to anomalous supply by the ulna nerve. It is vital to meticulously assess patients prior to performing a low median nerve palsy reconstruction as the most common method of median nerve injury is sharp laceration in the distal forearm which often results in injury to the flexor tendons as well. This could well affect the choice of tendons for reconstruction of opposition.

In high median nerve palsy, the long flexor to the thumb (FPL), all the FDS muscles and the lateral two FDP muscles to the index and middle fingers are not functioning. The clinical deficit is inability to flex these three digits when trying to make a fist – the so called Benediction sign (Fig. 2.13). All the FDS musculotendinous units are innervated by the median nerve but the little and ring fingers can still flex under the influence of the ulna nerve innervated FDP's. Before discussing the tendon transfer reconstruction of high and low palsies it is mandatory to mention again the critical sensation supplied to the hand by the median nerve. As discussed above, the median nerve has an enormous cortical representation indicative of its importance as a sensory organ and its critical role in the functioning of the hand. Suffice to say that the hand with a successfully reconstructed sensibility will do much better than that with poor sensation to the thumb, index and middle fingers.

Low Median Nerve Palsy

The surgical procedure required here is opponensplasty. This action can be recreated by applying a force from the APB tendon in an ulnar direction. To successfully restore opposition you need a motor, a pulley and an insertion. Remember one of the principles of tendons transfer is full passive range of motion. Both the surgeon and the hand therapist must be vigilant to maintain full passive opposition and avoid any contracture

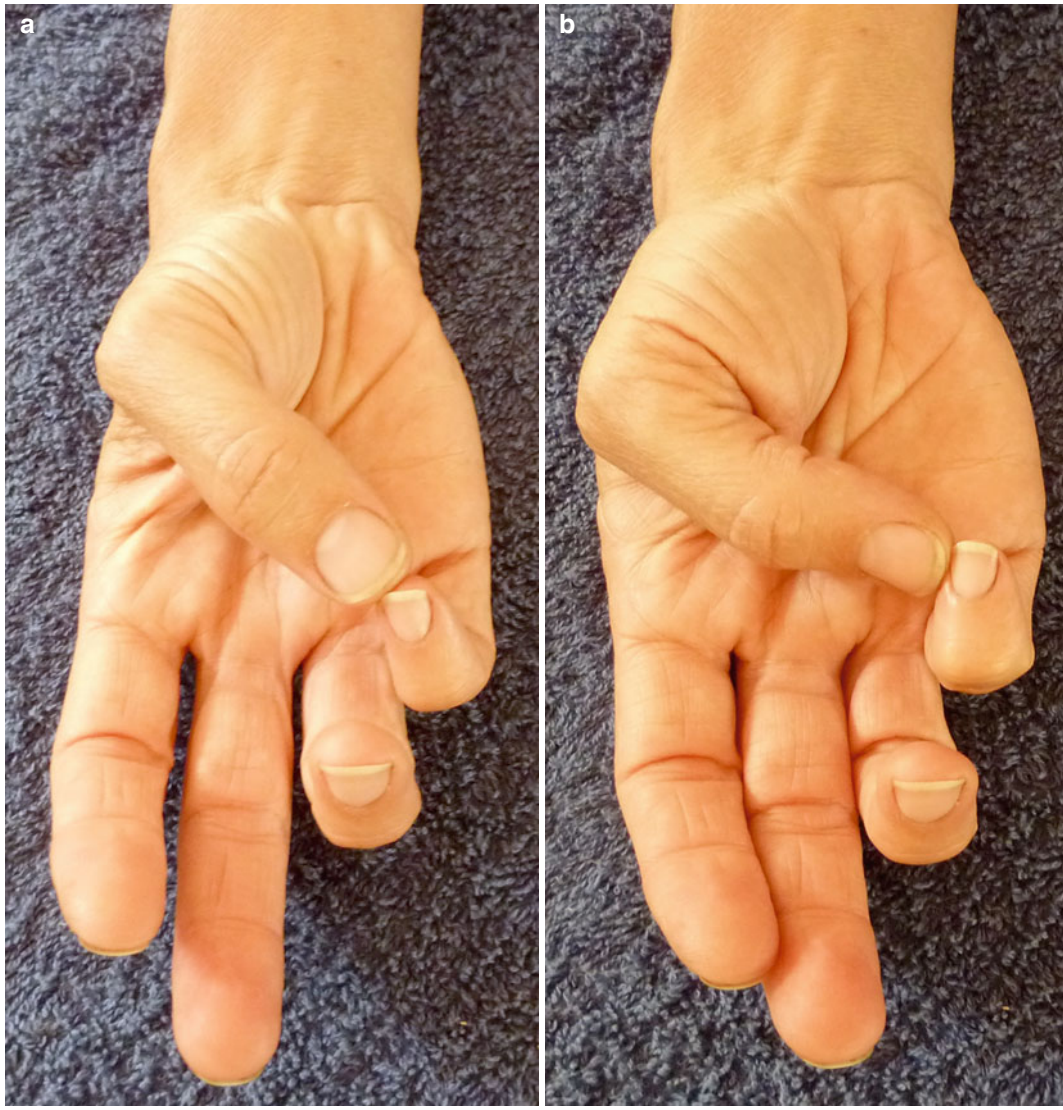


Fig. 2.12 (**a**) Pseudo-opposition – the thumb nail is perpendicular to palm. (**b**) True opposition – the thumb nail is parallel to palm

of the first web space. Often a short opposition splint or web space splint is utilized.

The most common motors are the FDS from the ring finger and the EIP. Royle [26] was the first to use the ring finger FDS. He brought the tendon out proximal to the wrist and tunneled it with the FPL. The attachment was distal in the thumb. Thompson [27] modified this by creating a mid palmar pulley around the palmar fascia at the distal end of the carpal tunnel. Bunnell [28] finally modified this option by creating a pulley at the level of the pisiform. A distally based slip of FCU is looped back on itself around the FDS which is tunneled subcutaneously across the palm to the thumb. The insertion will be discussed later. The FDS transfer is a useful and reliable option with two drawbacks. Firstly, harvest of the FDS to the ring can result in symptomatic swan neck deformity and secondly, there is commonly a concomitant injury to the FDS tendons in the wrist area which precludes this transfer.

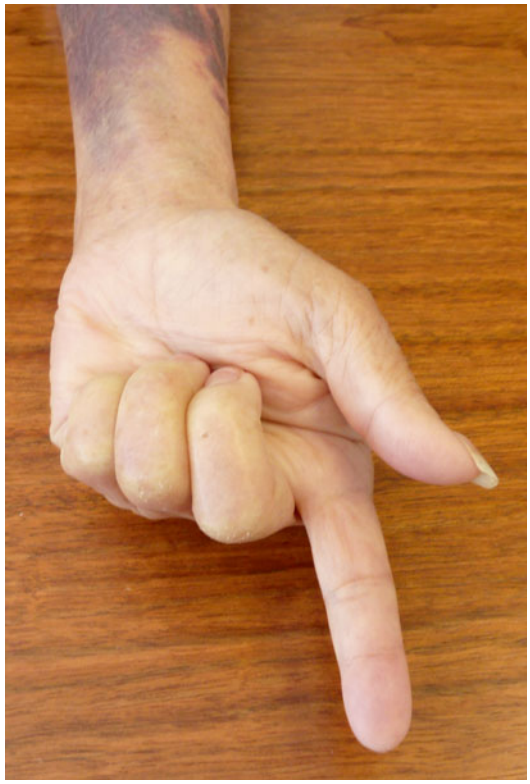


Fig. 2.13 Benediction sign of high median nerve palsy

The use of EIP has many advantages and few disadvantages. It is out of the zone of injury and has little or no donor site morbidity. The pulley is the ulnar side of the hand and the tendon is routed from the pisiform across to the thumb.

Many different techniques of distal transfer attachment have been described. They all include at least an attachment to the APB tendon. This allows a combination of palmar abduction and some pronation. All the original descriptions by Royle, Thompson and Bunnell advise attachment to the bone. In order to improve thumb rotation Brand and Riordan felt the attachment should be on the dorsal and/or ulna side of the thumb MP joint. Riordan's attachment to the dorsal capsule including EPL can result in marked thumb hyperextension especially if the FPL is non-functional (in high median nerve palsy). Brand has suggested attachment all the way over the top to the adductor. It was Littler and Li [29] who inferred that attachment only to APB was sufficient to achieve reasonable opposition. A biomechanical study by Cooney et al. [30] showed that thumb abduction and flexion results in obligatory opposition

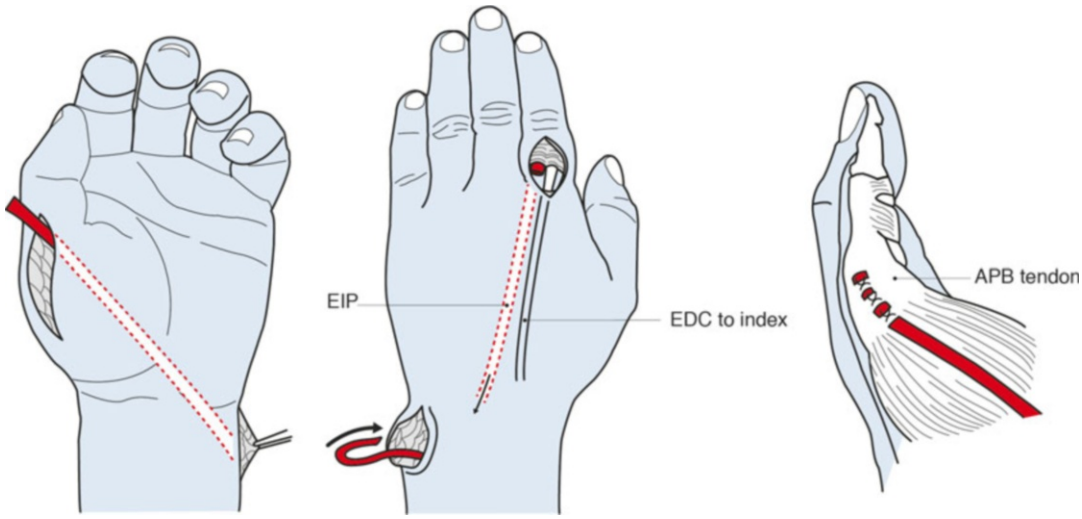


Fig. 2.14 Line drawing of EIP opposition Transfer

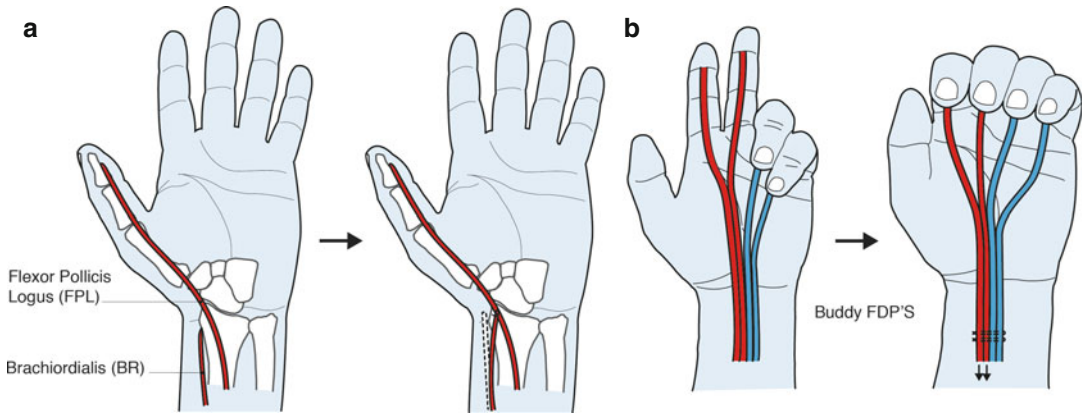


Fig. 2.15 Line drawing of (**a**) BR to FPL and (**b**) Buddy FDP's

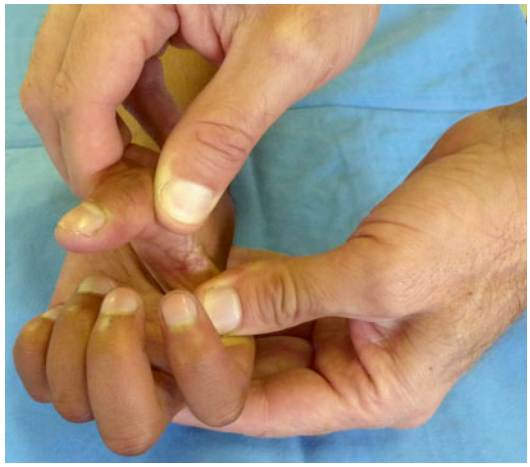


Fig. 2.16 An example where an attempt to reconstruct opposition will fail. Patient cannot initiate flexion of the fingers at the MP joint and must 'roll-up' the fingers. These will miss the opposed thumb. Rather try to achieve strong lateral key pinch

due to the anatomical shape of the thumb CMC joint. Most (including this author) agree with this concept.

Other motors use include EPL, FPL, ECU, ECRL, EDM, PL and abductor digiti minimi. These last two deserve mention. Historically, many patients presented with end stage carpal tunnel syndrome categorized by severe thenar wasting. A slip of palmaris longus was elevated in the carpal tunnel incision and tunneled subcutaneously to the thumb. This operation, called the Camitz [31] procedure, results in reasonable reconstruction of palmar abduction but poor flexion and pronation.

The use of ADM is called the Huber [32] transfer and is almost exclusively used in the reconstruction of opposition in cases of congenital thumb deficiency.

High Median Palsy

As mentioned previously, the motor deficit in these patients is to the long flexor of the thumb together with the long flexors to the index and middle fingers. This is in addition to the loss of opposition distally.

Opposition can be reconstructed by means of EIP. FDS ring finger as a motor is not available because of denervation of all the FDS muscles. Thumb IPJ flexion is augmented by a brachioradialis transfer. The brachioradialis needs to be extensively mobilized to create sufficient excursion to reasonably power the FPL. A simple side to side suture of the long flexors will buddy the functioning of the FDP little and ring, to those of the index and middle fingers.

Short Notes on Combined Palsies

Probably the most common of these includes the low median together with the low ulnar nerve palsy. These two nerves are located relatively superficial and close to each other in the distal forearm. The motor and sensory deficit is further complicated by invariable injury to the long flexor tendons as well. The hand assumes an intrinsic minus position with marked clawing of the fingers and absent opposition. Before embarking on opposition reconstruction, it is vital to first reconstruct integrated flexion of the MP joints using a radial nerve donor. The ECRL – four tailed graft operation should be utilized. If this transfer is successful and the fingers can flex from the MP joints rather than "roll up", then an EIP opposition transfer can be performed. If not, then it is better to aim for strong lateral key pinch as discussed. A dogmatic attempt at reconstructing opposition, in a hand where the fingers cannot be brought opposite the opposed thumb is a common mistake that must be avoided (see Fig. 2.16). For the rest of the combined palsies, the reader is referred back to the section on principles of tendon transfers where the assessment of what is required and what is available is the first step in a difficult algorithm.

Pearl

Do not reconstruct opposition if the opposed thumb will bypass the roll up action of the fingers.

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Extensor Tendon Reconstruction

3

Simon Ball and Rupert Eckersley

Keywords

Extensor tendon • Extensor mechanism • Mallet • Boutonniere • Sagittal band • Extensor Digitorum Communis • Extensor Pollicis Longus • Reconstruction • Reconstructive surgery • Hand • Hand function • Finger • Finger function • Stiffness

Introduction

The reconstruction of the extensor mechanism is not as simple as flexor tendon reconstruction because of the complex nature of the anatomy and kinesiology particularly within the finger. The reconstruction of the long extensors is straightforward involving either tendon buddying, tendon grafting or tendon transfer with a reasonably predictable result. The reconstruction of the extensor mechanism in the finger is far harder and much less predictable. It should always be remembered that the purpose of the hand is to hold onto objects and manipulate them. The worst thing a surgeon can do to someone's hand is to interfere with that function and in attempting reconstruction of the extensor mechanism cause loss of flexion and so inhibit that vital function. In some situations, for example in the burnt hand, the amount of fibrosis may act as a form of elastic recoil such that attempts at extensor tendon reconstruction may be futile and inappropriate. Realistic goals are vital in this area of reconstructive surgery. Therefore it is important that the function of the hand is assessed properly by an experienced hand therapist and the surgeon should discuss with the therapists what the realistic objectives are. Far too often surgeons do surgery that may satisfy their own objectives but fail to reach the patients expectations.

Summary Box/Key Points

Patients may have little functional deficit and do not require surgery

Extensor tendon reconstruction can cause stiffness and impair hand function

A finger must have full passive mobility prior to any surgical reconstruction

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Outcomes can be unpredictable and there are no large outcome studies for any technique described
The patient must be aware of the pros and cons before embarking on complex reconstruction
This section will deal with the individual deformities the surgeon comes across on a regular basis.

Chronic Mallet Deformity

Aetiology

Mallet finger injury is a discontinuity of the terminal extensor tendon resulting in an extensor lag. This is usually a closed injury caused by sudden flexion of the DIP joint with either a rupture of the terminal tendon or a bony avulsion of the terminal tendon attachment to the distal phalanx. It can also occur as the result of an open injury with division of the terminal tendon. The commonly affected fingers are the small, ring and middle. The majority of the injuries will heal satisfactorily if managed acutely. If however there is delayed presentation or failure of the initial management a chronic mallet deformity may develop.

When there is discontinuity of the terminal tendon the extensor tendon force is concentrated on the PIP joint with a resultant tendon imbalance occurring at this joint. With time this may lead to attenuation of the volar structures allowing hyperextension of the PIP joint and the development of a swan neck deformity.

Assessment

A careful history should be taken to establish the time of injury and the functional deficit that the patient experiences. It is important to establish what the patients' aims and expectations are before considering any treatment options. The passive range of movement of the finger in question should be assessed and the degree of extensor lag documented. In most chronic mallet deformities the tendon will have attempted healing and formed a pseudotendon. The PIP joint should be carefully assessed for evidence of hyperextension and resultant swan neck deformity. An x-ray should be taken to look for subluxation of the joint, non-union of a bony avulsion, and osteoarthritic change.

Treatment Options

Tendinous mallet injuries presenting several months after injury may be treated with extension splinting [1] and many surgeons will undertake a trial of extension splinting before considering any surgical intervention.

The first option is to take no further action. The loss of extension at the terminal joint may not cause any significant loss of function. The surgical treatment may result in a stiff joint and this can lead to loss of function if grip is interfered with by loss of full flexion.

If reconstructive surgery is considered it is important that all joints of the finger have a full passive range of movement.

If the deformity is exclusively at the DIP joint then surgery may be focussed at this level with the aim being to shorten the "healed" tendon. Several methods have been described including tenodermodesis, excision of scar and reattachment to the distal phalanx using a bone anchor or pull out suture, and plication of the "healed" tendon. This type of surgery will not however correct any hyperextension at the PIP joint and in this situation a central slip tenotomy or oblique retinacular ligament reconstruction may be considered.

DIPJ fusion is also an option particularly if the joint is degenerate but again this does not address any deformity at the PIP joint.

The surgical options are shown in Table 3.1.

Table 3.1 Surgical options for the treatment of chronic mallet deformity

Surgical options for chronic mallet deformity	
Principle	Technique
Tendon "repair" at level of DIP joint	Tenodermodesis
	Excision of scar and re-attachment of tendon back to bone
	Tendon plication
Tendon rebalancing	Central slip tenotomy
Tendon reconstruction	Oblique retinacular ligament reconstruction
Arthrodesis	

Surgical Techniques and Rehabilitation

Tendon Plication

Under digital block a curved incision is made over the terminal tendon. The pseudotendon is exposed. There are two methods described to shorten the tendon. The first is a simple plication leaving the tendon intact and the second is to divide the tendon and then to double breast the divided tendon. In both cases once the sutures have been placed the DIPJ should be in full extension. A K-wire is passed across the DIP joint to maintain full extension for 6–8 weeks. After K-wire removal mobilisation of the DIP joint is commenced with night splintage for a further 4 weeks.

Tenodermodesis (Dermatotenodesis)

This technique was originally described by Brooks in 1961 and later modified by Graner and is therefore sometimes referred to as the Brooks and Graner technique.

The operation is performed under digital block. An elliptical incision is made over the DIP joint and a wedge of skin and subcutaneous tendinous scar tissue is excised. The size of the excised ellipse may be determined by passively extending the DIP joint to produce a dermal fold over the DIP joint preoperatively. The wound edges should come into contact when the DIP joint is extended. The wound is closed using en bloc mattress sutures passing through skin and tendon on both sides of the wound. A K-wire is passed across the DIP joint to maintain full extension for 4 to 6 weeks. The rehabilitation programme is as for tendon plication.

Central Slip Tenotomy

This technique relies on a mature healed but elongated terminal tendon resulting in a persistent extensor lag. At least six months must have passed since the time of injury to allow full maturation of tendon healing. The maximum extensor lag that can be corrected with this technique is 35° [2]. There must also be a full passive range of motion of the DIP and PIP joints. The technique involves dividing the central slip which allows the extensor tendon mechanism to slide proximally thus increasing the tone in the terminal tendon which in turn improves the extensor lag. A boutonnière deformity is not created as the triangular ligament is left intact.

The procedure is best performed under digital block to enable active tendon movement intraoperatively. The tendon is exposed using a midlateral incision centred over the PIP joint. An extensor tenolysis may need to be performed over the middle phalanx as the central slip must not be prevented from migrating proximally by distal adhesions. The triangular ligament must not be damaged. The transverse retinacular ligament is divided and the entire extensor mechanism is elevated both proximal and distal to the central slip insertion. The central slip insertion is divided by sliding a scalpel from proximal to distal to take the insertion off the base of the middle phalanx. On active extension there should be improvement of the extensor lag at the DIP joint. If no improvement occurs the surgeon should perform an extensor tenolysis ensuring that the extensor mechanism is not tethered and is able to slide proximally. The finger should then be splinted with the PIPJ in 30° flexion and the DIPJ in full extension.

Post-operatively a dorsal blocking splint is used to maintain at least 20° of flexion at the PIP joint whilst the DIP joint is splinted in full extension. The aim is to maximise the extensor tendon force at the DIP joint. At 2 weeks the PIP joint splint may be discarded and full active mobilisation of this joint may be commenced although hyperextension should not be allowed. At this stage the patient may remove the DIP joint splint several times each day to mobilise the joint. At 4 weeks the DIP joint splint need not to be worn during the day but should be worn at night for a further 4 weeks.

Oblique Retinacular Ligament (ORL) Reconstruction

The ORL originates from the flexor sheath on the volar aspect of the PIP joint and inserts into the terminal tendon. During PIP joint extension the ORL tightens and assists in extending the DIP joint.

Reconstruction involves the use of a tendon graft (palmaris or plantaris). The graft is secured dorsally into the base of the distal phalanx which may be done using a pull out suture or bone anchor. The graft is then directed in a spiral fashion around the middle phalanx passing between the flexor sheath and the neurovascular bundle obliquely across the volar aspect of the PIP joint to the opposite side. The graft is then passed transversely through the base of the proximal phalanx and secured in place once the correct tension is achieved. Care must be taken not to over tighten the graft as this may result in a boutonnière deformity.

Outcomes and Complications

Several studies have reported on the outcomes of various techniques used in the management of chronic mallet deformity and are summarised in Table 3.2. Unfortunately, due to the heterogenicity of the studies and the small numbers involved it is difficult to make firm conclusions.

Tenodermodesis has been shown to give a reliable improvement in extensor lag although some studies involved only paediatric patients. Similarly excision of tendon scar and re-attachment using a bony anchor can give good results. All the patients undergoing surgery had a full passive range of motion pre-operatively. Surgery on the DIP joint does carry a risk of infection and stiffness but both complications were reported as being negligible in the papers reviewed.

Central slip tenotomy has been shown to give reliable results but can only improve the extensor lag by approximately 35°. Poor results have been associated with a pre-operative extensor lag of greater than 30°. The standard post-operative splinting protocol is described, however, Houpt et al. [10] does not believe that any immobilization is necessary. The aim of the rehabilitation is to achieve maximal proximal slide of the extensor apparatus while not causing an extensor lag or fixed flexion deformity at the PIP joint.

ORL reconstruction is used in patients who have developed secondary hyperextension deformity at the PIP joint. Thompson et al. [12] and Kleinman et al. [11] have reported reliable correction of both the PIP joint hyperextension and DIP joint extensor lag. However approximately 10 % of patients had the graft overtightened and required revision surgery. A further 5 % of patients required tenolysis. Again only small numbers of patients have been reported on.

Conclusions/Authors Preferred Approach

In the majority of cases our recommendation is to advise the patient to accept the deformity as the results of surgery are unpredictable and may leave them with a stiff finger with consequent loss of grip. Where the extensor lag is a functional problem our preferred approach is to shorten the tendon using a double breast technique. We have found this to give a good result in improving the extension of the joint without creating stiffness which is the reason we do not favour tenodermodesis. For those patients with a correctable swan neck deformity we prefer central slip tenotomy again this seems to achieve a good result. We prefer to use a ORL reconstruction in swan neck deformities caused by primary PIPJ pathology.

Table 3.2 Outcomes of different surgical techniques [3–12]

	No Pts	Pre-op deformity	Duration of deformity	Follow up	Technique	Results	Remarks
Surgery at the level of the DI P joint only							
Boeck et al. (1992)	4	>4 months			Tenodermodesis	100 % full extension	All children. No bony component.
Sorene et al. (2004)	16	30–70°	12–75 weeks	10–60 month	Tenodermodesis. K-wire for 4 weeks.	50 % full extension 37.5 % ext lag <20° 12.5 % ext lag >20°	No pts had restriction of flexion.
Ulker et al. (2005)	22	Mean 43.6° ext lag	4–30 month	6–28 months	Scar excised, re-attachment using bone anchor to create 5° ext, k-wire for 2.5 weeks then mobilise	15 full extension 5 ext lag 10° 2 ext lag 10–20°	Open and closed injuries included.
Kardestuncer et al. (2008)	10	>45° ext lag	>6 weeks	1–12.8 years	Tenodermodesis. 4–6 weeks immobilisation post op.	20 % full extension 80 % ext lag <20° 70 % full flexion	All children – age at surgery 1.4–17.8 years
Central slip tenotomy							
Bowers and Hurst (1978)	5	45–75°	>6 months	>1 year	Central slip tenotomy	80 % full extension 20 % ext lag 10°	1 pt had 12° ext lag at PIP jt
Grundberg and Reagan (1987)	20	Mean 37°			Central slip tenotomy	Mean ext lag 9°	
Lucas (1987)	11		>6 months	>1 year	Central slip tenotomy	80 % ext lag <10° 20 % ext lag >30°	11 of 16 pts reported
Houpt et al. (1993)	35	Mean 45°	>4.5 months		Central slip tenotomy	74 % full extension 23 % ext lag <20° 3 % ext lag >30°	Immediate motion post-op protocol

(continued)

Table 3.2 (continued)

	No Pts	Pre-op deformity	Duration of deformity	Follow up	Technique	Results	Remarks
Spiral oblique ligament reconstruction							
Kleinman and Peterson (1984)	12	8 pts had hyperext of PIP jt			Soft tissue attachment of palmaris longus graft to terminal tendon only. Graft then routed obliquely across the palmar aspect of the PIP joint and attached to the fibro-osseous flexor sheath. Axial K-wire to DIP jt in neutral and oblique K-wire across PIP jt in 10–15° flexion. Proximal wire removed at 3 weeks and distal wire at 4.5 weeks with additional splint for 1.5 weeks of DIP jt.	100 % full extension of DIP jt. 25 % 10–25° hyperextension at DIP jt. Hyperextension of PIP jt corrected in all 8 pts.	One pt had 35° PIP flexion contracture requiring lengthening of graft. One pt required flexor tenolysis.
Thompson et al. (1987)	10			>3 months	Bony attachment of palmaris tendon graft to dorsal distal phalanx and transversely across base of proximal phalanx.	70 % full extension 30 % 10–15° ext lag	Open patient required revision for over correction.

Chronic Zone III Injury (Boutonnière Deformity)

Aetiology

The primary cause of a chronic Boutonniere deformity is loss of central slip function. This may be due to failure of treatment or a delayed presentation of an acute injury. It may also be caused by a slow attenuation secondary to osteoarthritis, inflammatory arthritis or a prolonged flexion contracture which may be seen secondary to a burn or Dupuytren's disease.

The pathophysiology of the deformity is that loss of central slip function will result in an extensor lag at the PIP joint and hyperextension at the DIP joint as the extensor force is now concentrated on this joint. Over time the triangular ligament stretches which allows the lateral bands to subluxate palmarly and to then lie palmar to the axis of rotation of the PIP joint. The flexor tendons and lateral bands cause increased force of flexion at the PIP joint and because of the relative shortening of the lateral bands an increased extensor force at the DIP joint. The development of this deformity occurs over time and the initial posture of the finger after division or rupture of the central slip may not be abnormal.

Initially the deformity will be flexible with a full passive range of motion of the interphalangeal joints. However, with time the lateral bands will contract causing a fixed deformity. Consequently a joint contracture may develop secondary to contracture of the collateral ligaments and volar plate.

Burton classified chronic boutonnière deformity into three stages with a fourth stage being added later:

- Stage I: Flexible deformity (passively correctable)
- Stage II: Fixed deformity due to contacted lateral bands
- Stage III: Joint contracture due to joint fibrosis and contacted collateral ligaments and volar plate
- Stage IV: OA of the PIP joint plus stage III

Assessment

The history should establish the cause of the deformity (as above), treatment to date, and the degree and rate of progression.

The severity of the deformity and whether it is flexible or fixed should be documented. Careful consideration must be given to the functional deficit the patient experiences as most patients with a boutonnière deformity retain full flexion and grip strength. Any treatment aimed at correcting the deformity and gaining extension can jeopardise flexion and therefore impair hand function.

It is important to differentiate a true boutonnière deformity from a "pseudoboutonnière" deformity where there is flexion contracture of the PIP joint without a fixed DIP extension deformity. This is most commonly caused by a hyperextension injury resulting in damage to the volar plate which subsequently scars and contracts and is also seen in Dupuytren's disease with ORL contracture.

Treatment Options

As with chronic mallet deformity the first option may be to advise the patient to have no treatment. This is particularly so if the finger remains flexible at both the PIP and DIP joints and no progression has been noted over time.

If reconstructive surgery is considered it is important that all joints of the finger have a full passive range of movement.

Stage II deformities should be managed with hand therapy using splinting or serial casting to achieve full passive PIP joint extension. Once PIP joint extension is achieved it should be maintained with the use of a splint for 6–12 weeks. During this time active and passive exercises of the DIP joint should be done which help realign the lateral bands and rebalance the extensor mechanism. If full PIP joint passive extension and active DIPJ flexion is achieved and an extensor lag persists then surgery can be considered.

Surgical reconstruction to correct the deformity must never be attempted in the presence of a

Table 3.3 Surgical options for the treatment of chronic boutonierre deformity

Surgical options for chronic boutonierre deformity	
Principle	Technique
Tendon repair	Excision of scar and re-attachment of central slip with or without V-Y advancement
Tendon rebalancing	Terminal tendon tenotomy
	Mobilisation and relocation of lateral bands
Tendon reconstruction	Lateral band transfer
	Using slip of FDS

fixed deformity. The first aim should be to achieve a full passive range of movement and if hand therapy fails then a surgical joint release should be done as a first stage. Following the joint release the patient should undergo appropriate splinting and an exercise program. In some patients the extensor mechanism will rebalance and thus the second stage reconstruction will not be required.

The surgical options for correction of a chronic boutonniere deformnity are shown in Table 3.3. Some of these are discussed below.

Surgical Techniques and Rehabilitation

Excision of Scar and Re-attachment of Central Slip

A curvilinear incision is made over the PIPJ under local anaesthetic. As in mallet injuries the central slip injury may result in pseudotendon formation. In a similar manner the pseudotendon and any scar tissue is excised and the native central slip is advanced and repaired to the base of the middle phalanx using bone anchors or a pull out suture. If the central slip is too tight a formal V-Y advancement may be performed to gain more length. A k-wire is inserted across the joint for a period of six weeks during which time the DIPJ is mobilized. After the wire is removed the patient will continue with a splint both active and dynamic for a further 4–6 weeks.

Terminal Tendon Tenotomy

Division of the terminal tendon enables proximal migration of the extensor mechanism which in turn increases the extensor force at the PIP joint, thus improving the extensor lag. This technique may be used in patients with full passive extension of the PIP joint and is contraindicated in patients with a fixed PIP joint flexion deformity. A dorsal incision is made over the middle phalanx. The terminal tendon is divided transversely, distal to the triangular ligament. The ORL should be identified and preserved. Although a "surgical mallet finger" is created, clinically a mallet finger rarely develops which is thought to be due to function of the intact ORL and partly due to the contracted capsule and ligaments of the DIPJ causing rebound extension after the joint is flexed. Postoperatively active movement of the finger is commenced immediately although a resting extension splint for the DIP joint is worn in between exercises for 6 weeks.

Lateral Band Mobilisation and Relocation: Littler-Eaton Technique [13]

In this procedure all the active extensor force at the DIP joint is released and is concentrated on the PIP joint thus improving the flexion deformity proximally whilst enabling correction of the hyperextension deformity distally.

Through a dorsal incision the lateral bands are identified. Using sharp dissection the lateral bands are freed from the lumbrical tendon and the spiral oblique retinacular ligament thus completely separating the extrinsic and interosseous intrinsic tendons from the lumbrical and oblique retinacular fibres. The liberated lateral bands are folded dorsally and are sutured to each other and to the remnant of the central slip in the midline whilst the PIP joint is held in full extension. If there is excessive redundancy of the attenuated central slip the relocated lateral bands should be sutured into the base of the middle phalanx. The joint is held in full extension with a K-wire for 2 weeks post-surgery after which mobilisation begins.

Free Tendon Graft Reconstruction

Many techniques have been described which utilize a free tendon graft to reconstruct the central slip. In a method described by Littler a tendon graft is passed through a bony tunnel in the base of the middle phalanx and is then passed dorsally around the lateral bands in a figure of eight configuration and is finally sutured proximally to the interosseous tendons.

Lateral Band Transfer: Matev Procedure [14]

The lateral bands are identified and released from any scar tissue so that they are mobile. The lateral bands are divided at different levels – one proximal and one distally over the middle phalanx. The lateral band which was divided more proximally is passed through the remnant of the central slip and sutured to the base of the middle phalanx to reconstitute the central slip. The lateral band cut more distally is then sutured to the stump of the opposite lateral band hence lengthening the terminal extensor mechanism. This decreases the extensor tone at the DIP joint enabling correction of the hyperextension deformity. The MP joint is held in 45° of flexion and the IP joints in full extension for 20 days post-surgery. A modification of the procedure has been described in which the immobilisation is for 6 weeks post-surgery.

Reconstruction of Central Slip Using Distally Based FDS Slip [15]

A midlateral incision is used to give access to the flexor sheath and also to the dorsal aspect of the PIP joint. The flexor sheath is exposed between the A2 and A4 pulleys. A short transverse incision is then made at the level of the distal palmar crease and the FDS tendon exposed just before the A1 pulley. One half of the FDS is tenotomised, leaving the residual FDS tendon intact and functioning. The divided half of the tendon is then drawn back to the level of the window created between the A2 and A4 pulleys. A 3 mm hole is then drilled obliquely from dorsal to volar in the base of the middle phalanx, entering where the central slip inserts. The distally based slip of FDS is then delivered from volar to dorsal using an interosseous wire loop. A substantial tendon length emerges from the dorsal aspect of the middle phalanx at the anatomic site of the central slip insertion. The slip of FDS tendon is then weaved into the extensor tendon over the proximal phalanx to reconstruct the central slip.

Postoperative therapy is the same as for other extensor tendon repairs at the same level. A static gutter splint may be used and intermittent early active mobilisation within a "short-arc" regimen may be commenced immediately, increasing the range over a 4 week period.

Curtis' Staged Reconstruction [16]

This procedure is performed under local anaesthetic to enable the surgeon to assess active extension after each stage. If adequate active extension is achieved the operation is stopped; if not the surgeon proceeds to the next stage. The 4 stages described are as follows:

1. A lazy S incision is made centred over the PIP joint. The transverse retinacular ligament is freed both proximally and distally and an extensor tenolysis performed. If full active extension is achieved the operation stops.
2. The transverse retinacular ligament is divided which enables the lateral bands to move dorsally. If full extension is achieved the finger is then splinted with the IP joints at 0° and the MP joint at 70°. After one week dynamic splinting of the PIP joint is performed.
3. If after stage 2 the extensor lag is greater than 20° the surgeon should bypass stage 3 and proceed to stage 4. If the lag is 20° or less then the surgeon should perform a step cut tendon lengthening of the lateral bands over the middle phalanx.
4. The central slip is dissected free and advanced approximately 4–6 mm after the intervening scar tissue has been excised, and attached to the middle phalanx. The lateral bands will now be slack and so are loosely sutured to the central tendon.

Outcomes and Complications

Stiffness is the most common complication of complex extensor tendon reconstruction. Therefore, a pre-requisite for surgical reconstruction is a fully

mobile finger. Only a limited number of patients are suitable for complex reconstruction and as a consequence there are no large outcome series available in the literature.

In patients who have a mild flexible deformity excision of the scar and re-attachment of the central slip has been shown to give good results and Le Bellec reports that 33 of 34 patients had excellent results [17]. The modified Matev procedure has been reported to give 85 % good or satisfactory results in 14 patients who had full passive movements pre-op as opposed to 67 % good or satisfactory results in 6 patients who had a pre-op PIP joint contracture [18].

Curtis [16] reported on 23 patients who were treated with a staged reconstruction and followed up for 1 year. Seventeen patients had an average pre-operative extensor lag of 41° and were successfully managed by some combination of stages I, II and III with an average extensor lag of 10° post-operatively. Six patients had an average extensor lag of 55° pre-operatively and required stages I, II and IV. The average extensor lag post-op was 17°. Twenty patients had increased flexion ability which was measured in relation to the distance from the distal palmar crease on maximal flexion.

Conclusions/Authors Preferred Approach

The problem with chronic Boutonniere deformities is, as with all extensor reconstruction, that the consequences of surgery may be to create a stiff finger with loss of PIPJ movement and so a reduction in function. Therefore the most important step is to have a proper discussion with the patient as to the functional problems in their hand, and part of this discussion should include a hand therapy assessment. We would strongly advise against surgery unless there is significant loss of function and the prospect of a functional improvement is therefore good. We have tried nearly all the above procedures with varying degrees of success and would not recommend one above the others.

It is important however to recognise that deformities of 30° or less are very difficult to improve with surgery and that the consequences of surgery maybe to create a stiff finger with loss of PIPJ movement and so a reduction in function.

Sagittal Band Reconstruction

Aetiology

The sagittal bands arise from the volar plate of the MP joint and the intermetacarpal ligaments and insert into the extensor hood. They maintain the central position of the extensor tendon at the level of the MP joint. A superficial and deep layer of the sagittal band has been identified. Ischizuki [19] noted that with "spontaneous" dislocation of the extensor tendon only the superficial layer of the sagittal band was disrupted whereas in the traumatic dislocation both the superficial and deep layers are torn. Rayan and Murray [20] later classified sagittal band injuries into three types. Type I is a contusion without tear of the sagittal band and without instability. Type II involves tearing of the sagittal band with snapping of the extensor tendon but without complete dislocation. Type III consists of tendon dislocation into the groove between the metacarpal heads. Shinohara et al. [21] suggested that laxity of the extensor mechanism at the metacarpophalangeal joint predisposes patients to traumatic extensor tendon dislocation.

The sagittal band may be damaged secondary to open or closed trauma although closed trauma is more common. Acute closed sagittal band rupture may be caused by a direct blow to the MP joint or by resisted extension of the finger which is often seen in the elderly. Chronic sagittal band rupture or attenuation may be caused by chronic repeated injury as in boxing or an inflammatory arthritis. Otherwise failure of closed treatment or delayed presentation of an acute injury is the usual cause. The radial sagittal band of the middle

finger is usually involved followed by ring and little finger involvement. Ulnar sagittal band rupture has also been reported.

Wheeldon [22] observed that the cross-sectional shape of the extensor tendon of the middle finger over the MP joint is rounder and less well anchored than that of the other extensor tendons at that level. Kettlekamp et al. [23] demonstrated that the oval long extensor tendon is more prone to dislocate because the extensor hood is attached more distally from the joint than that of the adjacent tendons.

Assessment

The symptoms and signs will depend on the degree of the tear. In the early stages the patient will describe a snapping sensation felt as the extensor tendon moves back and forth from between the metacarpal groove when the joint is flexed and the central anatomical position with the joint in extension. Care must be taken not to mistake it for a trigger finger. The examiner should look carefully as the joint is flexed and extended. The snap will be seen and can then be prevented by firm pressure on the ulnar side of the tendon during flexion. The extensor tendon can become trapped in the intermetacarpal groove during flexion and the patient will not be able to actively extend the finger thus presenting with a "dropped finger". The examiner or the patient can usually bring the finger passively into full extension and the extensor tendon will relocate and the patient will be able to maintain extension against resistance.

The diagnosis may be confirmed with ultrasound which is the investigation of choice as dynamic imaging may be performed. An MRI scan may also be performed to delineate the extent of the soft tissue injury.

Treatment Options

Patients presenting more than 3 weeks from injury, those who have failed non operative management and those with chronic traumatic injury are candidates for surgery. In inflammatory disease the sagittal band rupture may apply to all four fingers and under those circumstances and where the MP joints are not damaged the procedure of choice is a centralisation of all four tendons.

There are many different soft tissue procedures described to achieve centralisation of the EDC tendon. These may be classified as follows:

- Tendon balancing by partial release of the ulnar sagittal band.
- Excision of scar and "primary repair" or imbrication of attenuated sagittal band
- Sagittal band reconstruction using Junctura Tendinum (Wheeldon) [22]
- Sagittal band reconstruction using EDC tendon slip wrapped around:
  - Lumbrical (McCoy) [24]
  - Collateral ligament [25, 26]
  - Deep transverse metacarpal ligament (Watson) [27]
- Pulley construction MPJ (Kang) [28]

Some of these techniques are described in brief below. We would recommend reading the original articles for the precise techniques.

Surgical Techniques and Rehabilitation

Excision of Scar and Repair or Imbrication

A dorsal approach to the extensor apparatus is made. The site of the sagittal band tear is identified and an appropriate amount of scar tissue is excised to enable centralisation of the EDC tendon. A direct repair of the defect is then performed. Alternatively the scar tissue or attenuated tendon may be imbricated in order to centralise the tendon. The repair should be protected with an appropriate splint in extension postoperatively. The MPJ should be held at 30° for three weeks allowing active extension and performing intrinsic exercises to move the IP joints. At three weeks the MP joints are allowed to flex to 60°. After this the patient is left free to mobilise.

Sagittal Band Reconstruction Using Junctura Tendinum

In this technique the ulna-sided junctura tendinum is released from the adjacent tendon. It is then passed dorsally over the affected EDC tendon and sutured to the radial aspect of the aponeurotic hood thus correcting the ulna subluxation of the EDC tendon following radial sagittal band disruption.

Sagittal Band Reconstruction Using EDC Tendon Slip

A distally based segment of the extensor digitorum communis tendon to the affected digit, consisting of approximately one quarter to one third of the width of the tendon, is developed on the same side as the tear in the sagittal band. It is begun approximately 4 cm proximal to the MP joint and extended distally to the level of the joint. This segment must be of adequate length to permit subsequent weaving. The tendon segment is passed through a small opening created in the remaining extensor tendon at the desired level of the sagittal band. This prevents further unraveling and distal migration of the separation in the extensor tendon. The tendon segment is then looped through the deep transverse metacarpal ligament on the side of the tear. The free end of the tendon segment is then woven into the long extensor tendon and sutured after the tension is adjusted to maintain the extensor tendon in a centralized position. The proximal and distal positions of the band are important. In full MP joint flexion, the centre of the band should lie 8–14 mm proximal to the articular surface of the proximal phalanx, depending on the hand size. If the band is positioned too proximal, joint extension will be limited. If the band is positioned too distal, tendon subluxation will recur. A K-wire is used to immobilise the joint in approximately 15° of flexion for 3 weeks.

This technique may be modified to involve passing the EDC tendon slip around the lumbrical or the collateral ligament. McCoy and Winsky [24] described the "lumbrical loop operation" using a proximally based slip of tendon that was passed around the lumbrical tendon and sutured to itself under proper tension. Kilgore et al. [25] described a technique using a distally based segment of tendon developed along its radial side, passed under the radial collateral ligament, and sutured back upon itself. Carroll et al. [26] modified Kilgore's method by using a slip of tendon developed on the ulna side of the affected tendon prior to passing it around the radial collateral ligament and suturing it to itself.

Pulley Construction MPJ (Kang) [28]

A dorsal skin incision is made over the MP joint. A 3–4 cm length of palmaris longus is harvested. A drill hole is made through the neck of the metacarpal from dorsal midline through the radial cortex at the head neck junction. The graft is threaded through and round the tendon. The new pulley is sutured ensuring the tendon is not subluxating and that the joint has a full range of flexion. The joint is immobilised in extension and allowed to start movement at 3–4 weeks.

Outcomes and Complications

Watson et al. [27] reports excellent results utilising his technique described above. Twenty-one sagittal band reconstructions were performed 16 patients. The series included 18 cases of ulna subluxation secondary to radial sagittal band disruption and three cases of radial subluxation secondary to ulna sagittal band disruption. Pain was eliminated in all cases and there was no recurrence of subluxation. There were no complications reported and the range of motion was also maintained with an average post-operative range from −1 to 90° at the MPJ.

Carroll et al. [26] reported on five ulna subluxations in three patients. Again there were no recurrences and a full range of motion was achieved post-op.

Kang reports this technique used on 6 extensor tendons. The report stated all fingers obtained 80°+ of flexion, full extension, and successful of the tendon subluxation. No complications were reported

Conclusion/Authors Preferred Approach

Our approach to the chronic sagittal band disruption is to try and excise the scarred area and repair the sagittal band directly. In our experience this

works in most cases where there has been a traumatic injury that has failed to heal. In other cases where the dislocation of the central tendon has developed spontaneously or the tissues are inadequate we have always used the EDC reconstruction. This is a straightforward procedure with predictable results.

Chronic Zone 6, 7, and 8 Injuries

Aetiology

Injury to extensor tendons in zones 6, 7 and 8 are most commonly caused by open trauma. These injuries will normally be managed with direct repair. However, if there is a delayed presentation, failure of the initial repair or complex injury resulting in loss of tendon length then tendon reconstruction may be appropriate.

Tendon rupture may also be secondary to inflammatory conditions or may be attritional rupture caused by prominent plates and screws, bone or fracture fragments. In these circumstances the tendon is not normally amenable to primary repair and so tendon reconstruction is necessary.

Assessment

When considering tendon reconstruction in zones 6,7, and 8 it is first of all essential to assess the functional deficit that the patient experiences. It is then important to establish which tendons are injured or missing, which tendons are working and which tendons may be available for transfer.

All of the joints which a tendon acts on should be assessed. A full passive range of movement of all of these joints is as always a pre-requisite to any surgery to reconstruct the tendon that acts on those joints.

The soft tissues through which the tendons run must also be supple and be able to provide an appropriate bed on which the reconstructed tendon can glide. If the soft tissues are inadequate then a staged reconstruction maybe necessary with flap coverage and insertion of tendon rods and second stage tendon grafting.

Treatment Options

Occasionally lacerated tendons in zone 6 remain tethered in position by the juncturae tendinae allowing for delayed primary repair several weeks after the initial injury. If direct repair is not possible one of the following tendon reconstructive procedures may be employed:

- Tendon transfer
  - Side to side of EDC
- Tendon interposition graft
- Two stage tendon reconstruction using a silicone rod
  - May be used in severe cases of tendon loss when the soft tissue envelope is adherent.

The prequisite for all tendon reconstruction need to be met. Full or near full passive movement of the joints is essential. If necessary joint release may be needed before embarking on tendon reconstruction. This should be done as a separate procedure to tendon reconstruction. This is to avoid the conflict in the post operative therapy regimens.

Surgical Techniques and Rehabilitation

Free Tendon Interposition Graft

The technique of interposition grafting is to create a join at both ends that is strong enough to allow immediate mobilisation. There are a number of techniques ranging from a standard tendon weave to a side to side suture. The most important aspect of the procedure is to get the tension correct. This means testing the graft using the tendodesis effect of moving the wrist. With the wrist in neutral the MPJs should be held at 20–30° flexion and with full extension should allow MPJ flexion to 60° and full flexion should achieve 0°. You can test the amount of extrinsic tightness by performing Bunnell's test; with the MPJ in flexion you assess the amount of PIPJ flexion. If you cannot flex the PIPJ to 90° the reconstruction is too tight. As mentioned in the overview it is wrong to overtighten an extensor as this will cause loss of function by holding the MPJ in extension, this is a common error in extensor reconstruction in this area. The patient may express dissatisfaction that

you have not given them full active extension but they will be far more unhappy if you prevent flexion that prevents grip.

Tendon Buddying

This is performed for loss of extensor tendon function in either inflammatory disease or occasionally in traumatic injury where the rupture has taken place at the wrist level. The commonest indication for this technique is loss of EDC & EDM of the little finger where the tendons are sutured side to side to the adjacent ring EDC. Sometimes it is possible to do this for loss of both ring and little finger extensors using the middle finger EDC as the donor. The problem with this technique is balancing the tension and also for the little finger not creating an abduction deformity due to the abnormal pull.

Outcomes and Complications

There is limited literature available which examines the functional outcome following extensor tendon reconstruction in zone 6–8.

With regard to reconstruction following extensive soft tissue loss on the dorsum of the hand there is an option of performing an immediate tendon reconstruction or performing a staged reconstruction using tendon rods initially in order to create a bed for the tendon graft. Sundine and Scheker [29] compared immediate and staged reconstruction in 14 patients (7 in each group). They found that patients undergoing immediate reconstruction had a significantly faster return to maximum range of motion, significantly fewer operations and a greater chance of returning to work. A further report by Koul et al. [30] demonstrates good results following single stage extensor tendon reconstruction in 18 patients who had complex injuries requiring soft tissue cover.

Conclusion/Authors Preferred Approach

In the fingers for a single EDC tendon failure our preferred reconstruction is to use an interposition graft. For loss of the index finger EDC and EIP if interposition grafting is not an option we prefer to use a transfer of EDM. For multiple loss of tendon function the reconstruction will depend on the nature of the loss. Where there has been tendon repair that has failed then again our preferred option is to use primary interposition tendon grafts. Where the loss of tendon is due to extensive loss of soft tissue over the dorsum of the hand and wrist then this may require a two stage method with insertion of silicone rods and provision of soft tissue cover prior to a second stage insertion of tendon graft. If there is loss of all six extensor tendons as may occur rarely in an inflammatory arthritis then it is our preference to use a tendon transfer, using one or two FDS tendons.

EPL Reconstruction

Aetiology

Injury to the EPL tendon may be secondary to an open injury. These injuries should be treated with early exploration and repair. However, if there is a delayed presentation or if there is failure of the initial repair a tendon reconstruction may be necessary.

Due to the anatomical course of EPL around Lister's tubercle it is prone rupture from synovitis which may be secondary to inflammatory conditions such as Rheumatoid Arthritis. Rupture may also be caused by attrition from an underlying fracture or prominent metalwork. It is however also seen following undisplaced distal radius fractures which suggests a possible ischaemic aetiology. Spontaneous idiopathic rupture has also been reported. In these scenarios tendon reconstruction is indicated.

Assessment

It is important to establish the time of rupture especially in open injuries as this will help determine whether or not the patient is suitable for primary repair. The exact aetiology of tendon rupture should be established and underlying

inflammatory conditions should be confirmed or excluded.

The EPL tendon may be examined by asking the patient to place their hand flat on the table and then lift the thumb up off the table. The thumb range of movement should be assessed and if reduced the patient should undergo intensive hand therapy to maximise the passive range of motion prior to any surgical intervention.

USS is the investigation of choice to confirm the rupture. It is also helpful to assess options for reconstruction and if a tendon interposition is to be considered an USS should be performed to confirm the presence of palmaris longus and/or plantaris which are suitable donors.

Treatment Options

The options for surgical reconstruction of the EPL tendon are as follows:

- Delayed direct repair +/− re-routing to gain length
- Tendon transfer
  - EIP to EPL
  - APL to EPL
- Free tendon interposition graft

If a tendon transfer is to be performed it is essential that the thumb joints that are to be powered by the transfer have a full passive range of motion pre-operatively. Details of the different surgical techniques and rehabilitation are discussed below.

Surgical Techniques and Rehabilitation

EIP to EPL Transfer

Incisions

1. Transverse proximal to MPJ index finger
2. Transverse distal edge extensor retinaculum
3. Sinusoidal over ulnar side 1st metacarpal and MPJ thumb

Procedure

Harvest EIP by identification of the tendon on the ulnar side EDC at the MPJ. Identify EIP at the distal edge retinaculum. Check EDC to index functions. Divide EIP at the MPJ and retract tendon to wrist incision. Identify EPL in thumb incision and check satisfactory IPJ extension by traction on the tendon. Transfer EIP from the wrist wound to the thumb wound using a straight tendon passer. Close the finger and wrist wounds. Weave EIP into EPL. Tension is correct when flexion of the wrist results in full IPJ extension and adduction of the thumb and with wrist extension the thumb can be brought across to the line of the middle finger. Use three weaves which is strong enough to allow early mobilization. Hold thumb in extension and radial abduction.

Post Operative Management

Once sutures are removed the thumb should have a splint made to hold extension and radial abduction with removal to allow active mobilisation. This programme should be followed for 6 weeks and thereafter the splint can be discarded.

APL to EPL Transfer

Incision

Single curved incision across radial aspect wrist.

Procedure

EPL is divided from any pseudo tendon and a tenolysis performed to allow a straight pull along the line of APL. A part of APL is divided at its insertion onto the 1st metacarpal and the 1st extensor compartment is released. The APL and EPL are the weaved together. The tension is such that with the wrist in ulnar deviation the thumb is held in full radial abduction and with radial deviation the thumb can reach to the base of the middle finger.

This procedure achieves radial abduction of the thumb with extension of the IPJ. It does not assist with adduction of the thumb in the same way that EPL on its own produces and that the EIP transfer replicates.

The post operative regimen is the same as in the EIP transfer.

Free Tendon Interposition Graft

If one is to consider a free interposition tendon graft it is essential that the musculo-tendinous unit

proximally is still functioning. Timing is therefore important as structural changes within the muscle may take place that prevent satisfactory function after a certain time if the muscle has not been used. MRI scan can be used to asses the degree of structural change if there has been a significant delay.

Palmaris longus is the most common donor used. In the absence of PL, plantaris or a long toe extensor may be used. The tendon graft is attached both proximally and distally to "healthy" EPL tendon once the abnormal section of EPL has been excised and a tenolysis performed. It is essential that the tendon graft is long enough to enable two secure anastamoses either by tendon weave or side-side anastamosis. The type of tendon anastomosis should also be strong enough to enable early active mobilisation. The tendon anastamoses should take place in a supple soft tissue envelope otherwise the tendon reconstruction will become tethered. A straight line of pull should be created and so the reconstructed tendon should pass superficial to the retinaculum.

Tensioning of the graft and the rehabilitation is the same as that described for EIP to EPL transfer.

Outcomes and Complications

Magnussen et al. [31] comment that EIP tendon transfer for rupture of the EPL tendon is a simple a reliable procedure with few complications. In their small series of 21 patients 19 were described as having a good result and 2 as fair at a mean follow up of 5.3 years. However, the strength of extension compared to the uninjured side was only 51 %. Independent extension of the index finger was achieved in all patients but the strength of the extension was reduced by 49 % of that of the normal finger.

Due to the potential risk of extensor lag or residual weakness of extension of the index finger following EIP transfer some surgeons prefer an interposition graft which utilises the intact EPL neuro-muscular unit. The surgery does however require two anastomoses which are potential sites of failure.

Schaller et al. [32] performed a retrospective analysis of 28 EIP transfers and 17 interposition grafts and found no difference between the two groups with regard range of thumb movement. Pillukat et al. [33] compared the outcome of 48 palmaris longus interposition grafts with 40 EIP transfers. Both groups were comparable with regard demographics and aetiology of rupture. The outcome was good for both groups and there was no difference in the thumb range of motion but it was noted that both methods only restored 60 % of the normal retropulsion. All had independent index finger extension but more powerful extension of index finger was noted in the free tendon grafting group. The authors therefore conclude that the use of an interposition graft should be the preferred option for patients with special demands such as musicians.

The definitive position of the reconstructed EPL tendon is also a topic of debate. The EPL pulley may be reconstructed in order to re-create the normal line of pull of the EPL tendon. Alternatively the EPL tendon may be left subcutaneously out of the pulley. In a cadaveric biomechanical study, transposition of a normal EPL or a reconstructed EPL out of the pulley significantly reduces the adduction moment arm at the thumb CMC joint; an effect which is exacerbated by wrist flexion [34]. The clinical significance of this finding is unknown.

Rehabilitation options post-operatively include static immobilisation (3 weeks), early dynamic extension splinting and free active mobilisation. Studies comparing these three different regimens found that early motion resulted in a better range of motion at 3 weeks without a higher complication rate [35, 36]. At 8 weeks there was however no difference between the groups with regard range of motion and grip strength. It should however be noted that the size of the study groups were small.

Our preference is for early controlled active motion with the patient being rested in a static splint between exercises.

Conclusions/Author's Preferred Approach

The most important message about EPL reconstruction is to make sure the patient wants and needs it doing. Many patients in our experience

manage to adapt to life without a functioning EPL. Our recommendation to the patient who has a rupture of the EPL is that it is not necessary to have immediate surgery. Particularly for the older patient who has a rupture following a distal radius fracture or as the consequence of Rheumatoid disease. In the younger patient where the rupture is the consequence of a failed primary repair we prefer to use an interposition tendon graft. In the patient where the rupture is the consequence of a fracture or inflammatory arthritis we prefer to use EIP. In our view this gives good extension of the IP joint, retroversion and has the bonus of adding adductor power.

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Flexor Tendon Reconstruction

4

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Keywords
Flexor tendon • Reconstruction • One-stage • Two-stage • Tendon graft • Pulley reconstruction • Silicone rod • Adhesions • Tenolysis

Introduction

Primary or delayed primary flexor tendon repair followed by early guided motion rehabilitation protocols are considered the gold standard treatment of flexor tendon injuries. Ideally, prompt end-to-end repair follows early diagnosis of an acute flexor tendon laceration. In general, primary repair can be attempted up to 3–6 weeks after zone I–V injuries in uncomplicated cases.

However, not all patients with flexor tendon injuries are eligible for primary repair. In these cases a one- or two-stage tendon reconstruction should be considered. A delay longer than 3–6 weeks often makes primary repair impossible due to retraction of the proximal tendon stump and scarring in the flexor tendon sheath. This is also often the case if a previous primary tendon repair fails. Other indications for reconstructive alternatives to primary repair are in patients with significant associated soft tissue (crush) injury, wound infection, segmental tendon loss or destruction of the flexor tendon sheath.

In general, the term flexor tendon reconstruction includes (1) tenolysis of flexor tendon adhesions, (2) one-stage tendon grafting or (3) two-stage tendon repair. Indications and reconstructive options vary both per injured flexor tendon and per zone and are directed by the associated problems of the involved digit.

For example, limited range of motion in a previously injured, and repaired, digit may be caused by simple adhesion formation around an otherwise intact or repaired tendon. Surgical exploration may reveal an adequate pulley system, but flexor tendon adhesions and a PIP joint flexion contracture. This particular case may be treated adequately by tenolysis, release of the PIP joint and vigorous post-operative hand therapy. On the other hand, if extensive damage to the pulley system and segmental tendon scarring or undiagnosed tendon rupture post-operatively are

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encountered, a more elaborate two-stage reconstructive strategy including pulley reconstruction is warranted.

In the present chapter, flexor tendon tenolysis and one- and two-stage tendon reconstruction for zone II to V flexor tendon injuries of the fingers and thumb will be discussed.

Tenolysis

Background

Any injury to the tendon initiates the classic healing response of inflammation, proliferation, collagen synthesis and apoptosis. The cellular response to injury occurs within the tendon itself and its surrounding synovial tissue. The tendons become surrounded by a fibrin rich inflammatory exudate which may later convert to scar tissue [1–3]. Adhesions primarily form at the original site of injury and repair, but often spread to involve a much larger segment of the tendon.

Surgical manipulation also plays an important role in added tissue damage and adhesion formation. For this reason Bunnell reported the importance of minimal, atraumatic tissue handling, a bloodless field, strict asepsis and preservation of pulleys [4, 5]. He also addressed the importance of postoperative hand therapy. Indeed immobilization after injury allows for collagen depositions to form between the tendon and the synovial tissue, shaping the adhesions [1]. Therefore early mobilization programs are essential in the prevention of adhesion formation.

Presentation and Investigation

Patients with flexor tendon adhesions typically present with complaints of limited range of motion (ROM) of the involved digit. The digit has been subject to previous damage to the flexor tendon or its sheath caused by trauma, infection or previous surgery [6].

On examination, active excursion (active ROM) of the flexor tendon is incomplete despite a palpable, powerful muscle belly contraction and the passive ROM of the digit is significantly greater than active ROM [6–8]. Passive flexion is greater because the examiner can exert more power on the joint than the muscle-tendon unit which is being limited by the tendon adhesions. Typically, extension of the involved digit is also limited, but the passive and active extension deficits are often equal. The active and passive deficits are both caused by the adhesions on the flexor tendon blocking further extension [8]. Adhesions in the forearm can usually be easily identified by puckering of the skin on contraction of the muscle. In the hand, locating the exact site of adhesions can be more difficult. Careful adjustment of the surrounding joints can help to determine the location of the adhesion. Also, tenodesis of the wrist and/or metacarpophalangeal joints will also help to exclude intrinsic joint contractures as a cause of diminished ROM.

Thorough history and physical examination are often sufficient to adequately diagnose the presence of adhesions, but some surgeons value the help of additional imaging techniques such as ultrasonography or MRI. Ultrasonography can detect the presence of an intact tendon in its sheath, which helps to distinguish between adhesions and a tendon rupture [9]. MRI has been demonstrated to be 100 % accurate in distinguishing between a rupture or adhesions at the site of primary flexor tendon repair [10].

Treatment

Flexor tendon tenolysis should not be thought of lightly. Strickland once described it as the most demanding of all flexor tendon procedures [11]. Therefore, careful patient selection is essential. Criteria that must be met before an attempt at successful flexor tendon tenolysis can be done are listed in “Clinical pearls: prerequisites for successful flexor tendon tenolysis” below [6, 7]:

Clinical Pearls: Prerequisites for Successful Flexor Tendon Tenolysis [6, 7]

All fractures should be healed in anatomic alignment
Wounds must have healed with soft, stable scars and skin cover
Joint contractures have been mobilized to near normal passive ROM
Tendons systems should be intact Muscle strength should be good
Muscle strength should be good
The patient is compliant and motivated
An experienced hand therapist should be available

Preceding tenolysis, at least 3 months of intensive hand therapy is recommended to obtain as much passive ROM as possible. As long as hand therapy improves ROM, tenolysis should be postponed. If no progress has been seen during the previous 4–8 weeks, tenolysis can be planned [11]. Tenolysis earlier than 3 months after primary flexor tendon repair or tendon grafting is considered potentially dangerous to tendon blood supply. This could put the tendon at risk for rupture during postoperative hand therapy exercises [12].

Any additional procedure that requires postoperative immobilization such as tendon grafting, free skin grafts or corrective osteotomies should not be planned concomitantly. In these cases reconstruction in multiple stages is advised.

Ideally, flexor tendon tenolysis is performed under local anesthesia with intravenous analgesia and a sedative [13]. Active involvement of the patient is helpful to fully judge the active ROM of the digit when the tendons have been freed of adhesions. Additional tenolysis at a more proximal or distal level may be required if ROM is still found to be limited. Another advantage of local anesthesia is that the patient can directly observe the progress made during the procedure. This helps the patient's motivation to preserve the results during the postoperative hand therapy program [6]. If the procedure will take longer than 1 h or the patient does not tolerate the local anesthesia with sedation, general anesthesia or axillary block should be used.

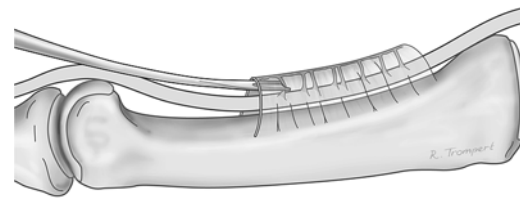

Fig. 4.1 A tenolysis knife is used to disrupt adhesions under the pulleys that are otherwise difficult to reach

Azari et al. described a step by step technique of flexor tendon tenolysis [6]. First, wide exposure of the entire flexor tendon sheath is obtained by Bruner type zigzag incisions or a mid-lateral approach. The flexor tendon is exposed proximally and distally to reach unaffected tissue. Then, both the flexor tendons are freed en-bloc from their surroundings proceeding from an unaffected area through the affected area. The pulley system, especially the A2 and A4 pulleys, is carefully spared as far as possible. Access to the tendons can be obtained through minimal transverse windows in the cruciate pulleys or, if need be, by sacrificing the A3 pulley [14]. Specially designed tenolysis knives are available to get to adhesions under the pulleys that are otherwise difficult to reach (Fig. 4.1).

When the flexor tendons have been freed from the tendon sheath, adhesions between the FDP and FDS tendons should be addressed by separating the tendons from one another [6]. If the tendons appear very frail or heavily scarred, the tenolysis procedure is stopped and one- or two-stage tendon reconstruction should be considered.

All adhesions are dealt with until the tendons glide adequately. At this stage, the patient can be asked to actively flex and extend the involved digits to assess the gliding. If the patient has general or axillary block anesthesia, gliding of the freed tendons can be assessed by the "traction flexor check", as proposed by Whitaker et al. [15]. In this maneuver, proximal traction on the involved tendons through a palmar or volar distal forearm

Fig. 4.2 The traction flexor check. To assess the presence of remaining adhesions, proximal traction on the involved tendon through a volar distal forearm incision is used to passively flex the digit

incision is used to flex the digit passively (Fig. 4.2). Additional tenolysis may be required if tendon gliding is still unsatisfactory.

Several techniques have been tried to prevent adhesion formation after tendon surgery. Local deposition of steroids at the end of the procedure has been suggested, but has also been hypothesized to have adverse effects on tendon healing [6]. Various interposition materials have been tried experimentally and clinically from as early as the 1940's. Materials included silicone sheets, gelatin sponge, and more recently Seprafilm, hyaluronan gel or hydrogel containing biocompatible phospholipid polymer [16–18]. Most however, prefer the use of early active mobilization programs to prevent tendon adhesions [19]. As early as the same day of tenolysis, hand therapy can be initiated. The hand therapist should be advised on the peri-operative findings so therapy can be adjusted to the individual patient's needs.

Outcome

In most, carefully selected patients, improvement of ROM after flexor tendon tenolysis is to be expected. Complete, unrestricted ROM however, is infrequently obtained. In 1989 Jupiter et al. reported an increase of ROM from 72 to 130 ° after tenolysis in replanted fingers [20]. In a series of 72 patients with flexor tendon tenolysis, Foucher et al. obtained an improvement in active ROM from 135° to 203° in 84 % of the fingers and from 65° to 115° in 78 % of the thumbs [21]. Tenolysis also improved ROM with 107 ° in a series of patients with flexor tendon adhesions after a phalangeal fracture [22]. More modest results were obtained in 19 patients with zone II flexor tendon adhesions, reported by Riccio et al. In this group tenolysis improved ROM by 28 % [18].

Complications

Tenolysis does not always improve digital function. In one series the large majority of patients had benefit of tenolysis, but no change or reduced ROM was observed in 16 % of the fingers [21]. So insufficient effect, or deterioration due to surgery are complications that need to be considered. The main cause for these complications is recurrence of adhesions again emphasising the importance of early postoperative active mobilization programs.

Tendon rupture is another known complication of tenolysis. In a series of 23 patients, flexor tendon rupture occurred after tenolysis in 16 % of the cases [23].

Other, less specific complications after tenolysis include would healing problems, cold intolerance or neurovascular injury. These complications are mainly due to the repeated surgical insult to the already compromised digit. Careful patient selection to meet the above mentioned selection criteria can avoid these problems.

One- and Two-Stage Tendon Reconstruction

Background

Potential candidates for flexor tendon reconstruction can present early or late. A patient seen immediately after injury has a completely different subset of problems to be addressed than a patient presenting weeks or months after the initial injury or after undergoing earlier surgical attempts at tendon repair.

In acute cases, crush or blast injury is often the cause of a mangled digit or hand. Injuries are often extensive and involve multiple digits, levels or soft tissue structures. This often makes primary tendon repair impossible. In these cases, problems such as inadequate soft tissue cover, infections or fractures need to be dealt before the flexor tendons can be reconstructed.

Patients that present late usually had less extensive initial injuries. Their causes of functional problems include tendon bed scarring, adhesions, joint stiffness or trophic changes due to associated nerve injury. Alternatively, impairment of flexor tendon function recurred after earlier attempts at primary repair. In these secondary cases, tendon adhesions, failure of the tendon repair or both require attention.

The classification system published by Boyes in 1950 is a useful tool in surgical planning, Table 4.1 [24]. In the most favorable cases, a single involved digit is in otherwise optimal condition (Grade 1). Presence of scar tissue renders the case more complicated (Grade 2). Stiffness of the interphalangeal joints requires additional hand therapy or capsulectomy (Grade 3). Associated digital nerve damage causes trophic changes making successful functional outcome less likely (Grade 4). Finally, "multiple damage" can be interpreted in two ways: multiple injured fingers or multiple lesions (*e.g.* bone, skin and neurovascular injury) in a single digit (Grade 5) [25]. In general, primary repair can be attempted in grade 1 injuries without significant delay. Tendon reconstruction in one or two stages should be considered in grades 2 to 5 [26].

Table 4.1 Boyes classification [24]

Grade	Condition
1	Minimal scar, mobile joints, optimal condition
2	Scarring
3	Joint damage/stiffness
4	Digital nerve damage
5	Multiple digits/lesions per digit

Tubiana has refined the indications for tendon reconstruction of injured digits since Boyes' publication [25]. In addition to assessment of associated injuries, timing of presentation (early, intermediate or late) is also considered in the decision making process. According to Tubiana, the indication for one- or two-stage tendon reconstruction arises if (1) the case presents late and significant scarring is present, (2) an addressed associated fracture remains unstable, (3) there is inadequate skin cover or 4) in case of multiple injuries (*i.e.* nerve, bone, joint or skin). In the latter, outcome of tendon reconstruction is poor and salvage procedures such as arthrodesis or amputation should be considered.

In short, flexor tendon reconstruction should only be considered if issues of soft tissue cover, joint stiffness, bone injuries and neurovascular damage can be or have been adequately addressed. It is essential that functional, passive ROM is present or restored before tendon reconstruction procedures are initiated. Pulvertaft summarized these conditions for successful tendon grafting (See "Clinical pearls: Pulvertaft's conditions for successful tendon grafting" below) [27].

Clinical Pearls: Pulvertaft's Conditions for Successful Tendon Grafting

The involved hand is in overall good condition
There is no extensive scarring of the tendon bed
Passive ROM is (nearly) full
Circulation of the digit is satisfactory
At least one digital nerve is intact
The patient is cooperative

The state of the involved digit or hand cannot always be judged completely by history and physical examination alone. In the acute situation conventional radiography is often required, but there is no place for other diagnostic imaging modalities [12]. Imaging techniques such as ultrasound, CT or MRI can be helpful in secondary cases. It can be difficult to distinguish between a tendon rupture or adhesion formation after earlier flexor tendon repair. In these cases ultrasonography is a useful, non-invasive imaging technique [12]. CT is capable of detecting pulley ruptures. MRI is expensive, but superior in diagnosing flexor tendon problems such as adhesions, partial or complete tendon ruptures or pulley damage.

Surgical exploration however, remains the only method to fully assess the amount of scarring, presence and location of tendon adhesions, the state of the tendon sheath and the tendon stump. Based on physical examination, imaging and operative findings a definitive reconstructive plan can be made.

In all cases a clear understanding of the patient's wishes, expectations and – very importantly – motivation, are of paramount importance when deciding if the patient is a good candidate for flexor tendon reconstruction. Thorough preoperative counseling is needed to discuss the options, the potential results, risks and complications of all reconstructive efforts. A multidisciplinary hand clinic is ideal to council patients seeking flexor tendon reconstruction. The involved hand surgeon and therapist can consult the patient together to guide decision making by all parties involved. Decisions should not be made hastily. If the patient has reservations or second thoughts, a follow-up appointment should be arranged for more counseling.

Flexor Tendon Reconstruction of Zone I Injuries

Presentation

In flexor tendon zone I, the FDP tendon is damaged distal to the insertion of the intact FDS tendon by avulsion from its insertion, laceration or failure of a previously performed primary FDP repair.

In 1977 Leddy and Packer categorized closed avulsion injury of the FDP insertion from the distal phalanx into three types [28]. A few years later, a fourth type was added [29, 30] (Fig. 4.3).

Type I injury involves rupture of the FDP tendon from its insertion on the distal phalanx. Vinculae are also ruptured, allowing the tendon to retract into the palm. Rupture of the vinculae causes a hematoma in the flexor tendon sheath. If not treated urgently, fibrosis in the flexor tendon sheath and fixed muscular contraction make primary repair impossible.

Type II FDP avulsions are more forgiving. The tendon ruptures from its insertion, but the intact vinculae only permit limited retraction of the tendon to the level of the PIP joint. Hematoma formation is less pronounced and limited retraction is unlikely to cause fixed contraction of the muscle. Type II injuries are therefore often eligible for delayed primary repair with good results. Successful primary repair of type II injuries after 3 months delay have been described [31].

Type III lesions are much like type II injuries to the extent that retraction is limited and the vinculae remain intact. In these lesions, a bony avulsion fragment of the distal phalanx prevents tendon retraction into the flexor tendon sheath. Type III lesions are therefore also more often suitable for delayed primary repair.

In 1981 Smith suggested adding a type IV FDP avulsion injury. In this fairly rare type of injury not only does the FDP tendon retracts into the finger or palm bit it also ruptures from an

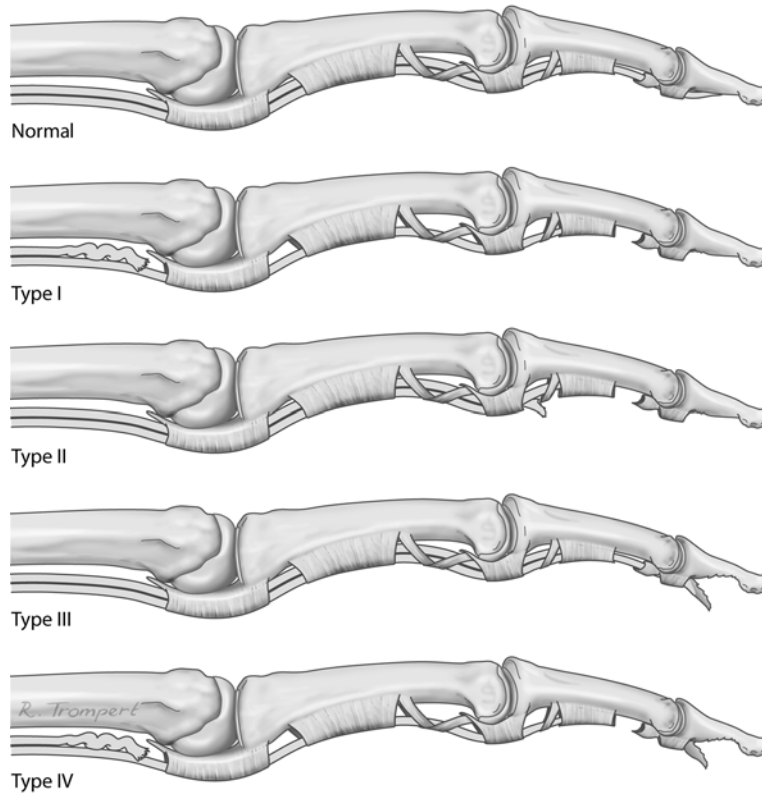

Fig. 4.3 Types I to IV avulsion injuries of the FDP insertion

avulsed bony fragment [30]. Other than the presence of a bony avulsion fragment, this is much like a type I injury and should be addressed in an urgent fashion.

In summary, flexor tendon grafting or two stage flexor tendon repair is usually indicated in type I and IV injuries. However, significant delay or associated injuries may require more this type of treatment in types II and III injuries.

Open lacerations of the FDP tendon in zone I have similarities to type II closed avulsion injuries; the FDP tendon usually remains tethered to its vinculae and retracts no further than the PIP joint or proximal phalanx.

Unfortunately, rupture of a previously repaired zone I primary tendon injury is not uncommon. If recognized within two to three days, ruptured primary tendon repairs can be successfully treated with a repeated attempt at primary repair. If delayed longer, one- or two-stage reconstruction should be considered.

Treatment

The indication for reconstruction of FDP function with an intact FDS remains controversial. The reasons for this debate are twofold.

Firstly, impairment caused by loss of FDP function is limited to inability to actively flex the DIP joint and reduced strength in the involved digit. It must be noted that particularly in the ulnar two fingers, loss of power grip can be quite restrictive. Also, active flexion of the DIP joint may be needed in particular cases (*e.g.* musicians). But if the DIP joint does not hyperextend during pinch and the patient does not have particular need for active DIP joint flexion, conservative treatment is a viable option. Alternatively, tenodesis or arthrodesis to stabilize the DIP joint are functionally valuable options.

Secondly, good outcome of FDP reconstruction with an intact FDS cannot be guaranteed. Some authors go as far as limiting reconstruction

to patients of 10–21 years of age [32]. If the wish for reconstruction is outspoken, the patient should be clearly informed that results can be disappointing or, in some cases, may even be functionally worse.

Clinical Pearls

Isolated loss of FDP is often functionally unimportant

More conservative options: – no surgical treatment, tenodesis procedure or arthrodesis are usually more appropriate

Consider one-stage reconstruction in younger and well motivated patients

One Stage Tendon Grafting

One stage tendon grafting can only be performed if the flexor tendon sheath is intact, there is minimal scarring and joints are supple. The tendon graft chosen should be thin enough to fit in the flexor tendon sheath together with the intact FDS tendon slips. In the majority of cases, fingertip-to-palm grafts suffice for zone I FDP tendon reconstructions with an intact FDS. The palmaris longus tendon or the extensor digitorum communis tendon to the index finger have been reported to be suitable grafts [11]. These tendons have sufficient length for tip to palm grafting. Characteristics and harvesting technique of available grafts for one- or two-stage reconstruction are described later in this chapter.

Bruner type zig-zag incisions or a mid-lateral approach to the flexor tendon system is obtained. Choice of approach depends on preference of the surgeon and on pre-existing scars. The zig-zag incisions provide the best exposure, but the mid-lateral approach reduces the amount of scarring directly over the flexor tendon sheath [33]. Exposure of the flexor tendon sheath is obtained from the FDP insertion at the distal phalanx to the mid palm. Remnants of the FDP tendon should be excised from the fingertip to the lumbrical origins. If possible a 1 cm stump of the distal FDP at its insertion on the distal phalanx should be preserved for attachment of the graft. The annular pulleys should be spared as much as possible.

Ideally, the graft is threaded carefully through the chiasma of the FDS tendon. But the chiasma is often obliterated by scarring. If so, the graft can be routed around the FDS tendon slips. Under no circumstance should the functional FDS insertions be sacrificed. Some authors however, have suggested a resection of one of the FDS tendon slips [34]. This is usually considered unnecessary [35].

The distal junction of the graft to the distal FDP stump or distal phalanx should be fixed first. Multiple techniques for the fixation of the distal juncture have been described. If the distal FDP tendon stump is of sufficient length, a graft-to-tendon suture technique is used. Otherwise, graft-to-bone fixation is warranted [34]. The same techniques apply for fixation of the distal graft junction as described previously for primary flexor tendon repair. Options include pull-out sutures through or around the distal phalanx that place the tendon end into small transverse trough in the volar distal phalanx, or alternatively the use of a small bone anchor. The decision on which fixation to use is largely based on surgical preference. Pull out sutures have the advantage of being non-permanent. They also allow for accurate placement of the distal tendon end into the bony trough. If the sutures are placed through the nail plate, deformities of the nail may result. The bone anchor has de advantage of a strong fixation without the need of suture removal or the risk of nail plate deformities. The downside is the use of a permanent implant, which some surgeons prefer to prevent if possible.

Skin can be closed distally before the proximal juncture in the palm or distal forearm is made. This facilitates skin closure before tensioning at the proximal juncture places the digit into a flexed position.

Typically for zone I injuries, the FDP motor is still intact and its proximal tendon is available in the palm. Proximal fixation of a slender graft such as the plantaris or palmaris tendon can be done by interlacing the graft through the FDP

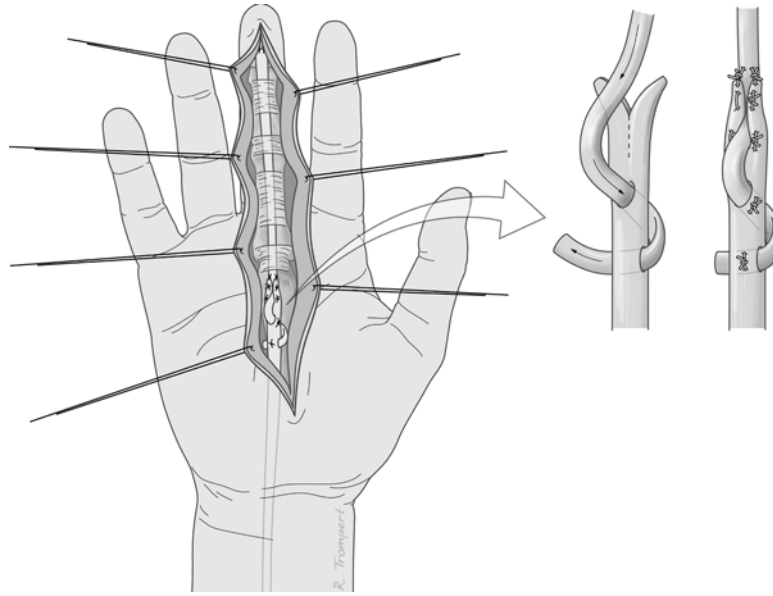

Fig. 4.4 The tendon graft is tunneled trough the flexor tendon sheath and attached proximally. If sufficient space is available, the attachment is made by interlacing the graft through the FDP tendon as described by Pulvertaft

tendon as described by Pulvertaft [36]. Care should be taken to place this fairly bulky connection sufficiently proximal to the A1 pulley to allow for unimpeded tendon gliding, if needed the A1 pulley can be vented to create additional gliding room (Fig. 4.4). Alternatively, an end-to-end juncture can be fashioned if the graft and FDP tendon are of similar caliber. If the proximal juncture is placed in the distal forearm, the Pulvertaft weave is recommended. With the wrist, MCP joint and PIP joint straight, tensioning of the graft should put the DIP joint in approximately 40° of flexion [35].

Strickland recommends postoperative immobilization of the hand with a dorsal splint for 3.5 weeks [11]. More recently, others allowed for gentle short-arc active extension and flexion within the first postoperative week, supervised by an experienced hand therapist [19].

Two Stage Tendon Grafting

A two-stage tendon grafting procedure is needed if scarring of the flexor tendon sheath does not allow for supple gliding of the future graft. Also, if the A4 pulley needs to be reconstructed or a stiff DIP joint needs release, this can be done at the first procedure together with placement of a silicone rod.

Stage One

Exposure of the entire flexor tendon sheath is obtained from the insertion of the FDP tendon on the fingertip to the site of the planned proximal juncture in the palm or the distal forearm. Previous incisions must be respected to ensure viability of the skin flaps. The state of the sheath and FDS tendon are evaluated. Any scarring of the sheath or pulleys is excised. Joint contractures are released at this stage if needed.

The A4 pulley is reconstructed if it is considered dysfunctional and irreparable. The remainder of the FDP tendon is excised to the origin of the lumbricals in the palm. If possible, the distal 1 cm of the FDP at its insertion on the distal phalanx should be spared for attachment of the silicone rod and the tendon graft in the first and second stages respectively. Also, the (healthy) proximal end of the FDP can be sutured to the A1 pulley in the first stage. This maintains length and prevents retraction.

Depending on the planned future tendon graft and the available space, a properly sized silicone implant is threaded through the (reconstructed) pulley system and fixed to the distal FDP stump. If the distal FDP stump is not available through- or around-the-bone suture techniques or screw fixation of the silicone implant can be used [37].

If the plantaris tendon is to be used at the second stage, a 3 mm silicone rod will usually suffice [12]. A future palmaris tendon graft requires a larger size implant: 4–5 mm, but space is usually limited due to the proximally intact FDS tendon. The implant can be placed trough or around the chiasma of the FDS and threaded proximally enough into the palm to allow for unimpeded gliding of the proximal end. If the palm is scarred or the lumbricals are damaged, these should be bypassed by a longer silicone rod to the distal forearm. In the distal forearm the proximal end is placed between the FDS and FDP tendons. The proximal end of the silicone rod is left unattached. Free gliding of the rod must be tested before closure of the wounds. The proximal tendon end in the forearm can be marked with an non-resorbable monofilament suture to make subsequent identification easier during the second stage. The hand is covered in a bulky compressive dressing with the wrist in slight flexion.

After stage one, post-operative hand therapy is aimed to keep the joints supple and the tendon sheath open. Passive guided motion exercises are started at 7–10 days. After sufficient time for the soft tissues to heal and the pseudosheath to form, the second procedure is scheduled. This is usually 3 months after the first procedure.

Stage Two

In the second procedure, only minimal exposure of the silicone rod and the newly formed pseudosheath is necessary at the fingertip and at the site of the proximal juncture. Distally, the silicone rod is released from its insertion. The tendon graft can then be attached securely to the silicone rod and very gently pulled proximally trough the pseudosheath into the proximal wound. Characteristics and harvesting technique of suitable grafts are described later in this chapter. With the tendon graft in place, the rod is detached and discarded. The distal juncture of the graft is secured as described above before closure of the distal wound.

Attachment to the original FDP motor is preferred. The combined FDP motor of the third to fifth digit is usually available and in good shape if only one digit had been injured initially. The individual FDP motor of the second digit is often contracted if the palmar tendon and lumbricals have been excised at the first stage. Contracture can be prevented by attaching the musculotendinous juncture of the FDP motor of the second digit to the periosteum of the radius under tension at the first stage so the motor can be used in the future The alternative of attachment of a tendon graft of the second digit to the combined motor of the third to fifth FDP is preferred by most. Alternatively, the FDS can also be chosen as a motor to the tendon graft if multiple digits need to be grafted, or if the FDP motors are of insufficient quality.

Enough tension is placed on the graft at the proximal juncture to place the DIP joint in 40° of flexion with the PIP and MCP joints in extension and the wrist held straight. Tenodesis testing at wrist level ensures proper tensioning and cascade of the digits. Depending on the caliber of the graft and the motor tendon a Pulvertaft weave or end-to-end tenorrhaphy is used for the proximal juncture. At the end of the procedure, the hand is put in a bulky compressive dressing with the wrist in slight flexion, the MCP joints in approximately 70 ° of flexion and the interphalangeal joints in extended or slightly flexed position [35].

Early mobilization under supervision of an experienced hand therapist can be initiated in the first postoperative week. If the graft and junctures are not considered to be strong enough, immobilization for 3–4 weeks can be chosen [11]. This does increase the risk of adhesion formation [19].

Outcome

In 1988 Ipsen et al. published a series of one-stage tendon grafts through or around an intact FDS tendon [38]. They concluded that early mobilization is safe with only one tendon rupture in 25 cases. All but one had increased total ROM at long term follow up. On the other hand, six fingers lost an average of 16° of PIP joint flexion.

Older series of one-stage tendon grafting for isolated FDP lesions also reported favorable results [39, 40]. In 1969 Goldner et al. demonstrated good

functional outcome but stressed that detailed preoperative assessment, meticulous surgical technique, careful selection of patients and surgical experience are necessary.

Wilsen et al. reported a series of delayed, two-stage tendon grafting in a series of twelve fingers with flexor profundus avulsions or lacerations. Total active motion improved 78°. Grip strength significantly improved in 8 of the 11 patients. One graft rupture occurred and in two cases secondary tenolyses were necessary [41]. Sullivan reported disappointing results of staged flexor tendon grafting for isolated FDP injuries. Only 7 of the 16 cases achieved satisfactory results [42].

Complications

Complications associated with repeated surgical interventions to the digits include skin flap necrosis, wound healing difficulties, scar contractures, cold intolerance and neurovascular damage. Careful selection of cases with the Boyes classification in mind reduces occurrence of these problems.

More specifically, damage to the intact FDS tendon or added scarring due to repeated surgeries need to be considered. Potentially, the patient could have no benefit or reduced function due to failed reconstructive efforts [42].

Suboptimal tensioning of the graft causes problems. If the FDP graft from the fingertip to the palm is kept too long, a lumbrical plus digit may result. Contracture of the FDP motor causes more tension on the lumbricals than the distal graft, causing paradoxical extension of the interphalangeal joints. Conversely, if a graft is tensioned too tightly a quadriga effect may occur, especially in the third to fifth digits. In this case, further (common) FDP muscle belly contraction is limited by the fully flexed reconstructed digit, leaving the muscle incapable to further flex the other fingers with their less tight/longer tendons.

Complications of two-stage grafting include infection, synovitis around the implant or rupture of the distal juncture between stages one and two. Other reported complications include median nerve neuralgia and carpal tunnel syndrome [42].

In all cases, adhesion formation is the most important reason for disappointing results and the need for additional interventions [43, 44]. If, in the months following reconstruction, active flexion diminishes in the presence of passive flexion, tenolysis should be considered. Reoperation however, should be delayed. If tenolysis is performed within 5 months of tendon reconstruction there is an increased risk of tendon rupture [35].

Flexor Tendon Reconstruction of Zone II Injuries

Presentation

Flexor tendon zone II contains both flexor tendons to the digits in the confined space of the flexor tendon sheath. Injuries in this zone are notorious for their difficulty to manage and poor functional outcome. It is for these reasons that this zone is also referred to as “no man’s land”. Cases become particularly difficult to manage if the opportunity for primary repair of the tendons has passed. In those cases reconstructive procedures such as one-stage free tendon grafting or two-stage reconstructions may be indicated.

Potential candidates for zone II tendon reconstruction often present with one of the following backgrounds:

- Delayed treatment of combined FDP and FDS lacerations
- Significant associated injuries to the soft tissues (*e.g.* crush injury)
- Tendon rupture or adhesion formation after earlier (repetitive) attempts at primary repair
- An injury that included segmental tendon loss

Treatment

The objective of one-stage or two stage tendon grafting in zone II is to excise remnants of the flexor tendons and to reconstruct the FDP. Two exceptions should be noted:

Firstly, if only the FDS tendon is severed and the FDP is intact no attempts at tendon reconstruction should be made [35]. In these cases the

remnant of the FDS is excised out from the flexor tendon sheath.

Secondly, in some cases one can choose to reconstruct the FDS and excise the FDP. For example, if the DIP joint is stiff, the distal phalanx is (partially) amputated or if the only available graft is too short to reach the distal phalanx [35]. Other indications for construction of a "superficialis finger" are tendon reconstructions in multiple digits or the need to reconstruct more than two pulleys [45]. A superficialis finger will avoid DIP problems, usually shows good active PIP flexion and minimal functional impairment [12, 46].

One-Stage Tendon Grafting

The most common indication for one-stage tendon grafting in zone II is delayed presentation. It should only be considered if wounds have healed, the finger has full passive ROM, the flexor tendon bed is not significantly scarred and the annular pulleys are intact. In general, only Boyes grade 1 candidates would be eligible for a one-stage reconstruction (Table 4.1). Otherwise, two-stage reconstruction is indicated.

Exposure of the entire flexor tendon sheath is needed. A volar zigzag incision or mid-lateral approach can be chosen based on the presence of previous incisions or traumatic scars and the preference of the surgeon. A thorough inspection should confirm the presence of a flexor tendon sheath that allows supple gliding and intact annular pulleys. Particularly the A2 and A4 pulleys are essential to prevent functional impairment due to bow-stringing. But the presence of more pulleys, especially the A3 pulley, is favorable [35].

Remnants of the injured FDP and FDS tendons are excised, keeping a 1 cm stump of the FDP at its insertion on the distal phalanx if possible. The FDP remnant is pulled proximally out of the flexor tendon sheath and transected in the mid palm so that only good quality tendon remains. If the lumbricals are scarred or adherent to their surroundings, they should also be excised.

The FDS remnant should be shortened sufficiently to prevent adhesion to the future proximal juncture of tendon graft juncture to the FDP. Tubiana suggested withdrawing the FDS proximally into an additional volar incision in the distal forearm and transecting it there [35]. Strickland advised putting distal traction on the FDS stump, through the palmar incision, and cutting the tendon as proximal as possible [11].

Passive ROM of all joints of the digit can be tested once more and the quality of the flexor tendon sheath and pulley system reassessed. If these are satisfactory, one can proceed with the one-stage tendon grafting. If pulleys require reconstruction or if joints need to be released, it is advisable to convert this to a two-stage reconstruction.

For fingertip-to-palm grafting the palmaris longus tendon is the preferred donor. It can be carefully threaded through the flexor tendon sheath using a tendon passer or paediatric nasogastric tube. The distal juncture to the FDP tendon stump is attached first outside the sheath to prevent added damage by surgical manipulation. Then, the distal portion of the wound can be closed and the proximal juncture to the proximal FDP tendon addressed. A Pulvertaft tendon weave juncture is the strongest option. But if this juncture is too bulky in the palm, and the calibers of the graft and the proximal FDP allow it, an end-to-end tenorrhaphy is chosen. If the proximal juncture is made in the distal forearm, there is usually enough space for the Pulvertaft weave.

The excursion of the motor tendon is tested using a traction suture. Excursion should be sufficient to allow for adequate digit flexion. With a fixed wrist, about 2.5 cm of excursion is necessary to fully flex a digit. However, more available excursion is preferred (*e.g.* >4 cm). It is then immobilized halfway along its course of excursion by a needle or temporary suture to the skin. A single pass of the tendon weave with one suture allows for testing of the digit's position. With the wrist in neutral position, tension should put the involved digit in slightly more flexion than the natural arcade of the digits. Tubiana suggested 15° of additional flexion compared to normal [35]. Then the fixating needle or skin suture is removed. With the wrist put in 40° of flexion it should be possible to fully extend the grafted digit. Full wrist extension should bring the finger's pulp-to-palm distance within 3–4 cm

[35]. If tensioning is satisfactory, the tendon weave is completed and the skin closed.

If the surgeon is confident enough that both junctures can withstand the strain, early passive motion programs such as those of primary flexor tendon repair can be initiated. Tubiana recommended 10 days of immobilization in the relaxed position followed by passive mobilization of the digit with the wrist and MCP joints immobilized by a dorsal splint. However, others prefer to immobilize the digit for 3–4 weeks before movement programs are started [11]. Immobilization should keep the wrist midway between neutral and full flexion, the MCP joints in 60–70° of flexion and the interphalangeal joints extended. Full extension should be prevented for several weeks after the start of mobilization [11].

Two-Stage Tendon Grafting

Joint contractures, extensive damage of the pulley system or scarring of the flexor tendon sheath preclude one-stage reconstructions. A two-stage procedure is a very elegant, but challenging method to restore functional digital ROM. At the first stage an optimal situation for the future tendon graft is prepared. This can include joint contracture release, pulley reconstruction and the preparation of a new flexor tendon bed by creation of a pseudosheath.

Stage One

At the first stage, the flexor tendon sheath is completely exposed as described for one-stage tendon grafting. Contracted joints are released. All scarred tissue is meticulously removed. Remnants of the flexor tendons often are adhered to the sheath and should be excised as far proximally as needed. The FDP tendon is excised as far proximally as the palm or distal forearm depending on the length of the future graft. The remnants of the FDS tendon are excised as far proximally to prevent adhesion to the future tendon graft.

A thorough inspection of the quality of the remainder of the flexor tendon sheath and pulleys follows. Dysfunctional pulleys are identified and the essential pulleys are reconstructed as described later in this chapter. At this stage, the silicone implant can be introduced. For men a 4–5 mm implant is usually chosen, whereas women and children often require a smaller, 3–4 mm implant. The implant is secured distally by connecting it to the FDP stump or to the base of the distal phalanx. It is then threaded proximally underneath the (reconstructed) pulleys into the palm or between the FDS and FDP tendons in distal forearm. The proximal end is placed close to the future motor tendon and left unattached Fig. 4.5).

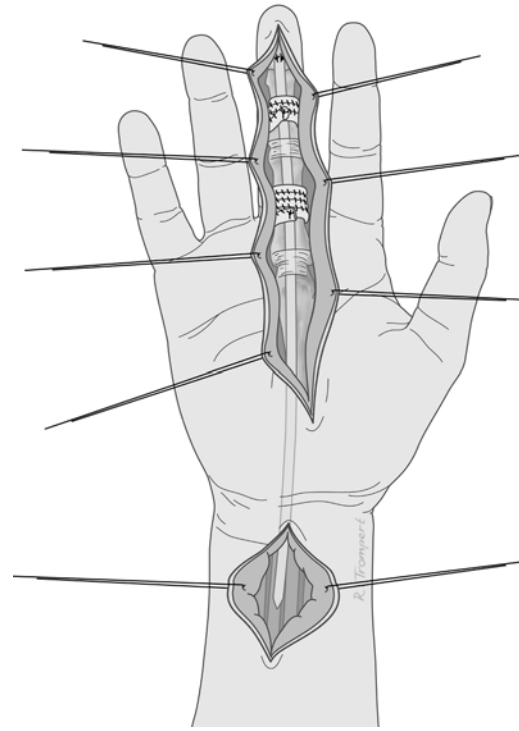

Fig. 4.5 The silicone implant is secured distally and threaded proximally through the (reconstructed) pulleys into the distal forearm. The proximal end is left unattached

The silicone rod should glide without restrictions. If it buckles with passive flexion of the digit, the source of the obstruction should be identified and adjusted (*e.g.* a reconstructed pulley that is too tight).

Then, the skin can be closed and the hand is covered in a bulky compressive dressing with the wrist and MCP joints in slight flexion.

Passive guided motion exercises are started 7–10 days postoperatively. Hand therapy between stages one and two is essential to prevent joint

contractures and to keep the silicone rod gliding in the new tendon sheath.

Stage Two

The second stage is scheduled after sufficient time for the soft tissues to heal and the pseudosheath to form. A 3 month interval between stages is recommended. Ideally, the second stage should only function to harvest, place and tension the final tendon graft in the prepared digit.

For tip to palm grafting, the palmaris longus tendon is a popular choice. If longer grafts are needed, or the palmaris longus is not available, the plantaris tendon or long toe extensors are effective alternatives.

The pseudosheath and the volar scars should be left undisturbed as much as possible. The silicone rod is approached through minimal incisions at the fingertip and the site of the proximal juncture. Distally, the silicone rod is released from its insertion. The tendon graft can then be attached securely to the distal silicone rod and very gently pulled proximally trough the pseudosheath into the proximal wound (Fig. 4.6). The rod is detached and discarded. Again, the distal juncture is fixed before the proximal juncture to the motor.

The original muscle to the excised tendon is the motor of choice. However, if passive excursion of the motor is less than 2–3 cm upon traction, it is advised to use an alternative. An adjacent FDP motor is a good second choice. If multiple tendons are reconstructed, or if the FDP is unavailable, the FDS can also be chosen as a motor to the tendon graft [11].

Tension to the graft is set as described above for one-stage tendon grafting. Also, the postoperative hand therapy program is similar.

Paneva-Holevich

An alternative to free tendon grafting is the pedicled tendon graft described by Paneva-Holevich [47]. A tendon loop is created by connecting the cut ends of the FDP and FDS tendons in the palm in an end-to-end fashion in the first stage of reconstruction after placing the silicone rod as for a conventional graft reconstruction. At the second stage, the FDS tendon is transected at the musculotendinous junction in the distal forearm. By placing distal traction on the FDS in the palm, its proximal end can be pulled into the palmar wound and threaded distally through the flexor tendon sheath to the distal phalanx. The advantages are the lack of donor site morbidity and the fact that after stage two, only the distal juncture needs healing since the proximal end-to-end juncture has healed from the first stage.

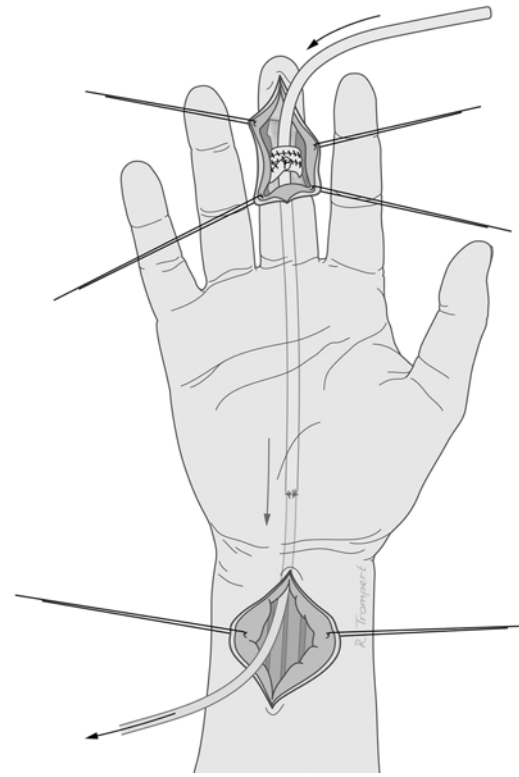

Fig. 4.6 The tendon graft attached to the distal silicone rod is very gently pulled proximally trough the pseudosheath into the proximal wound

Pulley Reconstruction

A functional, intact pulley system effectively transforms longitudinal tendon excursion into angular motion of the MCP and interphalangeal joints [48]. Disruption of the pulley system causes reduced efficacy of tendon excursions, resulting in reduced ROM and power grip. Intact A2 and A4 pulleys are the minimum necessary for near normal function [49–51]. Addition of an intact A3 pulley improves function further [35].

Damaged pulleys are primarily repaired if possible [52]. But as often is the case in flexor tendon reconstruction candidates, the possibility of pulley repair has passed and reconstruction is indicated. At least the A2 and A4 pulleys need to be reconstructed. In addition, the A3 pulley can be reconstructed if it seems suitable.

Location and width of the pulleys should be reconstructed as close to normal as possible for maximum efficacy. Also, careful tensioning of the pulleys and sufficient strength to allow for early mobilization are required [35, 52].

Most popular techniques for pulley reconstruction are the Okutsu triple loop, the Kleinert/Weilby tendon weave, the Lister extensor retinaculum wrap and the Karev belt-loop techniques. Each of these has their own advantages and disadvantages. What they do have in common is the use of autologous tissue only. Some studies suggest using synthetic materials, but this seems only rarely indicated.

The Okutsu triple loop technique uses a tendon graft (*e.g.* a resected FDP or FDS tendon) to encircle the proximal or middle phalanx for reconstruction of the A2 or A4 pulleys respectively [53]. The use of three loops is recommended for maximal strength and efficacy. Originally, the loops around the proximal phalanx were placed under the extensor apparatus dorsally and those around the middle phalanx over the extensor apparatus (Fig. 4.7) Others however, recommended the loops to always be placed deep to the extensor system [19]. The triple loop technique has been proven strong and effective [54].

Lister advised to reconstruct pulley using a strip of extensor retinaculum to wrap around the phalanx [55]. It provides excellent tendon gliding, but is less strong and requires disruption of the extensor retinaculum [52, 56].

The Kleinert/Weilby method uses remnants of the pulleys attached to the phalanges to weave a tendon graft through [57] (Fig. 4.8). Tensioning is considered to be easier with this technique, preventing issues with reconstructions that are too tight (causing synovitis and friction) or too loose (causing bowstringing). It does however, seem to be less strong [52].

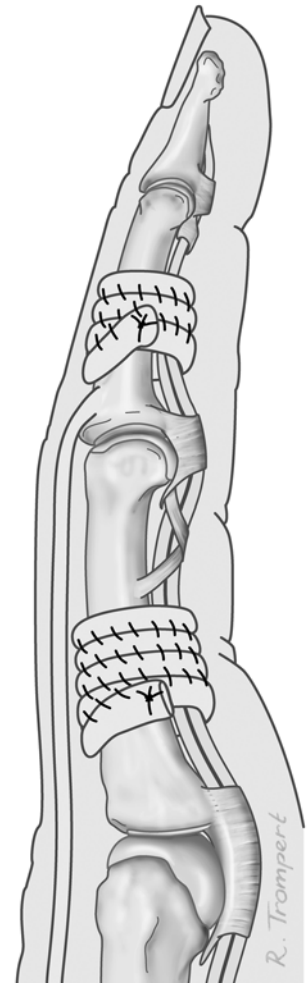

Fig. 4.7 The Okutsu triple loop technique for pulley reconstruction

Finally the Karev belt-loop is worth mentioning. The volar plate at the PIP joint is transversely incised proximally and distally and lifted volarly [58]. The resultant tunnel can be passed through by a tendon graft or repair. The resultant pulley is strong, but is unfortunately located at the A3 position and may be too tight [52].

Outcome

Unfortunately, not many reports on the results of one-stage grafting are available. Künzle et al. reported excellent results in 20 %, good results in

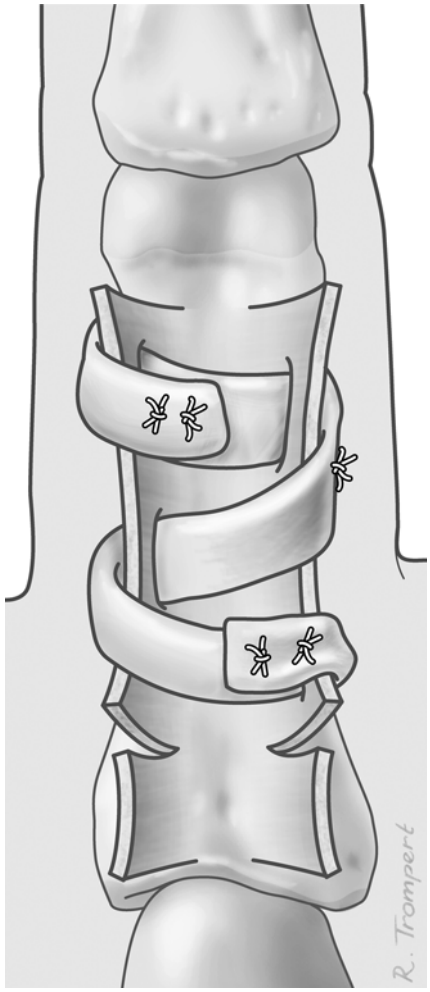

Fig. 4.8 The Kleinert/Weilby method of pulley reconstruction. A tendon graft is weaved through the remainders of the pulleys

36 %, fair results in 32 % and poor results in 12 % of their 25 cases [59].

Despite the notorious reputation of two-stage flexor tendon reconstruction in zone II, results of various large patient series published over the past decades show reasonable outcomes in the majority of their cases.

LaSalle et al. reported excellent and good results in 16 and 23 % of their 43 reconstructions [60]. In a larger series of 130 fingers published by Amadio et al., 54 % had good or excellent results [61] in reconstructions of zone I to V injuries. Coyle et al. achieved good and excellent results in 24 of 35 patients [62].

An average increase of 74° of total active motion and increased power from 20 to 79 % of normal was reported in a series of 150 fingers, 81 % of which were zone II injuries [63]. In another study of 33 fingers and 10 thumbs, the average time away from work was 44 and 101 days after the first and second stage, respectively [64]. In a recent retrospective analysis of 61 patients (106 fingers), good to excellent results were obtained in 84 %, fair in 12 %, and poor in 4 % of patients [65].

The Paneva-Holevich technique of pedicled tendon grafting combined with placement of a silicone rod at the first procedure resulted in good and excellent results in 73–82 % of the cases, depending on the used evaluation scale [66].

Most of these reports conclude that for the unfavorable cases of flexor tendon injury in zone II with poor prognoses, two-stage tendon reconstruction is a most useful therapeutic option.

Complications

Tendon adhesions are the most frequent complication of flexor tendon reconstruction. In fact, tenolysis is sometimes referred to as the "third stage" of tendon reconstruction [12]. Tenolysis rates of 7–47 % have been reported after two-stage tendon reconstructions [12, 60, 61, 67].

Other complications associated with two-stage tendon repair are infection of the implant, rupture of the tendon graft or junctures, migration of the rod, flexion contractures, reflex sympathetic dystrophy and problems associated with tensioning of the graft (quadriga or lumbrical plus) [63, 64].

In many cases, these complications require surgical intervention. In a series of 43 patients, Finsen reported 26 additional surgeries to be required after stage two in 18 of the 43 fingers or thumbs [64].

Flexor Tendon Reconstruction of Zone III to V Injuries

Presentation

As in zone I or II injuries, delay in presentation, failed primary repair or extensive associated soft

tissue injuries present the need for tendon reconstruction in zones III to V. Lacerations or crush injuries to the palm, carpal tunnel or distal forearm are frequently associated with neurovascular damage, contributing to functional impairment.

Non-traumatic, closed flexor tendon rupture can also present an indication for tendon reconstruction. Flexor tendons ruptures caused by attrition to bony prominences associated with carpal disorders (*e.g.* Kienböck disease), malunited distal radius fractures, a hook of hamate fracture or attrition to volar locking plates on the distal radius have been described [68–71]. In these cases often a segment of tendon substance has been damaged, which precludes primary repair.

Treatment

Taras and Kaufmann describe that reconstruction of flexor tendons in zones III to V can be done by placement of an interposition graft, transfer of an FDS tendon or by end-to-side FDP tenorrhaphy [19].

A tendon interposition or "bridging graft" can be placed in isolated defects of the FDP or FDS tendon. If both tendons are involved, usually only the FDP is reconstructed using the FDS remnants for grafting. Bridging grafts can vary in length from only few centimeters to fill a tendinous gap, to the length between the palm and the musculotendinous junction in the distal forearm [72].

Ample exposure is advised, using curved incisions extending from proximal to distal uninjured areas. This also facilitates the identification of nearby neurovascular structures. All affected tendons and surrounding scar tissue are excised. If damaged tendon resides in the flexor tendon sheath distally, the procedure is converted to a zone II tendon reconstruction.

After careful debridement, the gap that needs to be bridged can be measured. Suitable remnants of the FDS tendon or the palmaris longus are the grafts of choice. For a gap of 2 cm or less, sutures can be threaded longitudinally through the graft with Bunnell crisscross sutures placed in the tendon proximally and distally of the graft [72]. Individual tenorrhaphy sutures should be placed at the proximal and distal junctures if the graft is longer than 2 cm. Tensioning can be challenging when using interposition grafts.

Alternatively, an adjacent intact FDS tendon can be sacrificed to construct an end-to-end juncture to the distal segment of the injured tendon [19, 73]. If the juncture is to be placed in zone III, the FDS tendon should be routed dorsally of the neurovascular bundle. In zone IV, it should be passed under the median nerve.

A less invasive technique is the end-to-side juncture of the distal segment of the injured tendon to an adjacent, intact FDP. This method seems most suitable for zone V injuries, but favorable outcomes have also been published for tendon ruptures due to hook of hamate fractures [19, 74].

Passive ROM exercises are allowed from the first postoperative visit. Active ROM can be initiated at 2 weeks after surgery. After 4–5 weeks, resisted exercises can be started.

Outcome

Flexor tendon reconstructions in zones III to V have more favorable outcomes than those in zone I or II. Proximal to the flexor tendon sheath adhesion formation is less likely, and there is more space available for tendon junctures. Published reports of results however, are sparse.

Bridging grafts resulted in satisfactory function in 28 of 37 fingers in a series of Stark et al. [72]. FDS to FDP transfers resulted digital ROM greater than 180° in 10 out of 16 cases in a study published by Scheider et al. [73]. The end-to-side FDP junctures gave satisfactory functional results in a small series of Milek et al. [74].

Complications

Adhesion formation is less likely to occur outside the flexor tendon sheath. But zone III to V reconstructions are not devoid of problems associated with adhesions [73]. Also, ruptures of tendon junctures, problems with tensioning (*e.g.* loss of extension) and the need for additional surgical procedures have been reported [72, 73].

Reconstruction of Thumb Flexor Tendon Injuries

Presentation

The thumb is considered separately because of its unique anatomy. The flexor pollicis longus is the only tendon passing through the thumb flexor sheath. Anatomy of the flexor sheath is also distinctively different from its counterpart in the fingers. Of the three pulleys in the thumb flexor sheath, the oblique pulley has the most functional value. This should be considered if pulley reconstruction is required.

The majority of FPL injuries are caused by lacerations in zone TI or TII. Most of these injuries are eligible for primary repair up to 6 weeks after injury. But similar to the fingers, delay, scarring and extensive injury to multiple surrounding tissues or infectious complications may precipitate the need for one- or two- stage tendon grafting. In other cases, segmental tendon damage may require reconstruction. For example, an FPL tendon ruptures due to attrition against a volar plate on the distal radius, or against bony spurs in the carpal tunnel in rheumatoid arthritis patients (Mannerfelt lesion) may not be eligible for primary repair [75].

Selection of patients eligible for reconstruction of the FPL is based on the same principles as those for patients with flexor tendon injuries to the fingers. Important is the presence of good passive ROM of the interphalangeal joint.

Treatment

Alternative treatments to FPL reconstruction should always be considered. Fusion or tenodesis of the thumb interphalangeal joint often merits good functional results, but does reduce power grip.

Surgical techniques and postoperative hand therapy programs are similar to those described for the fingers [35]. A one-stage tendon graft between the distal tendon stump and the FPL motor is the treatment of choice if the pulley system and tendon sheath are functional. The palmaris longus is a suitable graft [35], but the plantaris or a strip of the flexor carpi radialis tendon have also been used [76]. Placement of the proximal juncture in the carpal tunnel should be avoided [35]. If there is extensive scarring or the oblique pulley needs to be reconstructed, a two-stage tendon reconstruction can be considered. If the FPL motor has contracted beyond function, an FDS tendon transfer from the fourth finger to the thumb can be used to replace the motor and the tendon [77, 78].

When tensioning the reconstruction, the wrist is held straight, the first metacarpal at 30° of anteposition and abduction, and the MCP and interphalangeal joints at 15 and 45° of flexion respectively [12, 35].

Outcome

Good functional outcome has been described for one-stage tendon grafting of the FPL [79]. Also, two-stage reconstruction has gained favorable results. In a series of 16 patients with two-stage reconstructions adequate function was restored in 75 % [76].

In a smaller series Weinstein et al. describes results of 5 Boyes grade 2–5 thumbs. Functional results were fair in two and poor in one thumb requiring pulley reconstruction. Two thumbs without pulley reconstruction had good and fair functional outcome [80].

Transfer of the FDS of the fourth finger had good results in 12 of 14 patients in a study by Schneider et al. [78]. Good postoperative mobility of the thumb interphalangeal joint was obtained after one- and two-stage FDS transfers described by Posner [77]. In the same study, mobility of the donor ring finger was unimpeded in all cases.

Complications

Complications of FPL reconstruction include the need for tenolysis or failures of the reconstruction due to tendon rupture. Also, complications associated with the silicone rod such as implant

migration or infection are conceivable. Sacrificing the FDS tendon for transfer can cause donor finger morbidity such as swan-neck deformity or insufficient finger flexion [81].

Flexor Tendon Reconstruction in Children

General principles and indications of flexor tendon reconstruction also apply to the pediatric patient. However, especially in young children, additional challenges may arise with all facets of care from diagnosis, imaging, consent, surgical technique, pain management to postoperative care.

Closed and open tendon injuries are often diagnosed late in children, resulting in delayed presentations. In injuries such as a jersey finger (closed FDP avulsion) or even in case of a laceration injury, the tendon injury can be missed on the initial presentation. Particularly if the patient is young, uncooperative and anxious, thorough clinical history taking and examination pose a challenge.

Observations of the parents on use and limitations of the child's hand and fingers are very valuable when obtaining a clinical history. Physical examination should evaluate the same aspects as in the adult population, including careful observation, location of scars, position and ROM of joints, including wrist tenodesis. Compression of the muscle bellies in the forearm can be helpful to assess passive digital flexion and thus tendon integrity in the pediatric patient. Ultrasound imaging may be a valuable addition in the diagnostic process.

Principles of operative technique of single and two stage tendon reconstruction are the same as in the adult patient. Dealing with smaller anatomy can present an extra challenge. Appropriately fine instrumentation and silicone rod size selection are warranted. One should be aware of physeal plates in the growing patient when anchoring the distal FDP insertion into the distal phalanx. Bone anchors or trans-osseous pull out sutures can damage the physeal plate [82]. Therefore, passing pull out sutures around the bone is preferred instead.

Postoperative management of children with flexor tendon reconstructions remains a topic of debate. Generally, postoperative immobilization of 3–4 weeks is considered safer than early active motion protocols in (young) children [61]. Pediatric patients are less likely to have long term tendon adhesions requiring tenolysis after flexor tendon surgery. Also, immobilization reduces the risk of morbidity involved with tendon ruptures. Some reports however, demonstrate good results with early mobilization protocols with strict therapist supervision [82].

Tendon Graft Donors

For flexor tendon reconstruction, most popular donor grafts are the palmaris longus and plantaris tendons. But other tendons are available. Each donor tendon has its own advantages and disadvantages making them more or less suitable for specific reconstructions.

The palmaris longus tendon has about 10–16 cm of usable length, making it suitable for fingertip to palm grafting [12, 83]. It is approximately 3–5 mm wide and 1–2 mm thick [12]. It is situated conveniently close to the operative site, but absent in approximately 15–25 % of people [19]. It is harvested using a tendon stripper trough a small, transverse incision at the wrist crease.

The plantaris tendon has 20–35 cm of usable length and has an average diameter of about 2–3.5 mm) [12, 83–85]. This makes it the preferred tendon for fingertip to distal forearm grafting. It is present in 80–97 % of patients [84, 86]. Its presence can be determined by CT scan preoperatively [12]. The plantaris is harvested with a tendon stripper through a 5 cm longitudinal incision anterior to the medial aspect of the Achilles tendon [19].

The extensor digitorum longus tendon to the second to fourth toe averages 35 cm in length and about 2–2.5 mm in diameter [85]. These extensors are always present, but like the plantaris requires surgery to an additional extremity. One or more of the tendons can be harvested with a tendon stripper through a transverse incision over the dorsum of the foot. End-to-side tenorrhaphy of the distal

remainder to an intact adjacent tendon restores extensor function to the donor toe.

The extensor digiti minimi and extensor indices proprius tendons are about 16 and 13 cm long respectively with an average diameter of 3 mm [83, 85]. The proprius tendons are identified ulnar to the extensor digitorum communis tendons of the same finger through a transverse incision over the MCP joint. They are transected and pulled out through a second incision proximal to the wrist. These tendons are close to the operative field, but their use can result in an extension lag in the MCP joints of their respective digits.

Conclusions

Flexor tendon reconstruction can be a very rewarding challenge in hand surgery practice. Careful diagnosis, patient selection and reconstructive planning are of paramount importance to outcome.

One-stage tendon reconstruction is indicated when primary end-to-end tendon repair is impossible. There should be no significant scarring of the tendon bed. Skin cover and the pulley system should be adequate and no additional procedures on bone or joints should be required. In these cases the tendon defect can be replaced in a single procedure by a tendon graft.

A two-stage procedure is needed if one-stage reconstruction cannot be done due to scarring of the tendon sheath, suboptimal state of the joints or the need for pulley reconstruction. In the first stage, pulleys are reconstructed and capsulectomy of stiff joints can also be performed if required. A silicone rod is implanted under the native or reconstructed pulley system. During the following months a pseudosheath forms around the silicone rod. The second stage is planned if scars are matured and a sufficient gliding sheath has formed, usually 3 months after the first stage. The silicone rod is then replaced by a tendon graft that is placed within the newly formed pseudosheath.

Before embarking on a reconstructive path, time should be invested in thorough preoperative patient counseling and patient selection. Generally, results are good but one- and two-stage procedures are far from devoid of complications. These complications can be minimized by careful surgical technique using ample exposure of the operative field, meticulous tissue handling and close cooperation with an experienced hand therapist for post-operative rehabilitation.

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Malunion/Nonunion of the Digits

5

Carlos Irisarri

Keywords

Phalangeal malunion • Metacarpal malunion • Phalangeal non-union • Metacarpal non-union • Phalangeal remodelling • Metacarpal remodelling • Phalangeal osteotomy • Spike osteotomy • Metacarpal osteotomy • Finger arthroplasty

Introduction

The vast majority of fractures of the metacarpals and phalanges have a strong tendency to unite, especially when located within the cancellous part of these bones, and can be treated with closed manipulation and splinting in the position of function. Unfortunately simple hand fractures are often misdiagnosed or treated poorly, with consequent deformity and stiffness.

Fingertips point to the scaphoid tuberosity when a fist is made. Observing the plane of the fingernails after closed manipulation or fixation of the fracture, any residual rotational deformity is easy to see. Unfortunately, however, it is not always detected and becomes more obvious only after healing. Ideally any malrotation should be corrected at the first treatment, as it is more difficult to undertake later. In children, early recognition is imperative because given the rapidity with which their fractures heal, by the time the patient is referred to a hand surgeon, weeks or even months may have passed.

If bone healing has been achieved with significant loss of the normal anatomic geometry, we are faced with a malunion. Non-union is defined when bone healing after a fracture is absent and without the option for spontaneous union. The management of malunion and non-unions is very complex and must be individualized. Not only the skeleton needs to be treated, but also the gliding of flexor and extensor tendon mechanisms, as well as joint mobility, need to be addressed. Concomitant problems related to skin cover, sensibility and vascularity may also limit the functional outcome for the injured finger.

Aetiology

In the phalanges, non-unions are often due to insufficient reduction and/or excessive distraction of the fragments. Too short a period of immobilization is another frequent cause,

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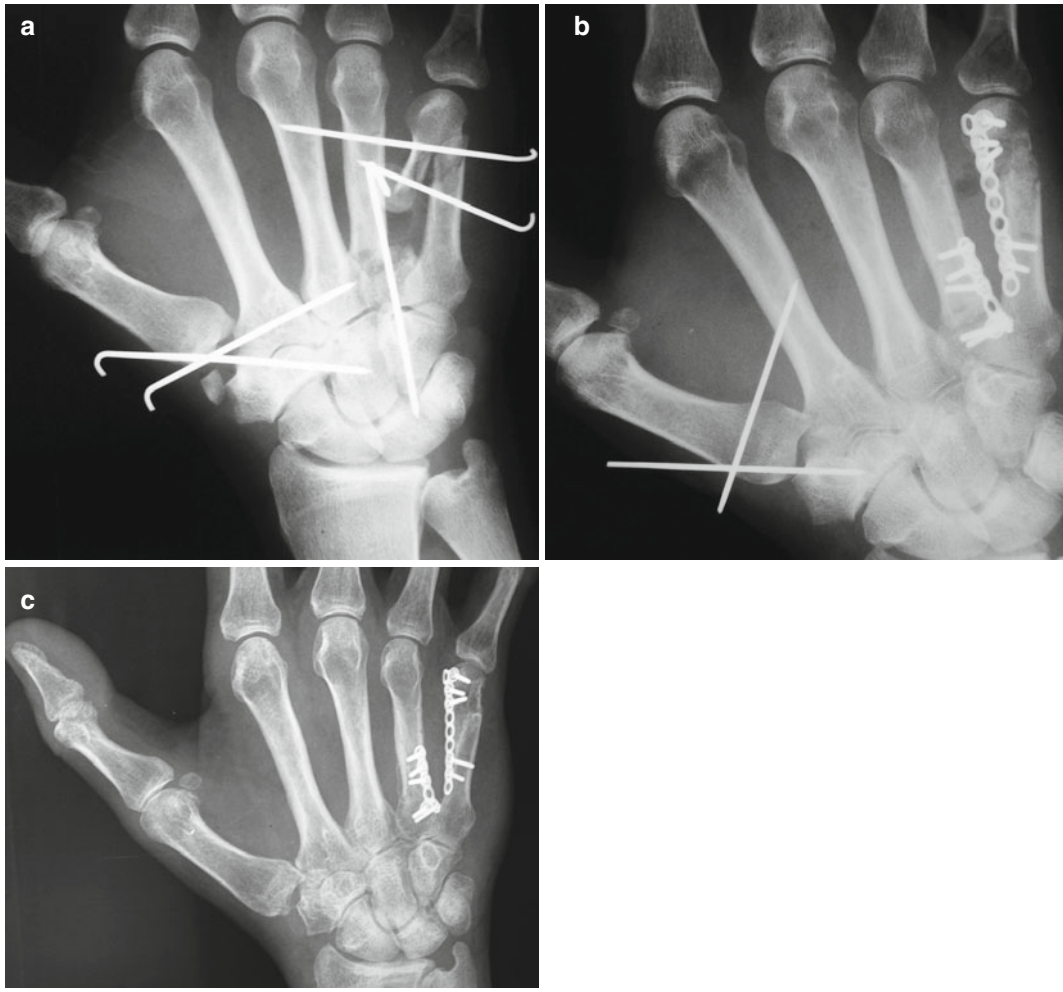

Fig. 5.1 (**a**) Crush injury with TM dislocation, and fractures of the fourth and fifth metacarpals. Initial treatment with percutaneous K-wires, performed at an outside Hospital before transfer to our institution. (**b**) TM joint ORIF. Dorsal plate in the fourth metacarpal, and laterally applied in the fifth metacarpal, with supplemental cancellous bone graft. (**c**) Final X-ray

especially in young patients who do not take adequate care of their splints, or even take their splints off after a few days. For this group of patients open reduction and internal fixation (ORIF) may be the better option, even though a reasonable result may be obtained by conservative means. With other closed fractures, however, surgery is indicated, for instance in displaced unicondylar fractures with soft tissue interposition. In the metacarpal, non-unions are more likely to be seen following open injuries and often following a complex injury mechanism (machinery accidents and gunshot wounds).

Most non-unions can also follow inadequate operative treatment and are often related to poor fixation of the fracture, sometimes in a distracted position. K-wires holding the fragments apart has been encountered several times in our practice (Fig. 5.1a–c). Too early removal of the K-wires is another reason for failure.

It is difficult to definitively establish the presence of a non-union in a bone of the hand. The radiographic appearance alone is unreliable. The majority of closed hand fractures, without mal-alignment, are solid enough to begin motion at 3 weeks, which is long before

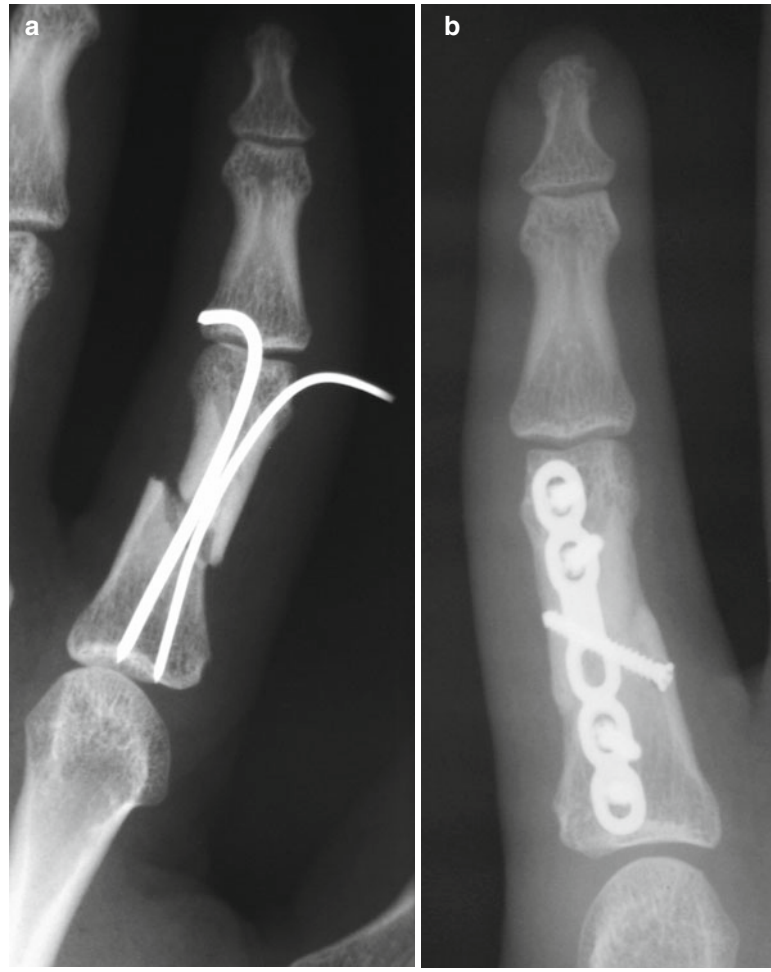

Fig. 5.2 (**a**) Failed percutaneous fixation with K-wires. (**b**) Secondary ORIF with a plate and a lag screw

union appears on x-ray [1]. Frequently it is difficult to distinguish between delayed and non-union, particularly when there is bone hypertrophy. Deformity and implant failure after operative treatment are, however, reliable indicators of non-union.

In delayed unions with hypertrophic callus formation, fracture lines can be seen radiographically for several months in a fracture that will subsequently heal uneventfully. If there is doubt then a ´wait and see´ policy is probably the best option, with continued splinting and only gentle mobilization. Surgical intervention only being undertaken when conservative treatment has clearly failed.

In contrast, in nascent malaligned fractures [2], our policy is to undertake open reduction and fixation (Fig. 5.2a, b). This approach saves time and often the procedure is easier. Conversely, children have the innate ability to remodel any fracture, the important factors being the skeletal age, the location of the fracture in respect to the growth plate and the amount and plane of malalignment [3]. In addition, in contrast to adults, stiffness is not a major concern in the pediatric population.

Classification

A Metacarpal Malunions

Metacarpal malunions may be classified as either extra-articular or intra-articular.

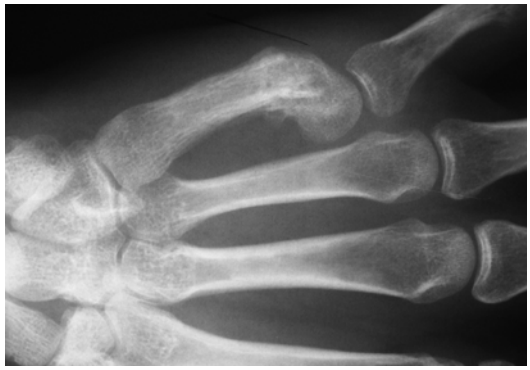

Fig. 5.3 Malunion after fifth metacarpal neck fracture. Disability was minor, and the patient refused surgery

Extra-articular malunions can be classified into three different patterns:

- Rotational malunion: appears after a spiral or oblique shaft fracture, causing a troublesome overlapping of the affected finger over an adjacent finger during flexion ('scissoring'). Each degree of metacarpal fracture rotation may produce as great as 5° of rotation at the fingertips. Cosmetic deformity is often marked and grip is impaired. Rotational deformities have poor prognosis in children, being the deformity that continues despite growth.
- Malunion with angulatory deformity: after a transverse shaft fracture, the pull of the interossei flexes the distal fragment, creating an apex dorsal deformity. This is particularly bothersome in the second and/or third metacarpals, as the index and middle CMC joints have less compensatory mobility than those in the ring and small fingers. Cases with severe angulation results in a dorsal prominence aesthetically displeasing and a prominent head in the palm. There may be compensatory hyperextension at the MCP joint that results in a pseudoclaw deformity when the digit is fully extended. In children, angulatory deformity in the plane of joint motion almost always remodels.

  Fractures to the neck of the second to fifth metacarpals are followed by characteristic volar angulation to a variable degree (Fig. 5.3). Because function is acceptable, minor degrees of angulatory malunion can be accepted, up to about 40° in the little finger, and 30° in the other digits [4]. Substantial angulated fractures of the metacarpal neck of the small finger (a so-called boxer's fracture) often present with a significant cosmetic deformity (sunken knuckle), although rarely, in our experience, palmar projection of the metacarpal head resulting in long term pain or indeed loss of function.
- Metacarpal shortening: usually appears after an oblique fracture with incomplete reduction. Loss of knuckle contour is noted, being located more proximal than normal.

  A severe shortened malunion occurs usually after an open crush fracture with bone loss. Strauch et al. [5] examined the relationship between shortening of the metacarpal and an inability to fully extend the MCP joint (extensor lag) in a study on cadavers. They observed an average 7° lag for every 2 mm of shortening. They speculated that the fact that this amount of lag is not usually observed clinically, may be related to the capacity of the MCP joint to hyperextend.

Intra-articular malunions have been classified into three types:

- Malunited intra-articular fractures of the base of the first metacarpal, which have healed with residual displacement of the joint surface. These cases are prone to develop secondary degenerative change in the mid to long term, although this is unpredictable. In addition, radiographic appearances do not always correlate with the clinical result.
- Malunited fracture-dislocations of the CMC joints of the long fingers. Unfortunately, early diagnosis is often missed as a clinical deformity is obscured by swelling on the dorsum hand. In addition, radiographic examination is also often inconclusive, requiring a CT scan to reveal the true nature and severity of the injury. If the fracture is missed, surprisingly in our experience the pain does diminish over 12 months or so, although the patient can be left with stiffness and deformity.
- Metacarpal head malunion: usually this occurs after an oblique fracture within the metacarpal head, often in the sagittal plane.

Any displacement of the joint surface leading to joint incongruity should be corrected as early as possible. This is also our policy with vertically or horizontally directed fractures in a coronal plane. In skeletally immature patients, subsequent deformity can also be a consequence of failure of normal growth due to an epiphyseal plate injury.

Phalangeal Malunions

Classified into four types:

- Rotational malunion: appears after oblique or spiral fractures of the proximal and middle phalanges. If severe, overlapping of the finger occurs whilst making a fist.
- Angulatory deformity: unstable shaft fractures of the proximal phalanx, typically present with angulation convex in a palmar direction. More rarely, lateral angulation can occur. Some deformities are mixed or multiplanar. Any angulatory deformity after a fracture of the middle phalanx is dependent upon the nature of the injury and location of the fracture. Distal fractures commonly show volar angulation, believed to be due to the strong pull of the flexor sublimis tendon on the proximal fragment, whilst the distal fragment is extended by the terminal extensor tendon. In a metaphyseal fracture, located proximal to the superficialis tendon insertion, the proximal fragment is pulled into extension through the action of the central extensor tendon. However, any deformity in displaced fractures is probably more related to the force and direction of the initial trauma, rather than any subsequent tendon pull.
- Articular malunion: the most frequent cause is a condylar fracture of the proximal phalanx. Weiss and Hastings [6] established an x-ray classification system, with four main patterns:
  type I: involved an oblique palmar pattern. The fracture line extends from the proximal metaphyseal flare of the distal aspect of the proximal phalanx, to the intercondylar sulcus at the distal articular surface. The small fragment lies palmar to the phalangeal shaft. This accounted for almost 80 % of condylar fractures, with the little finger the most commonly involved.
  type II: involved a long oblique fracture line, with the plane of the fracture sagitally oriented.
  type III: represents a dorsal coronal fracture, due to hyperflexion of the PIP joint.
  type IV: a small palmar coronal fragment, is produced by hyperextension of the PIP joint.

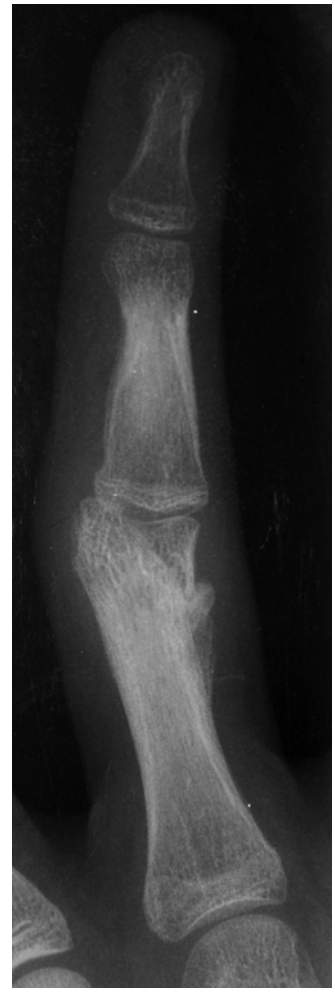

Fig. 5.4 Unicondylar displaced fracture, which was not properly treated, causing pain, deformity and limitation of flexion

Being highly unstable fractures, they are frequently misdiagnosed and/or inadequately treated (Fig. 5.4). Proper reduction with manipulation is impossible. The fractured condyle often suffers further displacement with significant

angulatory deformity and joint incongruity, frequently only discovered several weeks later. A malunited unicondylar fracture results in very considerable disability and is challenging to manage surgically. Preoperative evaluation is made by PA projection (to assess articular step-off) and lateral (to see palmar displacement of the condyle) and oblique radiographs to identify the fracture line.

Clinical Pearl

In unicondylar proximal phalangeal unstable fractures, oblique radiographs are mandatory to assess displacement. Reduction must be anatomic. Fixation with two screws is preferred, to allow early motion.

Malunion after fractures involving the condyles of the middle phalanx appears less frequently, especially after unicondylar fractures. In our series bicondylar fractures are more frequent. In these cases reconstruction is often impossible and a secondary DIP joint fusion may be necessary.

- Phalangeal shortening severe enough to be symptomatic is a rare complication. Vahey et al. [7] performed a cadaver study to determine the effect of shortening and angulation of the proximal phalanx on extensor lag at the PIP joint. However, a linear relationship was observed between the lag and shortening (12° of lag/1 mm of shortening) and increased angulation was seen to result in a proportionately increased lag. The severity of any associate soft tissue injury is more significant for the outcome than bone shortening.

Surgical Techniques in Malunions

Surgeons have long debated whether it is preferable to correct the malunion at the site of the fracture, or to perform a corrective procedure at a separate site. Osteotomy, at the site of the original fracture, has the greatest potential for correction of the deformity and subsequent contracture, although it also has the potential for producing adhesions. Extrafocal osteotomy is perhaps a better option after a complex fracture, allowing the surgeon to work with more healthy bone. Each case must be individualized and preoperative planning is crucial.

Surgical Techniques in Metacarpal Malunions

Extra-Articular Rotational Malunion

One option is the corrective step-cut osteotomy described by Manktelow and Mahoney [8]. Two hemitransverse osteotomies on opposite sides of the metacarpal diaphysis are produced and connected by a dorsal longitudinal cut. A longitudinal strip of dorsal cortex is then removed (it is important to remember that 1.3 mm of dorsal bone results in 1 cm of fingertip correction). Afterwards a longitudinal fracture is created in the volar diaphyseal surface. Bone fixation is undertaken by several lag screws. This osteotomy provides a large bone surface to promote healing, although it is technically a demanding procedure.

Osteotomy of the metaphysis is easier technically. Stern et al. [9] recommends placing a longitudinal mark on the metaphysis with an osteotome. The osteotomy is made with an oscillating saw, perpendicular to the mark. Afterwards a K-wire, inserted perpendicular to the distal shaft, acts as a ´joystick´ to correct rotation of the distal fragment. Fixation is accomplished with multiple K-wires, or preferably with a plate and screws.

Extra-Articular Malunion with Dorsal Angulation

A closing wedge osteotomy at the level of the fracture is a less demanding procedure technically. If possible, consider an incomplete osteotomy preserving the intact opposite cortex, or at least the volar periosteum allowing it to act as a hinge.

A triangular wedge is removed and a plate and screws are applied to allow good fixation and

early motion. Yong et al. [10] reported a double osteotomy, proximal and distal to the apex of the dorsal angulation, obtaining a trapezoidal segment of bone, which is rotated and re-inserted as a bone graft, again being fixed with a plate. If there is associated significant metacarpal shortening, an opening wedge osteotomy using a trapezoid interpositional iliac crest bone graft is undertaken, again fixed with a dorsal plate to allow early motion.

Metacarpal Shortening

If this is less than 1 cm and without angular deformity, it usually does not require correction. In extreme cases however, an opening wedge osteotomy with interposition of a structural bone graft, usually from the iliac crest, can be undertaken with a dorsal plate fixation. A gradual distraction with an external fixator is also an option, although associated soft tissue contracture may make this difficult.

Surgery after a high energy injury is more difficult and requires awareness with regard to local blood supply, concomitant nerve lesions and tendon injuries. In complex open fractures with bone loss, following thorough debridement provisional osseous stability is achieved with spacer wires, supplemented with external fixation. Rarely, skin coverage is attained in the emergency procedure and is usually performed over the next days when the wound is surgically clean.

For most open fractures with bone loss, a single block of corticocancellous graft harvested from the iliac crest and fashioned to fit into the defect, will provide the definitive bone reconstruction. In some more severe cases, however, a free vascular bone graft may be required.

Intra-Articular Malunion

The treatment of displaced intra-articular fractures of the base of the first metacarpal has improved with the use of fixation (K wires, screws and plates or mini external fixators), with the additional use of bone graft or bone substitutes as appropriate. This surgery, however, can be difficult and does not always have a favourable outcome. It is however worth attempting in young patients. To treat malunited symptomatic Bennett´s fracture, Giachino [11] reported removal of a block of bone of the radial aspect of the first metacarpal, big enough to correct the articular step-off, but leaving a radial basal fragment big enough to be fixed with two screws to the ulnar articular fragment. After complex articular fractures with residual depressed fragments, secondary reconstruction is rendered more difficult. In more long standing cases, if pain persists after initial conservative treatment, fusion of the TM joint is recommended, or alternatively in low demand patients, arthroplasty may be an option.

Fracture-dislocations of the CMC joints of the long fingers present in various guises. Often residual malunion with persistent CMC subluxation, particularly of the index and middle fingers, can be well tolerated. Chronic subluxation of the fourth and fifth metacarpals, however, is sometimes symptomatic and requires treatment.

In the early cases with a malunion of less than 8 weeks, the best option is an intra-articular corrective osteotomy through the fracture site. This should be supplemented by internal fixation. The aim of surgery is not only to achieve a congruent surface, but allow motion to begin as early as possible. Whilst K-wire fixation is an option, mini-cannulated screws may be more optimal. In the long term normal joint motion is rarely achieved, but reasonable grip and pain relief can be attained.

If congruent reduction is not possible, or there is severe cartilage damage, options include either arthrodesis, excision arthroplasty or silastic interposition. Trimming off any bony protrudement is also recommended.

Surgical Techniques in Phalangeal Malunions

A dorsal approach is usually preferred, preserving veins in the skin flaps and splitting the extensor tendon to expose the proximal phalanx. If possible, the tendon and the periosteum are elevated in separate layers. If tendon adhesions are present, tenolysis and /or capsulotomy is performed. In recent cases, especially in multiplanar deformities, we prefer to recreate the original fracture line

by mobilizing the fracture callus. Fixation has been improved with the new low profile titanium plates, easy to contour and even cut and available in a variety of configurations. The plate is applied on lateral surface when possible. Also self-tapping mini-screws are useful to fix step-cut osteotomies, which again allows early motion.

In mature malunions, different methods of treatment have been reported.

Rotational Malunion

The correction of rotational deformity is particularly difficult to undertake. Weckesser [12] recommended an osteotomy at the base of the corresponding metacarpal to correct this rotational deformity. He undertook fixation with a longitudinal K-wire and two horizontal K-wires through the distal fragment and adjacent metacarpal. Bindra and Burke [13] also described this procedure, this time securing the osteotomy by plate fixation.

This type of surgery is limited by the deep transverse metacarpal ligaments, which restrict rotation. Gross and Gelberman [14] undertook experiments on cadavers to determine the maximal rotational correction of a phalangeal malunion that can be achieved with a metacarpal osteotomy. It was possible to correct up to 18° of malrotation in the index, long and ring fingers and up to 30° of malrotation in the small finger. Releasing this ligament may result in loss of the transverse palmar arch and instability of the MCP joint and is therefore not recommended.

Angulatory Deformity

Volar angulation is difficult to detect when the fracture is at the base of the proximal phalanx, because it is almost always superimposed on the other phalanges on the lateral film. If, however, there is enough malalignment to predict functional loss, then surgery is undertaken. Our preferred technique is to recreate the original fracture line and undertake fixation with either cross K wires or a mini plate.

In mature malunion, an initial period of observation, particularly of the smaller deformity, is recommended to quantify the degree of dysfunction. If the latter is severe, a closing wedge osteotomy at the site of the malunion is performed, leaving, if possible, the opposite cortex intact, the latter acting as a hinge. Fixation is again performed with a mini-plate if proximal fragment is big enough.

For patients with angulatory deformity in the frontal plane, despite shortening the digit, closing corrective osteotomy leaving the opposite cortex intact, is the easiest procedure. Fixation is carried out using a mini-plate if the soft tissues are in a satisfactory condition and with 2 K-wires if they are not. Trumble and Gilbert [15] reported a practical way to design the osteotomy. Lines were drawn parallel to the shaft of the phalanx distal and proximal to the malunion. Afterwards, perpendicular lines are drawn at the location where previous lines intersect at the malunion, outlining in this way the wedge of bone to be removed.

The plate is initially secured to the proximal half of the proximal phalanx, although subsequently only one screw is maintained so the plate can be rotated out of the way, allowing the osteotomy to be undertaken. We would recommend using a power saw with a thin blade and saline irrigation to limit thermal damage. Afterwards the finger is rotated until it is parallel to other digits, with the PIP joint in 90° of flexion. Finally the plate is then secured to the distal fragment.

An opening wedge osteotomy, using a corticocancellous bone graft, requires a lateral plate fixation. This is, however, a difficult procedure and in our opinion very rarely indicated. Additional tenolysis and capsulotomy is carried out, when necessary.

Displaced neck fractures of the phalanges may be missed unless a true lateral radiograph is obtained, showing the rotated head fragment. If some bone contact is maintained, usually union is achieved, especially in young patients. As there is no epiphysis at the distal end of the phalanx, spontaneous remodeling of residual angulation is limited and only significant in young patients [16, 17]. If, however, there is a volar spike present, this can limit PIP flexion. Surgery to remove this spike, often termed osteoctomy or cheilectomy (Fig. 5.5a–c), restores the articular surface and can result in an improvement in flexion, particularly in children [18].

If the distal fragment is completely rotated, attempts with closed manipulation are often

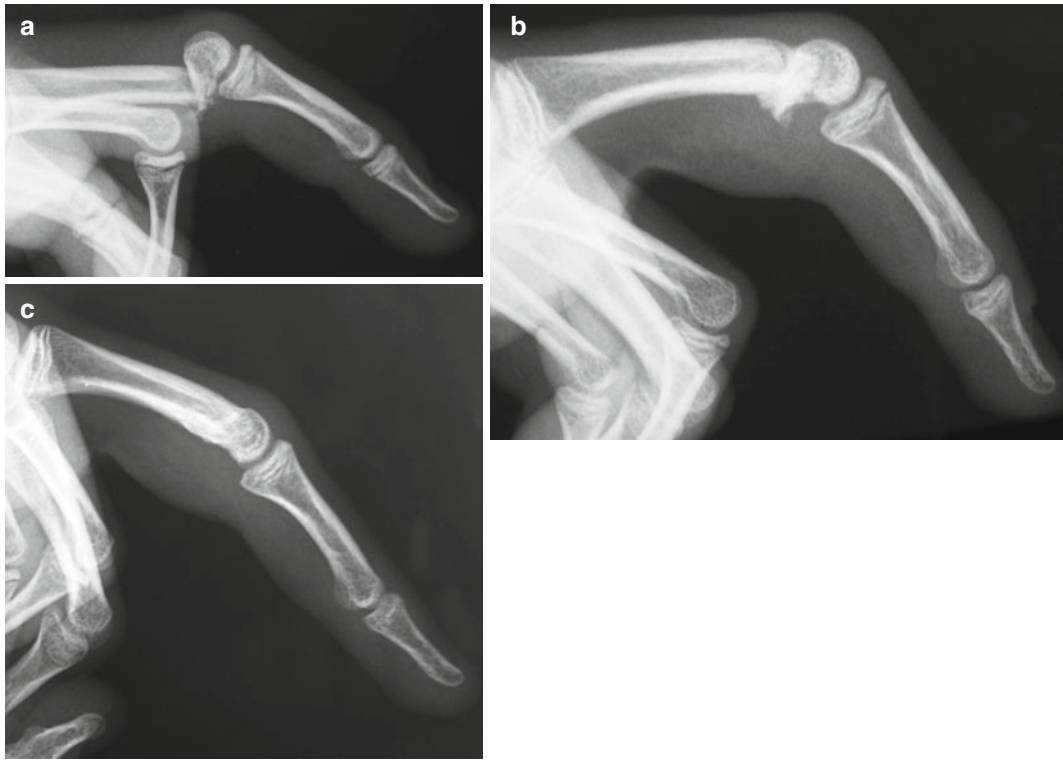

Fig. 5.5 (**a**) Displaced fracture of the neck of the proximal phalanx. (**b**) Malunion with volar spike blocking flexion. (**c**) Trim off the bony spike restored flexion

Clinical Pearl

Malunion of a neck fracture of the proximal phalanx, with flexion blocked by a volar spike, can be improved with osteotomy of the volar spike.

fruitless and open reduction and pin fixation is necessary. Even in children, some degree of residual PIP joint stiffness is usual. Waters et al. [19] described eight children with incipient extension malunion of proximal phalangeal neck fractures, treated with percutaneous K-wire reduction and fixation. The first K-wire was inserted into the fracture site, through the dorsal callus and used to mobilize the fracture fragment by breaking down the fracture. The wire then was used as a lever arm for fracture reduction. A second K-wire was used to pin the fracture.

Lateral angulatory deformity, after a neck fracture of the proximal phalanx is better treated with a closed wedge osteotomy. In young patients, fixation with two K-wires initially provides enough stability. In adults a mini-plate is preferred, despite being more demanding technically. In some spiral fractures, the distal end of the proximal fragment reaches into the side of the head of the phalanx. If the fracture is healed in a shortened position, a lateral bony spike may block flexion by obstructing the normal movement of the collateral ligament. Again, removing the bony spike often results in a satisfactory outcome.

Clinical Pearl

Proximal phalangeal malunion treated with corrective osteotomy, is fixed with a plate whenever possible. Minimal shortening is acceptable. If severe complications are present (osteomyelitis, joint stiffness, soft tissue loss), consider digit amputation.

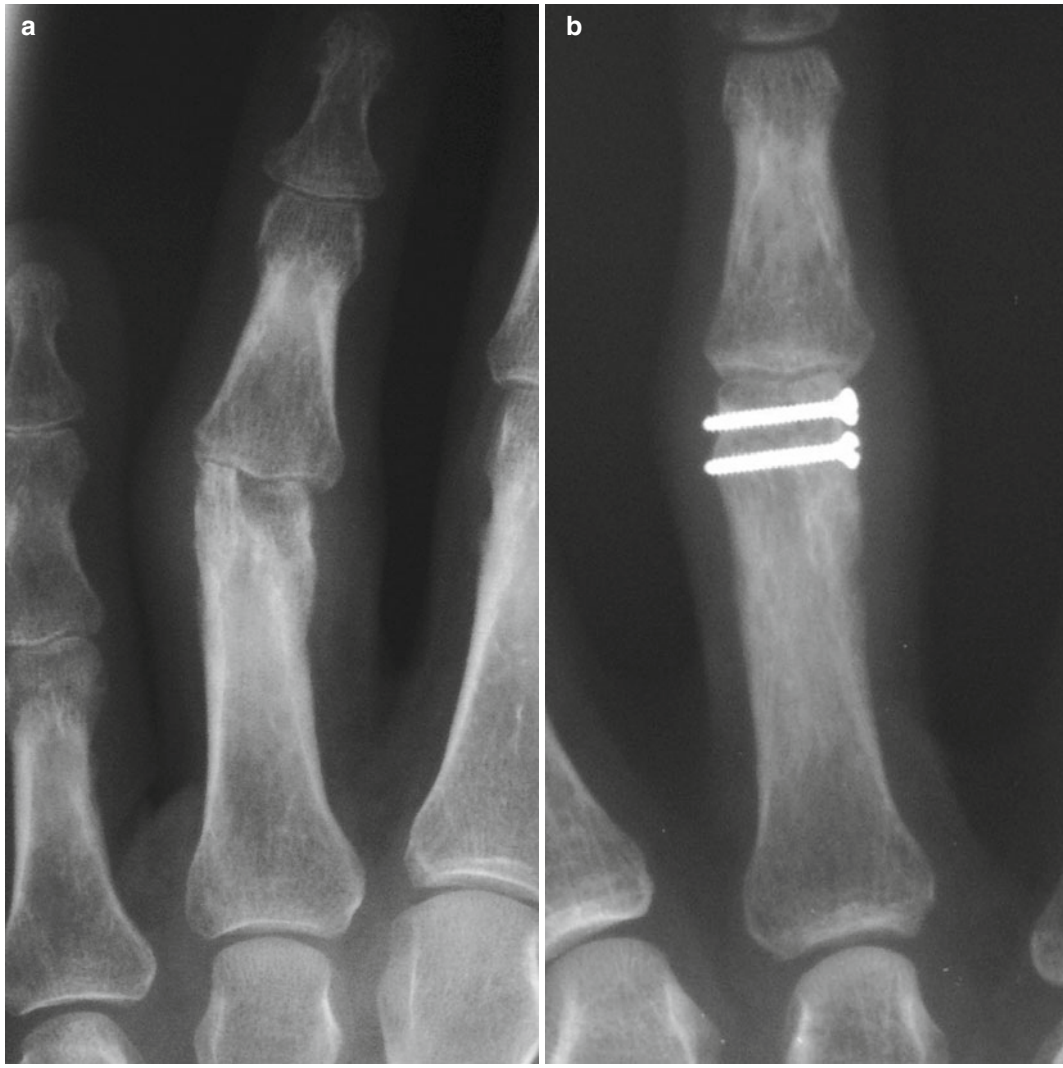

Fig. 5.6 (**a**) Nascent malunion after displaced unicondylar fracture. (**b**) Anatomic reduction fixed with two screws

In the middle phalanx, the more frequent angulatory malunion occurs with flexion of the proximal fragment [20]. We prefer to recreate the original fracture line and perform adequate reduction and fixation, usually with two crossed K-wires. Minor lateral angulation does not usually cause severe functional impairment.

Intra-Articular Malunion

The more common approach is between the central slip and the lateral band on the side of the fractured condyle. Flexing the digit allows the lateral band to move palmarward. Retraction under the central slip improves exposure of the joint. An alternative approach is a midlateral incision, displacing the lateral band dorsally before the arthotomy. When mobilizing the condylar fragment, take care not to detach the collateral ligament, to avoid avascular necrosis of the condylar fragment and future instability.

After a careful osteotomy through the original fracture line, undertaken with a sharp and fine osteotome, the condyle is reduced to its proper place using a dental pick. The reduction is maintained with a towel clip or small cannulated reduction forceps to facilitate transfixion, first with a thin K-wire and afterwards with a mini-screw. If the size of the fragment is big enough, the initial K-wire is replaced by a second screw (Fig. 5.6a, b)

to allow very early motion of the joint. It is important however, to make sure that the screw heads do not skewer the collateral ligament. This will limit their motion and result in stiffness of the joint. Similarly, the screws should only just engage the cortex on the opposite side. A too prominent protrusion will again lead to a soft tissue adhesion.

In cases with a small fragment, a single screw or even a K-wire can provide enough stability, provided it is supplemented by an appropriate period of post-operative immobilization.

Teoh et al. [21] reported in six patients a new 'condylar advancement osteotomy' for a malunited unicondylar fracture of the phalangeal head, to avoid the risk of avascular necrosis.

Their technique involves an intercondylar wedge resection, combined with a sliding advancement of the bone fragment (malunited condyle and supporting cortex). This larger fragment is easy to manipulate and can be fixed with two mini-screws. Supplemental cancellous bone graft may be necessary in some cases to fill a residual defect and insure healing. Bone substitutes can also be used.

Harness et al. [22] preferred a closing wedge osteotomy, based in the metaphysis proximal to the collateral ligament origins of both condyles. The initial cut is made with a fine blade, which is perpendicular to the shaft. The second and proximal cut is more oblique. The overlying periosteum is preserved and, where possible, fixation is made by an oblique K- wire with additional stainless steel tension band.

Froimson [23] and later Freeland and Lindley [20] published their experience in an established condylar malunion, of removal of a wedge of bone using a series of progressively smaller-diameter round dental burrs. After closure of the wedge with correction of the malrotation, stabilization is carried out with K-wires.

If the articular surface step is small, we often prefer to undertake an extra-articular osteotomy. We favour using a thin oscillating saw, or a fine osteotome after multiple 0.6 mm Kirschner drillings. Because union in a younger patient is not a significant issue, we would recommend K-wire fixation (Fig. 5.7a–c).

Articular malunions with severe degenerative arthritis are best treated by arthroplasty, if adequate soft tissues can provide a tight envelope for the implant [24]. Our preferred technique is to replace the joint with a silastic Swanson implant (Fig. 5.8a–c).

Many others options have been reported. Perichondrial resurfacing arthroplasty allows some motion, but with limited long-term success. Also, a costo-osteochondral graft has been used to repair defects of the head of the proximal phalanx [25]. Nonvascularized whole joint transfer ultimately develops avascular necrosis and progressive deterioration, with resultant poor long term outcome. Vascularized joint transfer avoids the problem of avascular necrosis, but it is a complex, expensive and time consuming procedure with potential donor site morbidity. The results are also variable.

Allograft replacement avoids donor site morbidity, but significant concerns exist regarding cartilage viability, host rejection and infectious disease transmission.

With regard to salvage procedures for complex fractures of the base of the middle phalanx, articular surface reconstruction has been attempted using a cortico-cancellous bone graft, especially hemi-hamate autografting. Even in the best of hands, however, this is a difficult procedure.

Phalangeal Shortening

Minimal shortening of the proximal or middle phalanx is well tolerated. Only in very rare cases, with severe shortening leading to an extensor lag of the PIP joint, does a bone graft need to be considered to lengthen the affected phalanx.

Surgical Techniques in Nonunions

Nonunions in the hand are usually associated with other problems, such as joint stiffness. In most cases, tenolysis and capsuloctomy are also required to improve the functional result. Firm internal fixation is necessary to maintain alignment, achieve healing and allow early motion. Distant autogenous bone-graft (especially from iliac crest), to encourage union, should be reserved for digits that will provide useful function once the fracture has healed. Finger amputation is preferred for non-unions associated with

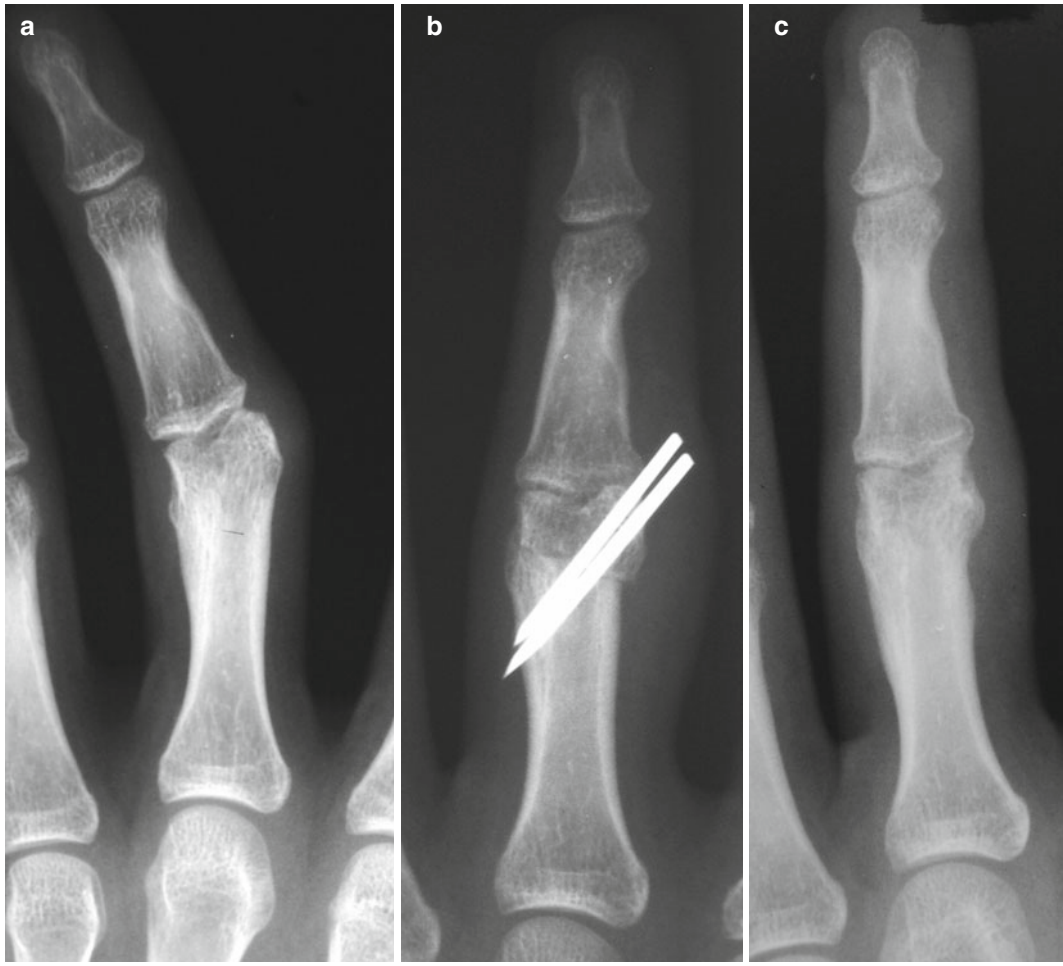

Fig. 5.7 (**a**) Malunited unicondylar fracture. (**b**) Extra-articular osteotomy. (**c**) Radiograph 1 year later

sensory loss and poor skin coverage. Such a finger is often excluded from use and can compromise whole hand function.

A stiff joint is unlikely to regain useful motion, even if the non-union heals. In these cases arthrodesis or arthroplasty may be a better option. In atrophic non-union, fibrous tissue is removed until fresh fracture ends are seen. Thereafter, the osseous gap is filled with a corticocancellous graft. Ring [26] described how a cancellous bone graft can be compressed and compacted into a syringe, providing a relatively compact and semistructural core of bone, inserted into the fracture site and the medullary canal of the phalanx, adding stabilization with a plate. A non-union of the thumb's proximal phalanx treated by this technique (case described with the permission of Dr. J. Jupiter's) is included in his report. Other techniques to promote bony healing have been described, including BMP, PRF, as well as ultrasound electrical stimulation. Reports of their successful use in the hand, however, are limited.

Infected non-unions are a very difficult challenge, especially in those cases with segmental bone loss. Treatment usually requires a two-stage procedure. At the first, debridement of infected and devitalized bone and soft tissue is carried out. Temporary external fixation and/ or a k wire adding a cement spacer (antibiotic-impregnated) maintains length and alignment after debridement. Well-vascularized skin coverage is, in most cases, possible with a rotational flap. A posterior interosseus flap is adequate to treat severe injuries of the dorsum of the hand. For resurfacing the dorsal

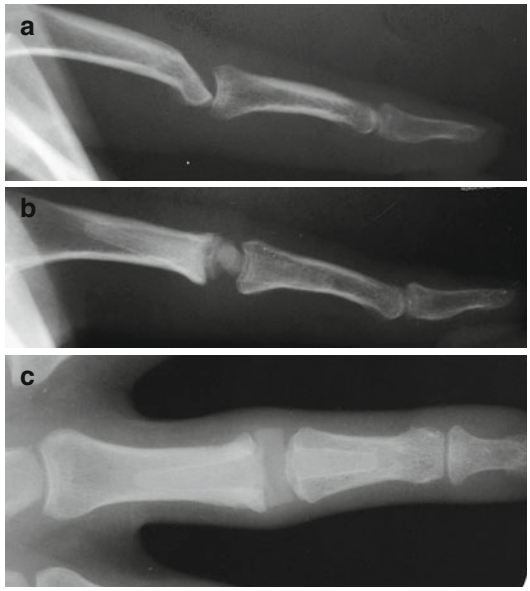

Fig. 5.8 (**a**) Coronal dorsal fracture. Fragment excised in the failed initial attempted ORIF. (**b**) Swanson implant was inserted 3 weeks later. Seven years follow-up. (**c**) Postero-anterior view

aspect of the thumb, the preferred option is a dorso-radial index flap. In long-fingers, the reverse dorsal intermetacarpal flap is very useful, especially the one based on the arterial axis from second metacarpal space. The dorso-commisural flap has also been used occasionally. Free vascularized flaps allow complicated salvage procedures, but do require greater surgical skills and expertise.

Once the milieu has been optimized at the second stage the cement spacer is removed and autogeneous bone-grafting can be performed. The bone itself should be shaped to fit the defect and provide intrinsic stability. A tailored, structural, cortico-cancellous bone graft from the iliac crest has been preferred in our practice. Supplemental cancellous bone graft is packed into any defect. If adequately fitted, frequently hardware can be minimal, with lower risk for infection.

Heroic efforts need to be balanced with donor areas morbidity, time and cost of treatment and clear cosmetic and functional outcomes. To be possible does not mean to be sensate. Frequently the patient is best served with amputation of the offending finger. It is also important to remember that tissue from an amputated finger can be used to reconstruct the adjacent digits.

Outcomes

We have found a number of reports on the results of treatment of metacarpal and phalangeal nonunions. Unfortunately, much of the available information is from retrospective studies, most of which are single case reports or a small series with little long-term follow-up. Malunions requiring surgical correction are even less frequent.

In addition meta-analysis is difficult because there are so many variations in patient selection, including the time to consider a fracture as a nonunion. Unfortunately, detailed evaluations of postoperative range of motion were not available for many patients. In our practice, malunions have been much less common in recent years. We believe this is due to improved assessments and treatments. Nowadays failed osteosynthesis is the more frequent aetiology.

- Outcomes after intra-articular fractures of the base of the first metacarpal:
  In some cases, where there is residual displacement of the joint surface, remodelling can occur. We have seen two patients with a clear step-off at the articular surface of the first metacarpal, but did not undergo surgery, both of whom have shown subsequent remodelling [27]. Whilst this is not the rule, most patients seem to function reasonably well. In our series 5 out of the 34 patients reviewed (including conservative and operative treatment), required a secondary fusion.
- Outcomes after fracture-dislocations of the CMC joints of the long fingers:
  We have reviewed 23 patients. One patient with residual subluxation in the second and third rays required fusion. Regarding chronic subluxation of the fourth and fifth mobile metacarpals, in our series only two heavy manual workers required a secondary fusion, in the first case after a malunited fracture-dislocation of the fourth and fifth metacarpalhamate joints and in the second one after an intra-articular fracture of the base of the fifth metacarpal, that have been missed initially, with residual dorsal subluxation of the CMC joint.
- Outcomes after fractures of the metacarpal neck:
  They are more frequent in the small finger and only rarely does palmar projection of the

metacarpal head become symptomatic. In doubtful cases prior to surgery, it is useful to compare with the other hand, to determine the normal neck-shaft angle (average 16.5° in the fifth metacarpal). Only few authors like Thurston [28] have recommended correction of malunion, arguing in his experience patients were frequently unhappy with the results of even 15° angulation. He reported 10 patients with fifth metacarpal angular malunion, corrected with a two-cut 'pivot'osteotomy at the metacarpal neck. One patient required a further procedure because union failed. In our series, the incidence of surgery was much lower and yet, it was only carried out in 2 young men out of 18 patients, all of whom had severe displaced malunions.

- Outcomes after intra-articular fractures of the metacarpal head:
  Significant improvement was reported by Duncan and Jupiter [29] in three cases, following an intra-articular osteotomy fixed with screws and/or plates.
- Outcomes in rotational deformities:
  Weckesser [12] reported the successful correction in two cases, with rotational deformity consequent to fractures respectively of the proximal phalanx of the index and ring fingers, by osteotomy at the base of the metacarpal. Otherwise, there is limited clinical data about this procedure and the literature does not provide firm guidance. Pieron [30] corrected a 15° rotational malunion after a oblique fracture of the first phalanx of the little finger in a professional violinist, performing the osteotomy through the proximal fourth of the fifth metacarpal. Fixation was carried out with a small plate, adding a volar plaster splint. The fragments were united in 6 weeks.

  Manktelow and Mahoney [8] reported uniform healing and functional recovery using step-cut mid-diaphyseal metacarpal osteotomy in ten patients with rotational malunions of the metacarpal. With the trapezoidal rotational bone graft osteotomy, Yong et al. [10] reported success in four metacarpal and two phalangeal angulated malunions. Pichora et al. [31] reported 16 phalangeal and 7 metacarpal osteotomies, all healed, but with residual stiffness in some cases after at least 3 months follow-up. They advised that metacarpal osteotomies may not fully alleviate rotational deformities. We found similar results in our own small series and Barton [32] also reported a variable experience with this technique.
- Outcomes in other diaphyseal deformities:
  Opening or closing wedge osteotomies at the malunion site have been reported for treating angular diaphyseal metacarpal and phalangeal deformities. Froimson [23] reported 5 children and 17 adults treated with opening/closing/rotational or combination osteotomies. The deformities were corrected and motion was recovered. Lucas and Pfeiffer [33] reviewed 36 patients treated by extra-articular metacarpal or phalangeal osteotomies to correct angular, rotational or combined deformities. Twenty-three patients were rated very good, eight good and five poor. Buchler et al. [34] reviewed the results in 57 patients, in whom a total of 59 osteotomies had been performed to correct deformities in one or more planes. The osteotomy was done at the site of the malunion and 50 % of the patients had a concomitant tenolysis and capsulectomy. Union was obtained in all patients and satisfactory correction was achieved in 76 %. A net gain in motion was observed in 89 % of the patients.

  Trumble and Gilbert [15] reported eleven patients, who had been treated with an extra-articular osteotomy secured with a plate and screws to correct a complex phalangeal malunion. Union and correction of the deformity were achieved in all patients. The average gain in motion at the proximal and distal interphalangeal joints was 15° and 10°, respectively.

  Jupiter et al. [35] reported 25 consecutive delayed unions and nonunions (16 in the phalanges and 9 in the metacarpals) in 23 patients. A variety of treatment techniques were used. Plate-and-screw fixation seemed to have achieved a more functional digit than Kirschner wire fixation, although treatment resulted in few digits with good function.

- Outcomes after implant arthroplasty:
  MCP and PIP articular malunions, with severe degenerative arthritis, treated with a silastic Swanson implant (Fig. 5.8a–c), have had satisfactory outcomes in the mid and even long-term [27]. In our experience, silicone synovitis has not been a problem. In some cases, however, joint mobility does diminish with time.

Complications

The two most frequent complications of this type of surgery are non-union and joint stiffness. Any associated stiffness should be addressed either prior to, or at the time of surgery. The surgery itself should include rigid fixation of the osteotomy/non-union where possible, thus allowing early mobilization. Despite this, however, postoperative stiffness can be a significant problem and patients should be warned of this. Rarely, an adjacent joint may spontaneously fuse. We have seen one case at the DIP joint [30] and a second one at PIP joint.

Subsequent non-union can also occur, particularly when the attempt is to salvage a digit after a very complex bone and soft tissue injury. Interestingly, these may be asymptomatic. It is important for the surgeon to remember that, even following optimal treatment, this may not result in a well-functioning digit. Final outcome is more often correlated to the severity of the initial damage. Extensor and flexor tendon tenolysis is particularly necessary to improve mobility. Patients should be aware that a second procedure may be often necessary to remove the plate. Intra-articular fractures, arthrolysis releasing the collateral ligaments and/or volar plate is frequently required again to improve motion.

When only a long finger is involved and the non-union is associated with nerve and tendon injuries that severely impair function, digit amputation should be considered, particularly in the presence of infection. Further treatment will require a considerable amount of time and effort and the cosmetic and functional outcome may again be poor. Only the thumb, although stiff and partially insensitive, deserves to be maintained.

We do not know exactly were non-union occurs most often in the hand. Reviewing our series, a similar incidence was found between the proximal phalanx of the thumb (most cases after failed operative treatment) and the distal part of the middle phalanx of the long fingers, usually after an initial and too brief period of conservative treatment. In several cases the cause was premature removal of K-wires.

Symptomatic non-union after a fracture of the shaft of the distal phalanx is rare. In some cases a permanent nail bed deformity occurs, which despite the cosmetic appearance, function is usually unimpaired and late intervention is seldom warranted. Pain may reflect damage to the soft tissues, rather than unhealed bone. In some cases, minimally invasive surgery, by way of fixation with a headless screw may provide enough mechanical stability to ease the pain. In cases of atrophic non-union with two large fragments, insertion of bone peg has been successful in the patient reported by Shinomiya et al. [36].

Other complications include complex regional pain syndrome, which, although uncommon, can lead to severe stiffness not only in the operated finger, but the hand generally. In the postoperative period, if the patient begins to complain of a disproportionate amount of pain and develops generalised hand swelling etc., it is advisable to involve a pain management consultant.

With regard to infection, this is obviously a catastrophic complication. Whilst initial treatment would involve the use of appropriate antibiotics, the outcome is dismal and amputation may be the only sensible option.

Conclusions

It is probably fair to say that many simple metacarpal and phalangeal fractures are overtreated. It is, however, important that we pick out those with a potentially poor prognosis and focus most of our attention on this subgroup. Indeed, this group are probably better treated in a specialised hand unit. Burkhalter in 1989 [37] recommended stability enough to allow early motion rather than rigidity. It is also our opinion that the original AO/ASIF

aim of ´rigid fixation with plate and screws to obtain primary bone healing´ is not necessarily the gold standard in the hand. Previously surgery was performed with plates and screws that were clearly too big [38]. Whilst this, to a significant degree, has been addressed and there are now a number of bespoke fixation systems available for open reduction and internal fixation ORIF in the hand, surgery with all of its complications should still be reserved for the appropriate cases.

Prior to surgery, consideration should be given to the advantages and disadvantages of the different devices available. Surgeon preference and experience plays a significant role. Over the years we have used numerous devices, including staples, intraosseous wires, intramedullary devices (threaded screws, straight or prebent nails), as well as mini-external fixators.

In most cases, however, titanium plates and self-tapping screws appear to be the best option. Refinements with lower profile and locking screws, whose heads are flush with the plate surface, diminished the risk of adhesions and stiffness. Further stability is achieved with a supplementary lag screw fixation, whenever the obliquity of the fracture permits. With the smaller plate periosteum elevation is minimised, with a tendency for tendon adhesions diminished.

Malunion can lead to functional and aesthetic problems, but not every malunited fracture needs surgical treatment. It is function, not the radiographic appearance that determines whether treatment is necessary. Slight overlap of adjacent digits due to rotational malunion may be unsettling and unsightly, however it can be consistent with good hand function. Ill-advised treatment usually fails to improve function and sometimes make it worse. Unless a deformity is gross, it should usually be accepted when motion in the surrounding joints is satisfactory.

The surgeon (and the patient) should be aware that corrective osteotomy is not a panacea, especially with associated soft tissue injury. It is mandatory to tailor the treatment to the patient; complex operations never do well in the wrong patient [39]. Outcome correlates particularly with the degree of associated soft tissue injury (especially tendons) and patients (age, motivation), as well as the quality of the surgeon´s ability.

Complex procedures, especially in phalangeal non-unions, are worthwhile, only when the goals of intervention are well defined and are achievable. In all cases, postoperative hand therapy plays a pivotal role. A well trained hand therapist must be involved before and after surgery.

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Scaphoid Non Union

6

Hermann Krimmer

Keywords
Scaphoid nonunion • Vascularized bone graft • Herbert screw • Humpback deformity • Carpal collapse • Proximal pole • Avascular necrosis • Nonvascularized bone graft • Femoral condyle • Bony union

Introduction

Non-union of the scaphoid presents as a disabling condition affecting the wrist joint. Non-union occurs as the result of either failed treatment of an acute injury, or missed diagnosis. The risk of non-union increases the more proximal the fracture, as a result of the pattern of intraosseous blood flow of the scaphoid, as well as with displacement, particularly in cases of additional carpal instability [1, 2].

The goal of treatment is to achieve bony union, thus preventing the onset of secondary arthritic changes; so called SNAC wrist pattern [3]. The current most widely used surgical technique involves resection of the pseudarthrosis, bone grafting and rigid stabilization, using a headless bone screw pioneered by Herbert [4].

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Many factors influence the outcome, specifically the longer the delay from trauma to surgery, the worse the prognosis [5, 6]. As a consequence a diagnosis is made and if there is no evidence of arthritic change, surgery should be undertaken as soon as possible [7, 8]. The factors at this stage which affect the union rate include blood supply as well as the position of the fracture and stability.

Diagnosis

Commonly, the patient complains of pain on the radial side of his wrist, especially under load. Typically, pressure in the anatomical snuff-box will reproduce symptoms. Less commonly, pseudarthrosis presents without clinical symptoms and is a coincidental finding.

Diagnosis is confirmed by plane x-rays, specifically PA, lateral and ulnar deviated view (Stecher). A more detailed analysis will be provided by a CT scan, particularly views longitudinally to the axis of the scaphoid. Vascularity can be assessed by contrast enhanced MRI.

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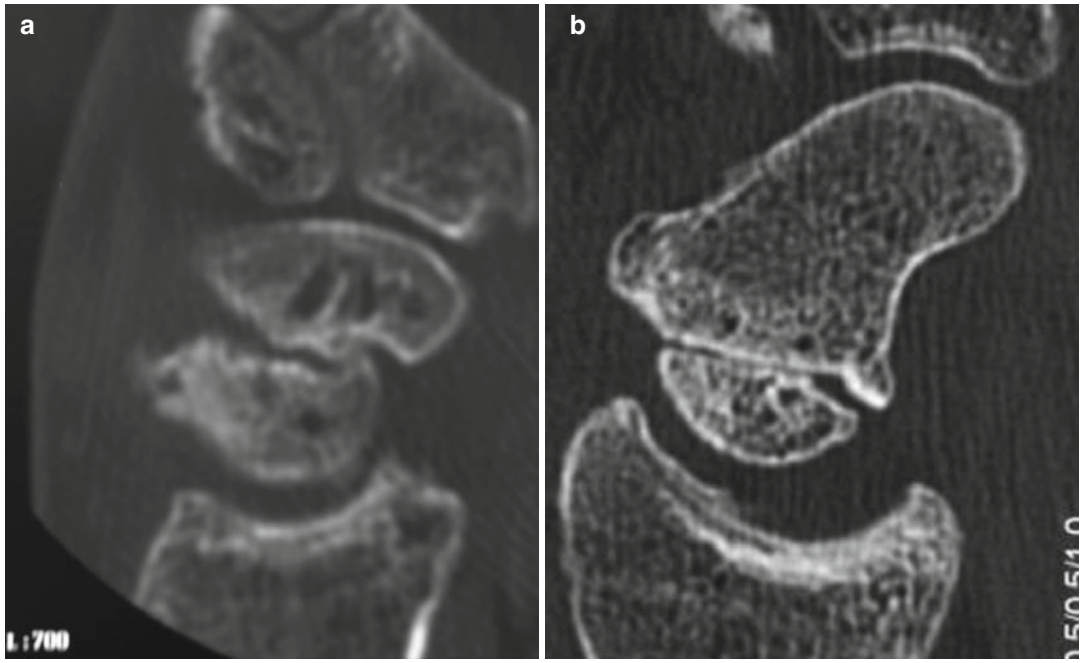

Fig. 6.1 (**a**) Humpack deformity of scaphoid non-union. (**b**) Nonunion of the proximal pole with preserved shape and length

Patterns of Non Union

Scaphoid non-union commonly occurs in the proximal or middle thirds of the scaphoid. Non-union through the waist of the scaphoid has an increased risk of displacement, leading to the "hump back" deformity with a collapsed, flexed and shortened scaphoid (Fig. 6.1a). In contrast, non-unions at the proximal pole usually do not lead to this type of deformity, although they do have a higher risk of avascular necrosis (AVN), due to the diminished blood supply (Fig. 6.1b). Stability, collapse and blood supply are major factors in influencing treatment strategy.

With respect to the morphology, in his original classification published in 1987 [4] Herbert differentiated, five types of non union, which were later modified and reduced to four [9].

D1 represents a fibrous non-union, where the shape of the scaphoid bone is maintained by strong connecting fibrous tissue. D2 means a complete non-union, with mobile fragments and significant resorption. In D3, sclerosis is seen at the non-union, with a defect leading to a hump back deformity. Finally in stage D4, necrotic bone, with loss of normal bony architecture, is observed (Fig. 6.2).

Assessment of Blood Supply

Intra-operative bleeding at the bone ending after the release of the tourniquet, is the most reliable indicator of vascularity, particularly of the proximal fragment [10]. Pre-operatively, however, the best imaging is provided by magnetic resonance (MRI). This has a high correlation with intra-operative findings, particularly if a contrast is used [11, 12].

A normal T1-weighted image represents viable bone. When there is loss of bone marrow signal in T1, blood flow is compromised (Fig. 6.3a). The use of gadolinium is mandatory to distinguish between avascular bone and marrow edema. T2 fat-saturated imaging may show an enhancement indicative of marrow edema (Fig. 6.3b) or no enhancement, which is synonymous with avascular bone (AVN, Fig. 6.4a, b). In a prospective

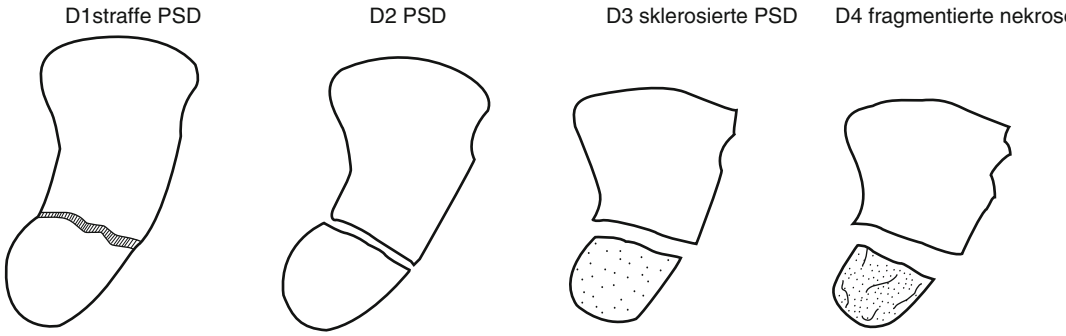

Fig. 6.2 Herbert's modified classification of scaphoid nonunion. *D1* fibrous non-union, *D2* mobile non-union, *D3* sclerotic proximal fragment, *D4* avascular necrosis with deformed and collapsed bone

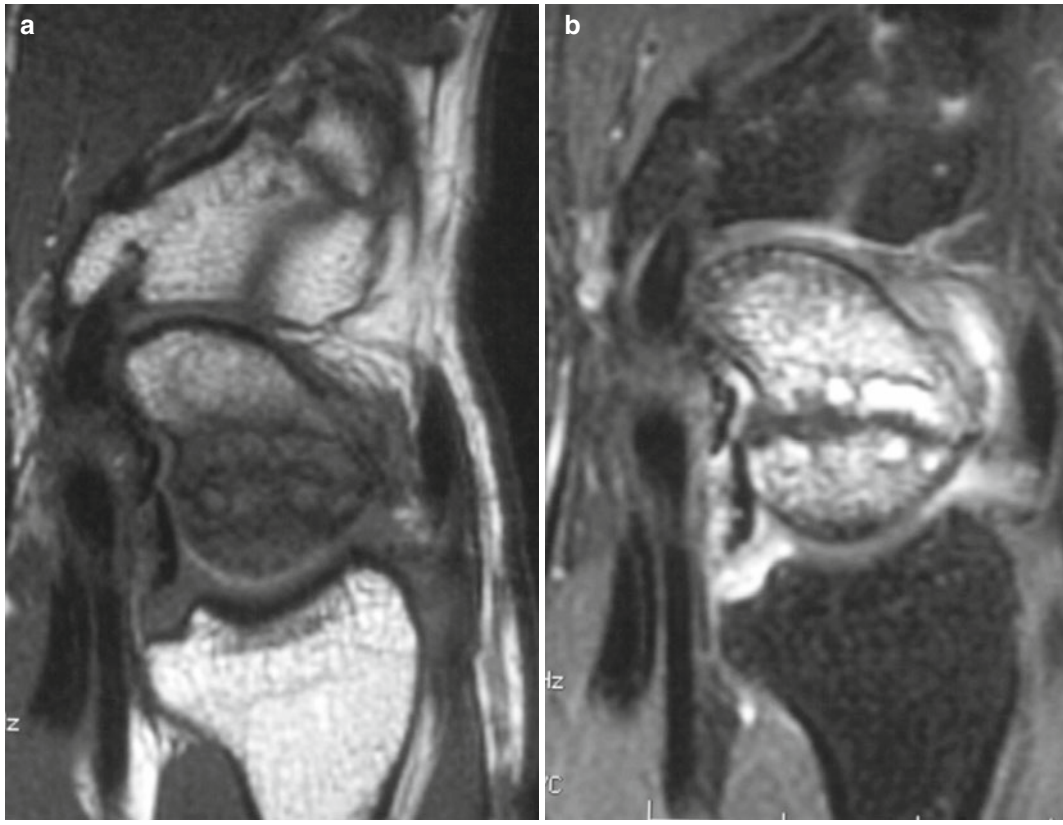

Fig. 6.3 (**a**) MRI with signal loss in T1 imaging (**b**) MRI with hyperenhancement by use of gadolinium in T2 fat saturated imaging as a sign of compromised blood supply

series, including 88 patients, the concurrence between MRI and intra-operative bleeding points was high with a rate of over 90 % [11].

Unfortunately, there is still some confusion over the interpretation of MRI findings. MRI can give information about blood supply, but not histology. This means that the diagnosis of true avascular necrosis (AVN) cannot be made by MRI alone, in effect, simply based on the loss of bone marrow signal during T1 imaging. MRI

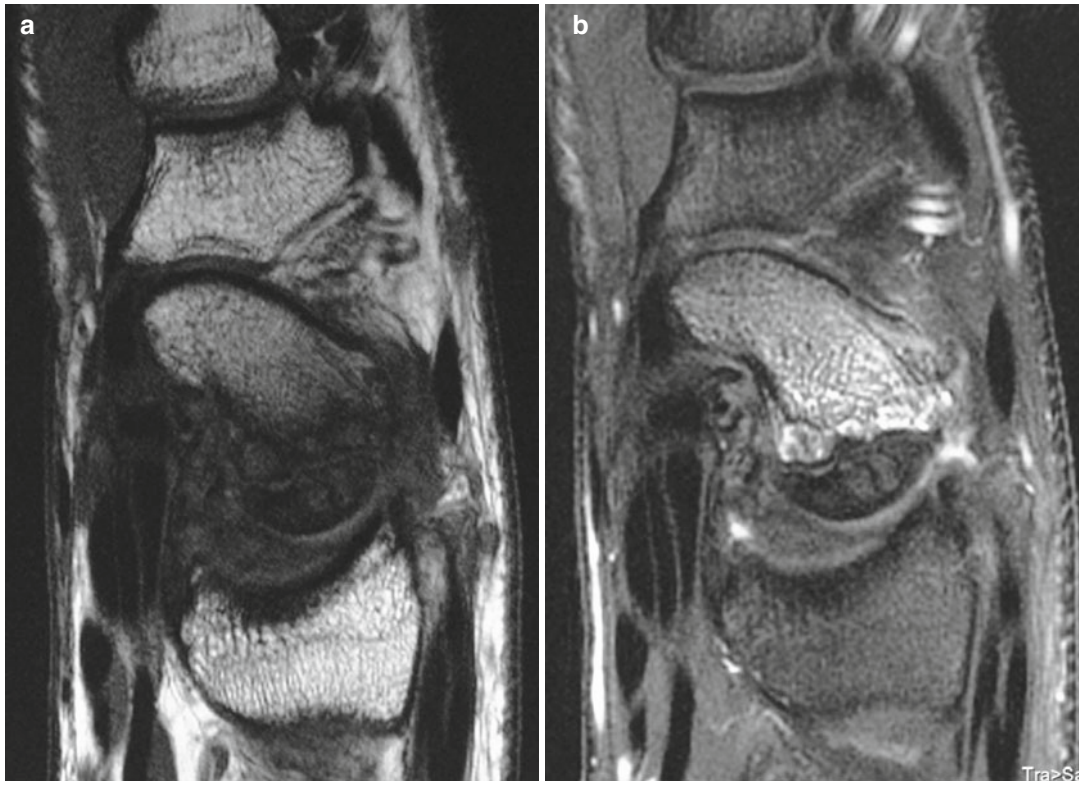

Fig. 6.4 (**a**) MRI with signal loss in T1 imaging (**b**) MRI with no contrast enhancement demonstrating avascular bone

scanning can, however, be used to distinguish between absent perfusion, which in effect means ischaemic bone, and disturbed blood supply with edema.

The precise definition of AVN is dead bone, with loss of the normal trabecular structure (Fig. 6.5). With this process, the bone tends to collapse and deform, in contrast to ischaemic bone, which appears dense on x-ray and sclerotic at the time of surgery. This dense bone does not collapse and can be stabilized by internal fixation, with the possibility of revascularization; this is not possible with true AVN. Most of the non-perfused scaphoid bones in cases of non-union still show some preserved bony architecture and, as such, are successfully amenable to surgery (Fig. 6.1). AVN, which means D4 according to the Herbert classification, also has destroyed bony architecture similar to that seen in Kienböck's disease. For this pattern, a true reconstructive solution does not currently exist, even with vascularized bone graft. The term AVN, as

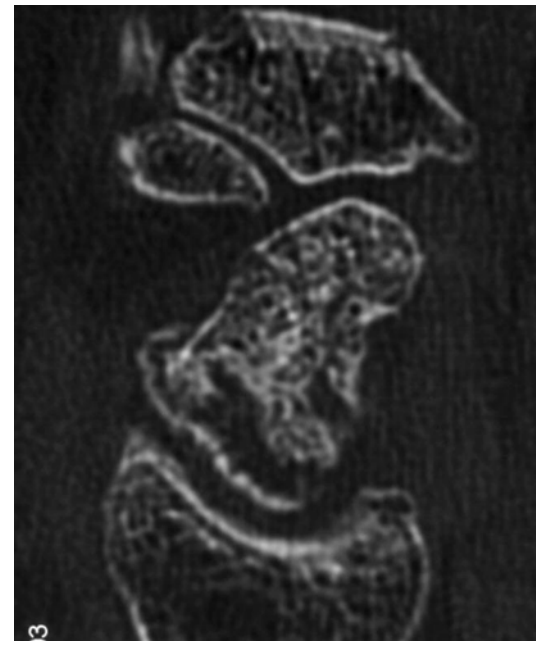

Fig. 6.5 True avascular necrosis with destroyed bony architecture

established in the literature, tends to mean an ischaemic proximal fragment with no contrast enhancement on MRI and the loss of bleeding points intra-operatively on release of the tourniquet.

With regard to Herbert's classification, D1 mostly represents viable bone in the proximal part, whereas D4 indicates avascular bone, as seen by contrast enhanced MRI. D2 and D3 mostly show compromised blood supply with edema or ischemia.

Variables of Non Union

At the level of the scaphoid waist we either see a stable non-union with preserved carpal alignment, where presumably the blood supply is intact, or the pattern of a "hump backed" deformity with carpal mal-alignment; here the proximal fragment either shows signs of compromised blood supply or even an avascular fragment. With non-unions of the proximal pole, we find a fibrous non-union with preserved blood supply, or complete non-union of compromised or avascular necrosis, with disturbed blood architecture. In these cases formal reconstruction is no longer possible. Other variables include failed previous surgery and secondary degenerative arthritis. In the latter, only those early cases, with arthritis limited to the radial styloid, are suitable for reconstruction. In the rest, generally a salvage procedure using either a four corner fusion, total wrist fusion or proximal row carpectomy is employed [13].

Principles of Treatment

It is not unreasonable to suggest that the different patterns of non-union need different surgical approaches. The ultimate goal of treatment, however, is to achieve bony union by resecting the pseudarthrosis until cancellous bone is seen, putting in a bone graft and restoring the original shape and length of the scaphoid and finally performing rigid stabilization. Stability and blood supply are the two most important factors for bone healing. Additional factors include smoking, age and delay between trauma and surgery.

With regard to a surgical approach, it is generally accepted that for the middle third a palmar approach is preferable and for the proximal third a dorsal approach. Rarely a "humped back" deformity is found in combination with a small proximal fragment. In these rare cases, a combined approach may be necessary [14]. With regard to the source of bone graft, generally a non-vascular graft taken from the iliac crest, distal radius or olecranon is satisfactory, as is a vascularized bone graft (VBG) pedicled on the distal radius. Finally, a free vascular bone graft taken from the medial femoral condyle (MFC) or the iliac crest can again be used. Obviously, however, these are more labour intensive.

Types and Technique of Non-vascularized Bone Graft

Fibrous non-union (D1), where the carpal alignment is preserved, can be treated by resecting the pseudarthrosis through a small window created in the scaphoid bone and inserting cancellous bone graft from either the **distal radius**, or alternatively, from the **olecranon**, with additional screw fixation through either a palmar approach for the mid-third and/or a dorsal approach for the proximal third (Fig. 6.5).

Conversely, D2 and D3 type non-unions require rigid solid autologous bone graft, preferably harvested from the **iliac crest**. This is particularly necessary in cases of "hump backed" deformity, where restoration of the length of the scaphoid by a large bone graft is required to allow stable fixation with a straight screw. If the "hump back" or flexion deformity persists, the screw can only fix through a small dorsal portion of the proximal fragment, resulting in less stability and a higher risk for failure. We prefer to harvest the graft from the iliac crest with a special reamer [15], as it is more convenient and less painful than conventional techniques and it provides a solid fragment of compressed cancellous bone; which can be trimmed to fit the defect perfectly

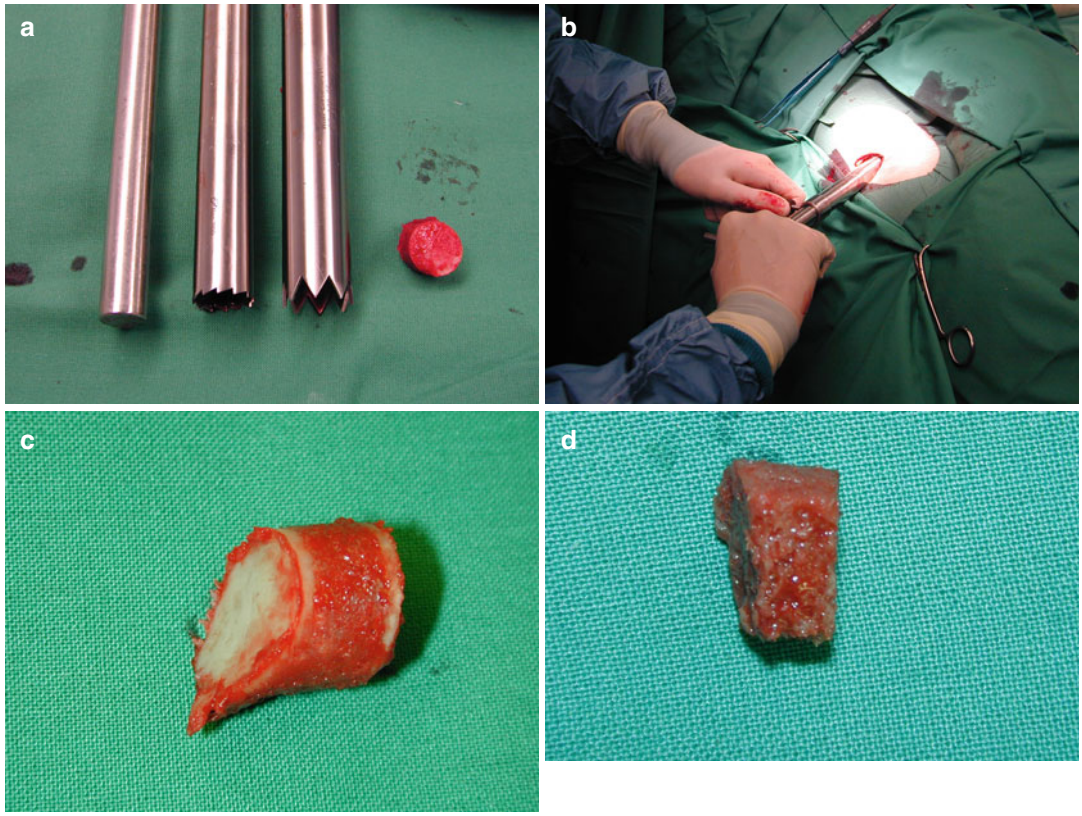

Fig. 6.6 (**a**) Reamer system for harvesting bone graft from the iliac crest (**b**) Intraoperative (**c**) Harvested corticocancellous graft (**d**) Trimmed bone graft

(Fig. 6.6). Added to this, we usually remove the cortical part, as this will accelerate bony union. Only when the graft does not seem stable would the anterior cortical part be left to support the restored scaphoid bone (Fig. 6.7).

In cases of longstanding "hump backed" deformity, where the distal fragment rotates palmarly, and the proximal fragment rotates dorsally with the lunate, it is usually helpful to initially correct the DISI deformity with a temporary K-wire, placed through the radius into the lunate with the lunate reduced. With this, mal-alignment is usually corrected and the proximal fragment is held in a stable position (Fig. 6.8).

Headless screws, which are mostly available in a cannulated, self tapping and self screwing mode, are the method of choice for fixation. For the small proximal fragment, mini types of these screws are preferred (Fig. 6.9c). They provide rigid fixation in contrast to the inlay graft technique of Russe, which was the previous method of choice and indeed requires a significant period of immobilization [10, 16, 17].

For failed surgery, where screw removal is necessary, revision will require a larger bone graft and possibly fixation with an external plate. This technique was used in the past by Ender, using a special condylar plate [18]. Nowadays, however, there are smaller, better shaped plates for this use. With this technique, again a higher degree of stability can be obtained (Fig. 6.10).

In cases of proximal pole non-union, where the proximal fragment is still vascularised, a dorsal approach, using a bone graft from the iliac crest and a mini headless screw, is recommended.

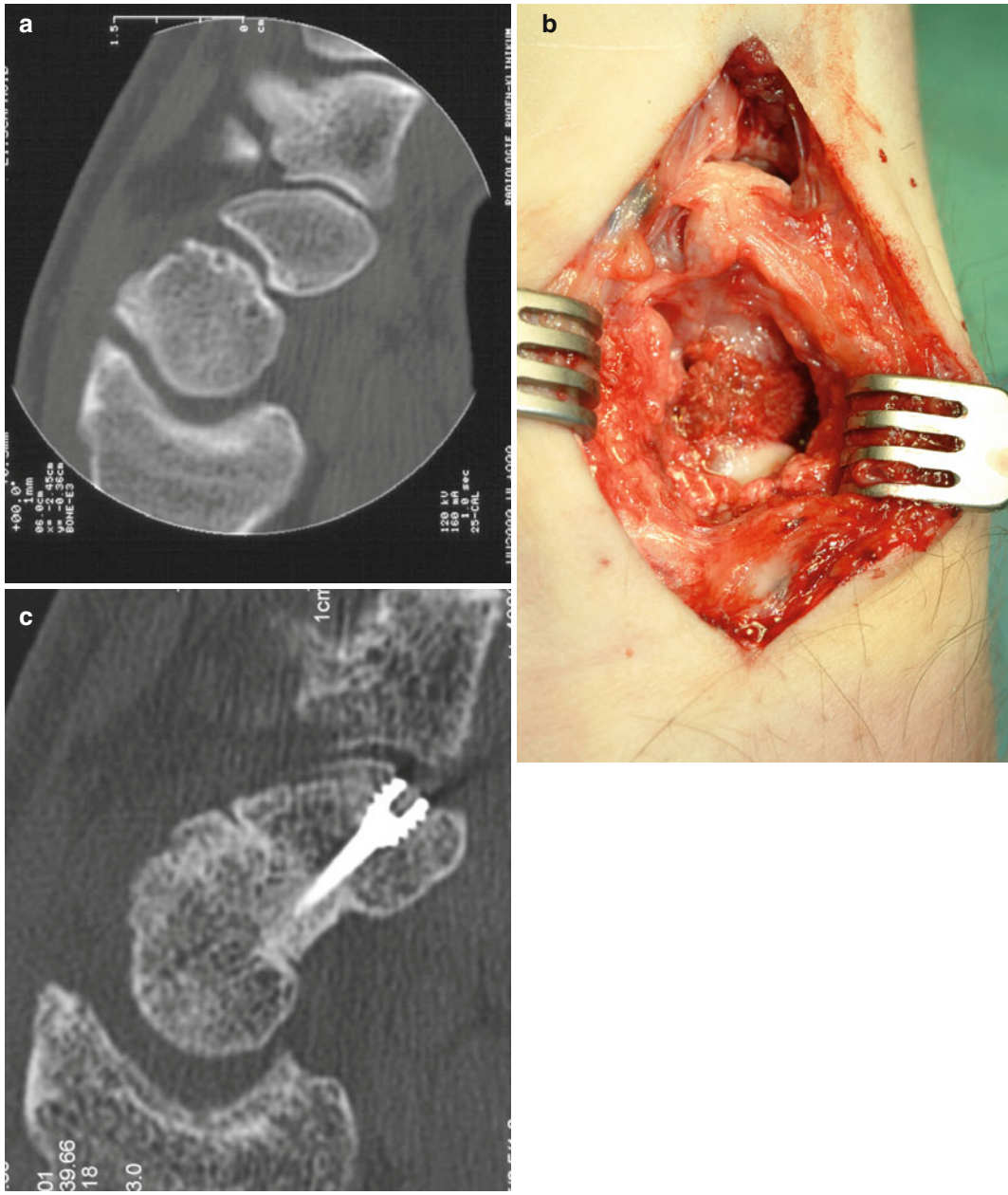

Fig. 6.7 (**a**) Scaphoid non-union with humpback deformity (**b**) Reconstruction with large bone graft (**c**) Confirmation of healing by CT scan

Clinical Pearl 1

Correction of the humpback deformity with stable bone graft represents the keystone for successful treatment of scaphoid non-union.

Types and Technique of Vascularized Bone Graft (VBG)

Vascularized bone grafts from the dorsal part of the distal radius have been popularized firstly by Zaidemberg [19] and subsequently refined by the

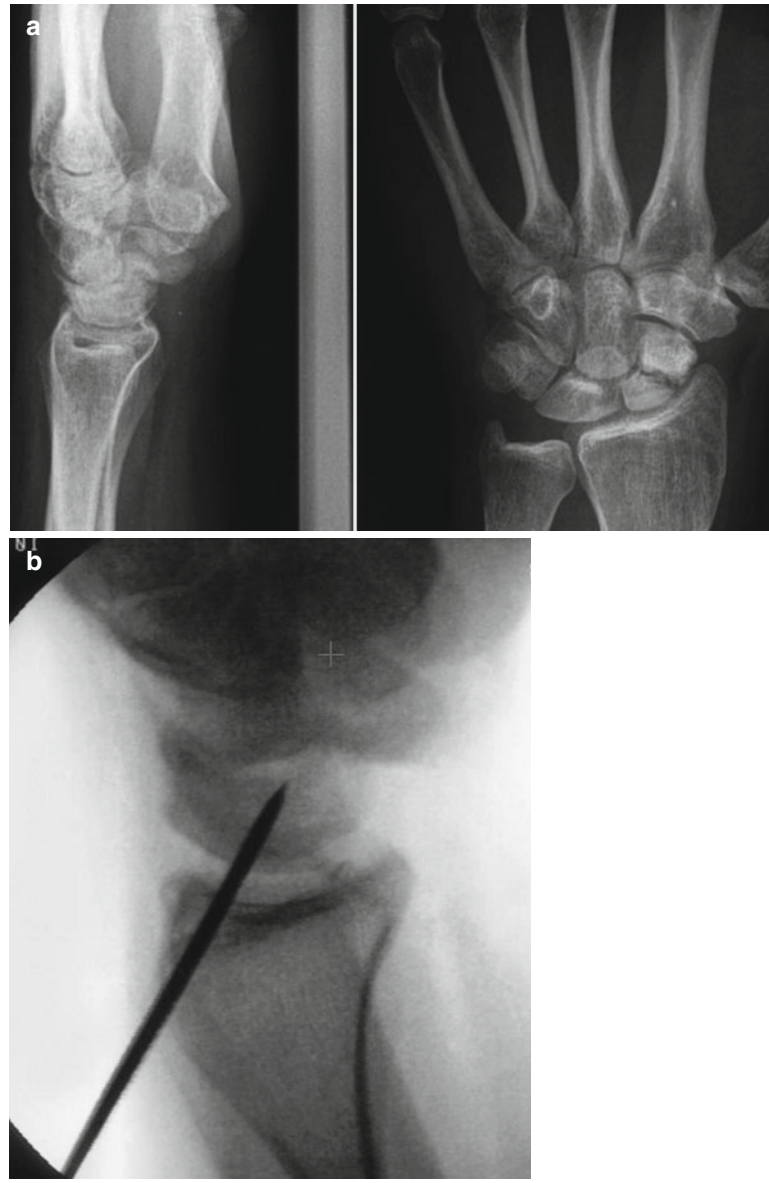

Fig. 6.8 (**a**) Scaphoid non-union with humpack and DISI deformity (**b**) Correction of DISI deformity by temporary K-wire

Mayo group [20, 21]. These are particularly for the small non-vascular proximal fragment. Sheetz et al. have extensively described the dorsal blood supply that provides an anatomic basis for these vascular grafts [22].

Our preferred procedure for proximal pole non-union (Fig. 6.9a–c) is a dorsal approach, harvesting the bone graft pedicle of the 1/2 supraretinacular intercompartmental artery (ICSRA). After identification of this vessel radially and ulnarly, the periosteum is incised in a proximal direction. The centre of the graft should be 1.5 cm proximal to the radiocarpal joint [22]. Once the graft is harvested, the pedicle is mobilized until it swings freely to the non-union site. The graft is then packed into the prepared non-union area and fixation is undertaken, again using a mini screw or, if this is not possible, by one or two K-wires. Correction of the "humped back" deformity, which requires the insertion of a volar wedge graft, is however difficult with this technique and as such it is probably best reserved for avascular proximal pole nonunion (Fig. 6.11).

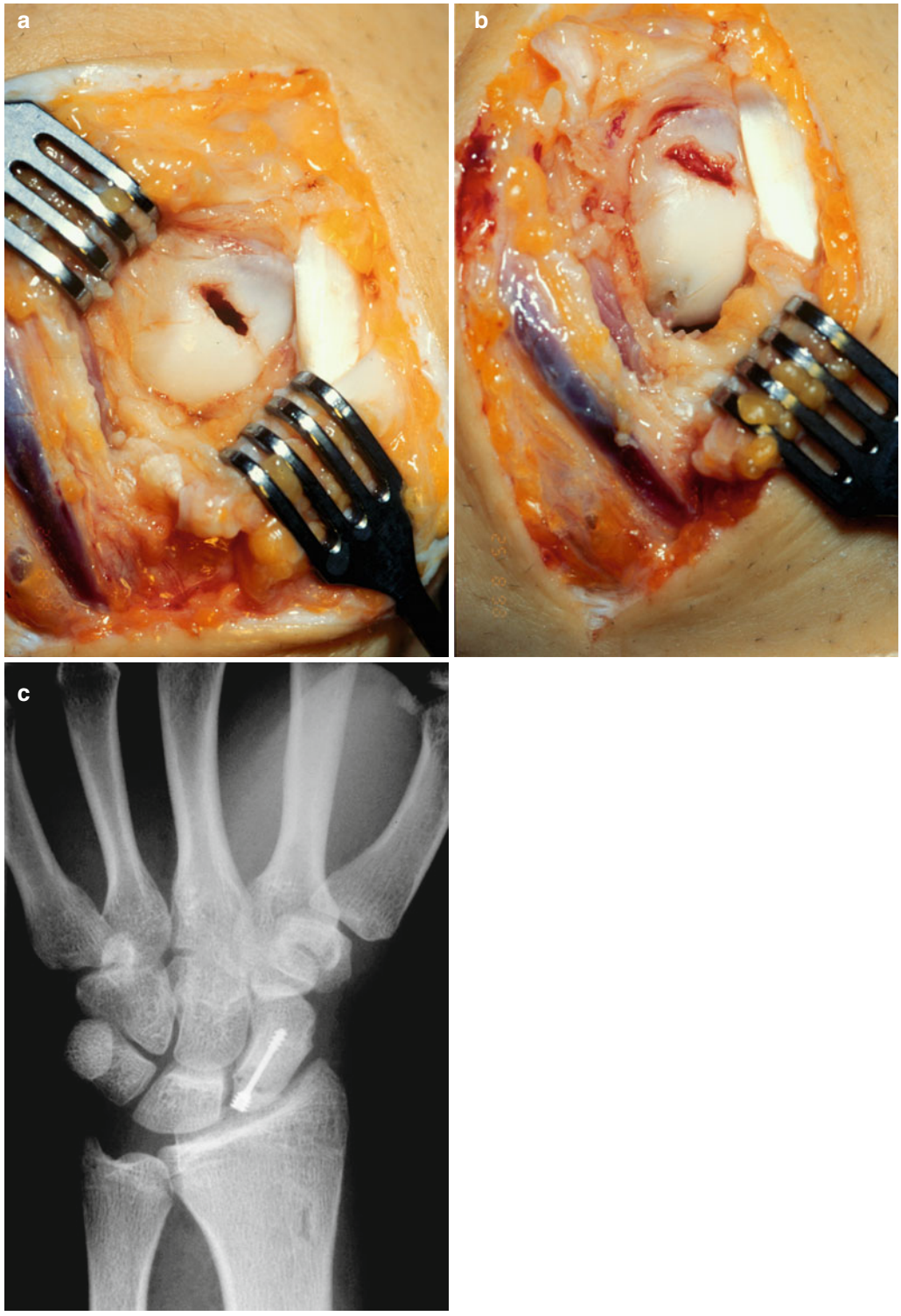

Fig. 6.9 (**a**) Proximal pole non-union D1 (**b**) Local bone graft from radius preserving the shape of the scaphoid and fixation by Mini Herbert screw (**c**) Postoperative radiograph

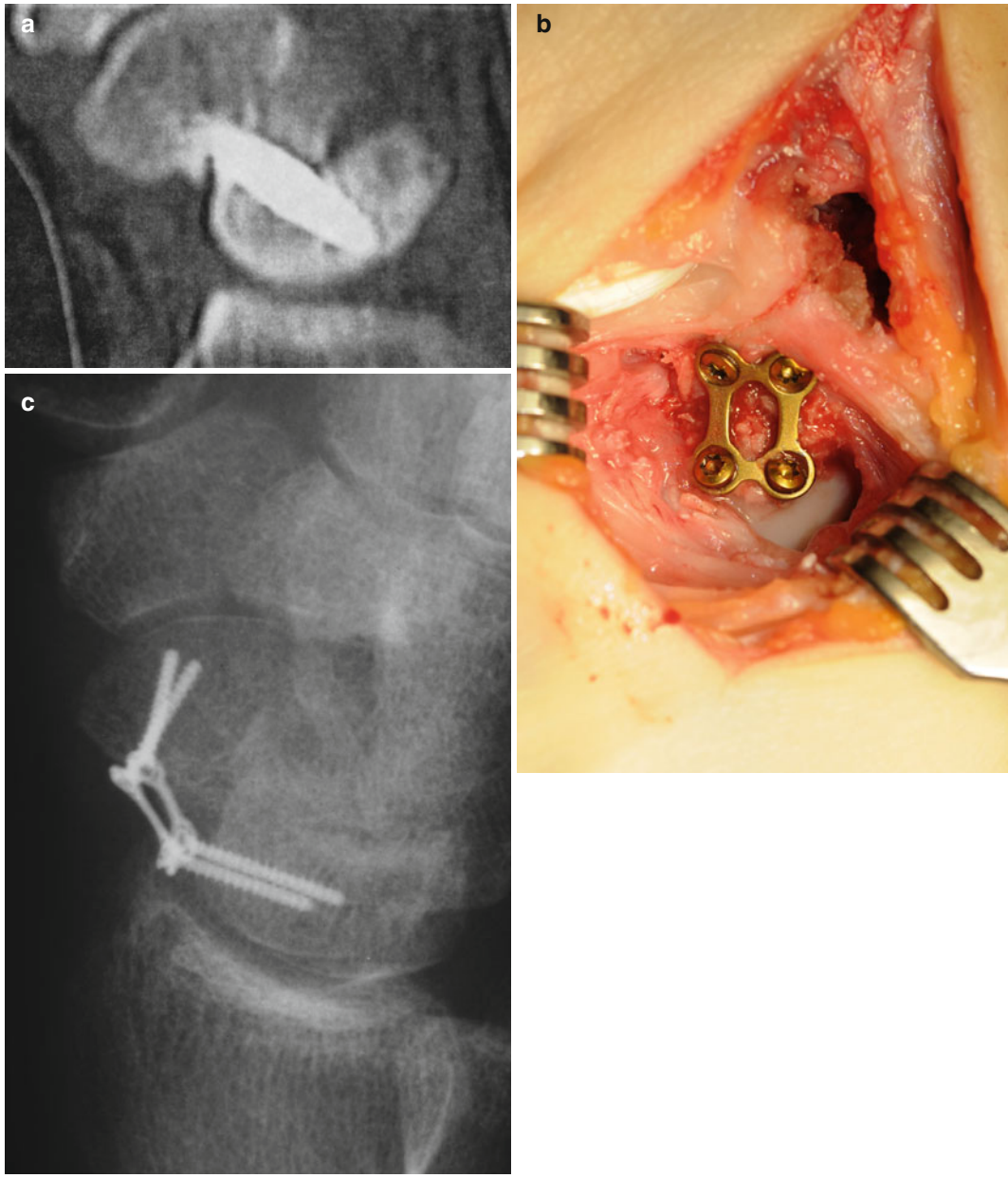

Fig. 6.10 (**a**) Nonunion with severe humpback after failed surgery (**b**) Revision surgery with large cancellous bone graft from the iliac crest and rigid fixation by a 2.0 mm grip plate (**c**) Bony union 3 months postop

An alternative for failed surgery with persistent non-union at the waist of the scaphoid, is a pedicled bone graft from the palmar surface of the distal radius as used by Mathoulin & Haerle [23]. The standard palmar approach to the scaphoid is used, with an extension proximally along the flexor carpi radialis. After identification of the radial artery, the distal margin of the pronator quadratus is prepared. The pedicle is a branch of the radial artery, which runs horizontally just distal to the pronator quadratus. The periosteum is then incised distally and proximally to the expected vessels. The bone graft is then harvested from the palmar-ulnar aspect of the radius, allowing enough length of the pedicle to reach the scaphoid non-union. Approximately 1 cm^3 of bone is harvested, using

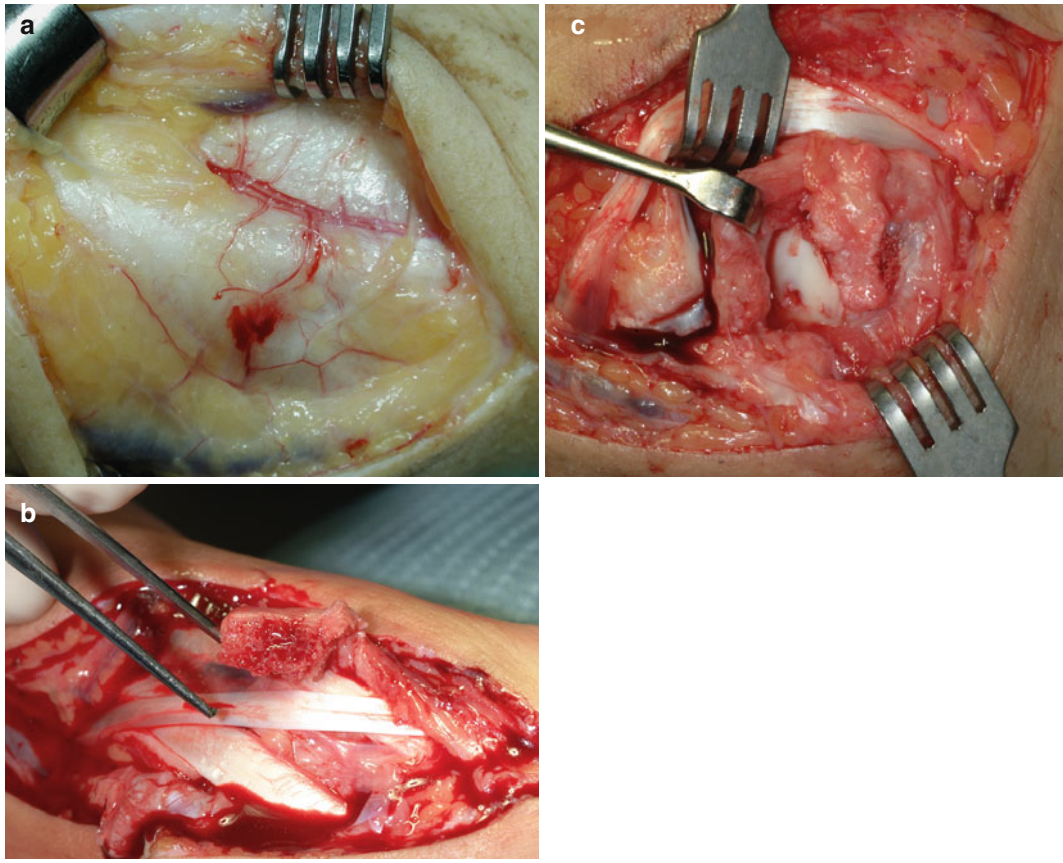

Fig. 6.11 (**a**) 1,2 intercompartimental (ICSRA) artery intraoperatively (**b**) Bleeding from the graft after releasing the tourniquet (**c**) Bone graft packed in the non-union side and fixed by mini Herbert screw

an osteotome on each side of the pedicle. Care is taken to avoid penetrating the distal radio ulnar joint. If there is concern, a temporary K-wire marking of the joint is often helpful (Fig. 6.6b). After harvest of the graft, the pedicle is mobilized and the graft is packed anteriorly in the prepared non-union area. If necessary, additional cancellous bone, from either the distal radius or iliac crest, can be inserted. Again, fixation is undertaken by a headless bone screw or alternatively two K-wires. Care is taken not to injure the pedicle (Fig. 6.12).

However, when there is severe carpal malalignment, VBG's from the distal radius are often unable to restore the scaphoid shape, as a consequence of the limited amount of bone that can be harvested and its poor structural rigidity. This often leads to poor results [24]. Alternatively, free VBG's either from the iliac crest or the medial femoral condyle can provide not only blood supply but also structural support from rigid cancellous bone, resulting in restoration of the scaphoid shape [25–27]. Free VBG's represent a consistent method, where both vascular bone graft and rigid stability are combined, with the possibility to harvest large grafts to correct any deformity and fill the defect in the scaphoid [28]. The surgical technique for free vascularized bone graft from the iliac crest can be difficult and, as a consequence, our preferred option is to take a VBG from the medial femoral condyle. The vascular anatomy at the medial femoral condyle is constant and does allow the harvesting of a bone graft with a long pedicle [29]. Fixation is undertaken using a headless screw and with an astomosis of the artery end to side to the radial artery and end to end to a concomitant vein [30]. This technique, however, is undoubtedly challenging surgically and time consuming and should be reserved for severe deformities and in the presence of avascular necrosis.

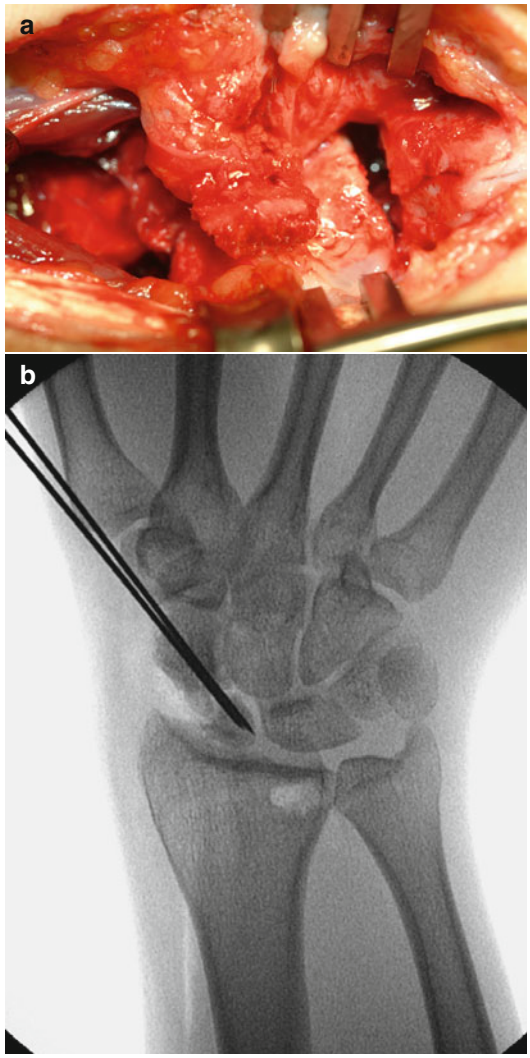

Fig. 6.12 (**a**) Pedicled bone graft harvested from the palmar aspect of the radius (**b**) Bone graft packed in the non-union side and fixed by two K-wires; note the donor side area

Clinical Pearl 2
Vascularised bone grafts should be reserved for the true avascular proximal fragment.

Postoperative Treatment

Whatever surgical technique for scaphoid non-union is implemented, immobilization in a short arm cast, leaving the IP joint of the thumb free, is instigated for at least 6 weeks. At that time, if x-rays show signs of bony union, mobilization including physiotherapy is begun. If not, the period of immobilization is prolonged and further x-rays and indeed a CT scan at 10–12 weeks are undertaken.

Clinical Results

Ramamurthy et al. [31] reported a cohort of 126 patients with scaphoid non-union treated by non-vascular bone graft and internal fixation. The overall union rate as seen on x-ray was 71 %. Adverse prognostic factors include the more proximal site of the non union, the longer time interval between the initial trauma and surgery and, to a lesser degree, the technique of internal fixation.

Other follow-up studies report a union rate for non-vascularised bone grafts in the absence of AVN at between 90 and 100 %. This drops down when AVN is present to less than 70 % [17, 32, 33].

Pedicled bone grafts from the distal radius, however, can achieve a better rate of union in cases of AVN; results have been reported at 80–100 % union rate [19, 20, 23, 34]. Unfortunately, however, patient selection is quite varied and often includes those with the poorest prognosis. Other groups have shown a significantly lower union rate of less than 50 % [35, 36]. The Mayo group, who initially popularised these techniques, reported in their first series union rates over 90 % [20]. Follow-up studies, however, had a union rate of only 34 of 50 patients, giving a 68 % success rate [37].

Finally, free vascularized bone grafts from the medial femoral condyle have reported to give a success rate of between 80 and 90 % and, as such, offer an attractive alternative for the most difficult cases [24].

Authors Preferred Method

At our institution, **non-unions at the waist** of the scaphoid are treated by conventional non –vascular bone grafting and internal fixation, as we

believe that stability is the major factor in obtaining union. This technique is used whether the proximal fragment is vascular or not. This technique restores the scaphoid to its appropriate length and shape, using a solid cancellous bone graft from the iliac crest, stabilized with a headless bone screw.

In revision surgery, where partial perfusion of the proximal fragment is present, we would again use bone grafting from the iliac crest with rigid fixation. At this time we would use a plate and screws. If, however, the proximal fragment is non-perfused, we would use a vascular bone graft. If there is little displacement then VBG from the distal radius would be appropriate. If, however, there is severe deformity, we would use a VBG from the medial femoral condyle.

Treatment of **proximal pole nonunion** again depends on the status of blood supply to the proximal pole, as assessed by MRI. If perfusion is present, then again standard bone grafting through a dorsal approach with fixation by a mini headless bone screw. If, however, no perfusion is seen a vascular bone graft again from the distal radius would be our preferred technique.

The advantage of vascularized bone grafts is undoubtedly their blood supply and, as such, an improved rate of healing. Disadvantages, however, include the wider dissection required, with additional scarring. VBG's taken from the distal radius are also unable to correct significant "humped back" deformities, due to the lack of rigid bone. Finally, in most if not all cases, fixation is undertaken by a headless bone screw. For these reasons in our practice, local vascularised bone grafts are normally reserved for non-unions of the proximal fragment.

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Distal Radius Malunion

7

Francisco del Piñal

Keywords
Distal radius malunion • Radial osteotomy • Wrist arthroscopy • Distal radius fractures • Intraarticular malunion

Introduction

Malunion is the most common complication following distal radius fractures and can cause considerable disability [1]. The malunion can affect the metaphysis (extra-articular malunion), the joint itself (intra-articular malunion) or both. Any of them may cause pain, restricted range of motion and arthrosis. Nevertheless, prognosis, urgency in surgical management and the impact of delaying the treatment are negatively tilted towards the intra-articular scenario. In this chapter the generalities regarding the pathomechanics of the extra-articular malunion will be discussed first, as it is quite common for an intra-articular fracture to have an extra-articular malunion component, which in turn may be responsible for part of the symptoms.

The surgeon should be warned that, although corrective osteotomy of the distal radius to reconstruct the natural anatomy is the foundation of treatment for this condition, malunion implies a major derangement of the wrist as a whole. In particular, most, if not all patients, will complain of decreased range of motion at the distal radio-ulnar joint (DRUJ) and/or ulnar sided pain. Many areas may need to be addressed and some of which may no longer have a good solution. Hence, before embarking on its management, a clear understanding of the aetiology of the patient's pain, limitations and the solutions planned, need to be discussed with the patient. Multiple problems (radio-carpal, ulno-carpal and distal radio-ulnar) may need to be addressed in the same operation, although they will be discussed separately for clarity.

Pathomechanics

When the radius loses its normal orientation in any plane, the load bearing area, the amount of load transmitted and the biomechanics of the radiocarpal, midcarpal and/or distal radio-ulnar

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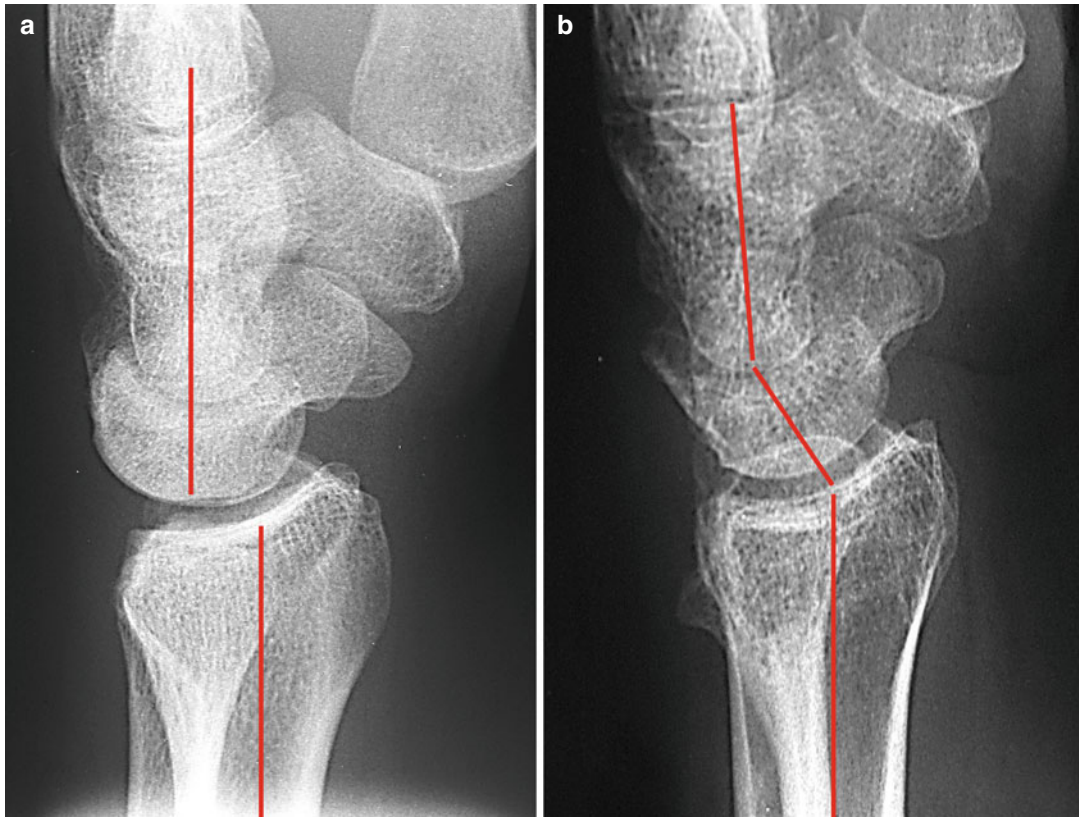


Fig. 7.1 The different patterns of carpal response to dorsal tilt. Both patients are young and have a similar degree of deformity. In (**a**) the carpus has subluxed as a whole dorsally. In (**b**) a zigzag collapse has occurred (Copyright Dr. Piñal 2011)

joint may suffer significant alterations. In a typical Colles' type fracture, the distal fragment will be dorsally angulated and shortened, often supinated and will lose some of the physiologic radial inclination. Conversely, in a volar type malunion (Smith type fracture) the distal radial fragment will flex and pronate. Although the malunion can take place in any of the planes, loss of the inclination of the radius in the sagittal plane and shortening has been proved to have the highest symptomatic and biomechanic impact.

Loss of inclination in the sagittal plane (the so-called physiologic volar tilt) has been demonstrated to significantly affect the carpal mechanics in different ways. In one group of patients the whole carpus may glide dorsally, whilst in others the lunate may stay in position and tilt. Dorsal slide (Fig. 7.1a) results in a dorsal shift in load bearing at the radiocarpal joint. In addition, it has been demonstrated that the load will concentrate on a smaller surface area [2–4]. As the cartilage there is not as thick as in the centre of the radius, the likelihood of increased wear and subsequent arthrosis is greater [2–5]. In the other group of patients, where the lunate stays in position, a carpal zigzag pattern occurs (Fig. 7.1b); painful synovitis due to the necessary hyperflexion of the distal row and abnormal load transmission may be the main complaint in less flexible individuals. Taleisnik and Watson [6] demonstrated that in the younger age (lax) group of patients a painful midcarpal instability would occur. As the lunate is already in hyperextension in the neutral position, further hyperextension will not be possible when the wrist is brought to ulnar deviation. The capitate will have to sublux out of the joint. Painful synovitis and a "clunk" on ulnar deviation will be the clues to diagnose this condition.

Park et al. [7] have further subdivided this DISI type pattern of "compensation" into two subtypes: A type I, in which the deformity is correctable after osteotomy and a type II (or fixed), in which the osteotomy will have no or minimal effect. It seems that the latter group may, in fact, be patients whom, in addition to having an extra-articular malunion, have an unrecognized scapholunate dissociation. Stiffening of the wrist ligaments in the older patient has also been implicated as responsible for the fixed nature of the type II. Prognosis after osteotomy for the type II group is considerably worse [8].

Loss of the volar tilt of the radius also has dramatic consequences on the ulno-carpal and distal radio-ulnar joint mechanics and load distribution. The load bone, by the head of the ulna, will increase by 50 % with a dorsal tilt of only 10° and by 67 % at 45° [2, 3]. Furthermore, the congruency and contact areas at the sigmoid fossa will be changed, thus creating a pre-arthrotic state and diminishing forearm rotation [9]. Besides this, Pogue et al. showed that shortening beyond 4 mm, 0° radial inclination, 30° of palmar inclination, or 15° of dorsal inclination could not be attainable unless the styloid were fractured, or the TFC were detached. This study gave biomechanical support to the frequent finding of DRUJ instability in the setting of malunions [10].

Symptoms on the ulnar side of the wrist and altered joint biomechanics will further worsen as a consequence of shortening of the radius that frequently accompanies radius malunion. The TFC will be locked as a result of shortening [3, 9, 11] and the load transmitted through the head of the ulna will be increased exponentially [3, 12]. In a series of experiments, Short et al. [12] demonstrated that only 2.5 mm of ulnar lengthening (radius shortening) increases the load borne by the head of the ulna from the normal 21% to as much as 42%, as the load through the radius decreases and concentrates dorsally.

Loss of radial inclination has a debatable effect. Radial inclination losses of up to 0° have minimal impact and, even then, only a slight decrease in grip strength has been reported. Malrotations in the axial plane [13], although responsible for lack of pronation-supination (particularly in Smith type malunions), are self-corrected at the time of surgery if a volar preformatted plate is used.

Intra-articular malunions have a much worse prognosis, due to the rapid development of secondary osteoarthrosis. Baratz et al. and Wagner et al. [14, 15] have demonstrated that step-offs at the radio-carpal joint cause abnormal concentrations at the edges of the step-offs (up to eight times the basal value) and abnormal motion of the corresponding carpal bone. Loss of cartilage will occur early in this setting and osteoarthritis will be the end result [16].

Indications

There is no universally accepted definition of distal radius extraarticular malunion to guide the treating surgeon as to when and how to treat. Acceptable limits of the three key radiographic parameters (volar tilt, radial inclination and ulnar variance) have not been even clarified. Although most authors use the contralateral wrist as a reference, Schuind et al. [17] reported that the normal wrist does not provide a better reference than normal values obtained from a database. Thus, Prommersberger et al. [18] defined the normal limits as within one standard deviation of the mean values of the general population: 8.5–15.5° for volar tilt, 21.5–26.5° for radial inclination and −2.5 to 0.5 mm for ulnar variance. To complicate things further, in the decision making process some patients (particularly older) might have severe deformities with minimal symptoms, while others (particularly young and active) with minimal deformities complain bitterly of pain or limitations. To summarize, in my view any patient who has lost any of the three key parameters of radius alignment and has pain and/or decreased range of motion and/or instability (at the radiocarpal, midcarpal or distal radio-ulnar joints) is a candidate for surgery provided he/she is fit for the operation. Prearthrotic conditions, such as dorsal tilt of more than 20° [2–5], is on itself an indication in a young-active individual.

Furthermore surgery is no longer contraindicated in older individuals with osteoporosis,

provided rigid volar locking plates are used. Previous reflex sympathetic dystrophy is also no longer a contra-indication, provided measures are taken to prevent recurrence at subsequent surgery. The involvement of a pain management consultant is strongly recommended.

As for the timing of surgery, there is general agreement that an intra-articular malunion should be undertaken as soon as possible, to avoid irreversible cartilage damage. In extra-articular malunions, some surgeons prefer to wait until tissue equilibrium has been reached and maximal function has been regained. Jupiter and Ring [19], in a comparative study, concluded that early surgery provided earlier return to work, less total disability time and less need for bone graft. The final functional results were equally good for both early or late osteotomy groups.

Surgical Techniques and Rehabilitation

Procedures to the radius, the ulna or both may be needed to treat distal radius malunion. To avoid confusion however, in this chapter they will be considered individually, although the surgeon should keep in mind the number of procedures that may have to be undertaken during the operation. Except in cases where there is no (or minimal) radial deformity (where an ulnar procedure is all that it is required) the operation always follows three steps;

1. a radial osteotomy,
2. an ulnar sided procedure to address impaction,
3. re-exploration of the ulnar side (including an arthroscopy!) to assess and treat DRUJ instability.

Dorsal Angulated Malunion

Simply speaking there are two ways of repositioning the dorsally tilted radius: a dorsal opening wedge, or a volar closing wedge. In the latter, further shortening of the radius obliges one to undertake a concomitant ulnar procedure to restore normal ulna variance. It is also important to remember that, whilst by definition all fractures in this group have the distal radial articular surface tilted dorsally, not all Colles's type malunions will have concomitant radius shortening. True shortening will occur only when there has been volar metaphyseal comminution, or when the continuity of the volar cortex has been lost, due to dorsal translation of the distal fragment. In many of the dorsally tilted malunions however, the volar cortex acts as a hinge point at the fracture line and the distal fragment rotates, but does not translate. This occurs if there is only dorsal comminution: the distal fragment, unsupported dorsally, pivots on the volar cortex, slowly rotating in the cast [20], or when the reduction was insufficient from the beginning. This represents what we have named as a "sagittal rotational malunion", where there is no shortening despite the appearance on a posterior–anterior radiographic view (Fig. 7.2) [21].

Such a sagittal rotational malunion can, however, be recognised on the lateral radiograph, by the "preservation" of the volar cortex and by having the anterior rim of the radius longer (more distal) than the head of the ulna, confirming the absence of shortening (Fig. 7.3a). This distinction is important with regard to management; a pure sagittal malrotation will respond to a much simpler de-rotational osteotomy. Whilst if there has been shortening it will need a more complex tri-dimensional reconstruction or a Wada procedure (see below).

When true shortening exists, the Fernandez's method [8] of preoperative planning using tracing paper on the deformed side and transporting it to the healthy side in order to give a three-dimensional model of the bone graft is recommended (Fig. 7.4). Although Fernandez's method is somewhat inexact as compared to computer-generated models [22, 23], it is of an enormous help at the time of surgery and technique accesible to all.

Opening Wedge Osteotomy- Volar Approach

Whether there is pure malrotation in the sagittal plane, or a combination with shortening or loss of

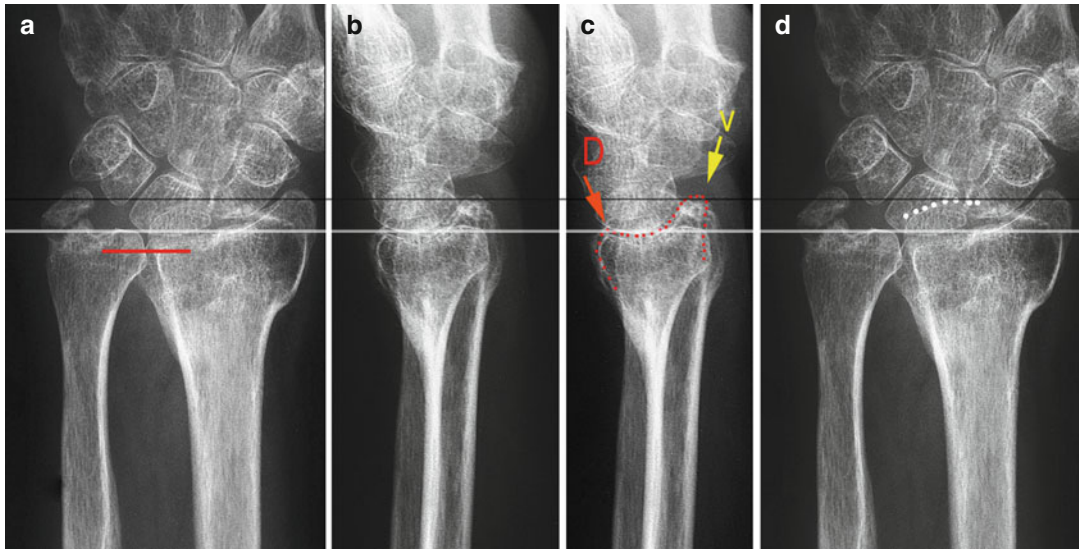


Fig. 7.2 Malunited distal radius fracture with true dorsal tilting and apparent shortening of the radius. (**a**) The sclerotic rim appears to show a positive ulnar variance, and the dorsal tilt of the distal radius (**b**). (**c**) The contour of the distal radius has been highlighted by *dots*, it can be seen that volar rim of the radius (*V*) is actually distal to the head of the ulna, although the dorsal rim (*D*) is proximal. (**d**) On the same P–A view as (**a**), the volar lip (with *dots*) is clearly distal to the head of the ulna confirming the inaccuracy of the variance measured in (**a**). A fine *grey line* has been drawn tangentially to the ulnar dome across all the radiograms. A fine *black line* marks the volar rim of the radius (From del Piñal et al. [21] with permission)

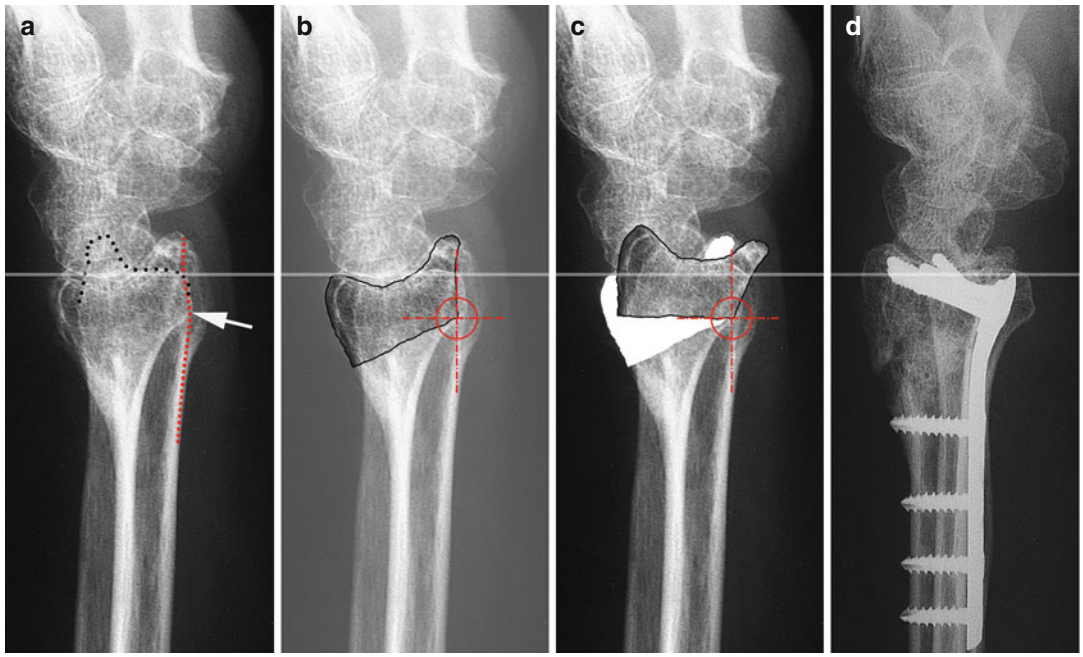


Fig. 7.3 Pure sagittal malrotation: diagnostic pointers, planning and execution (same case as in Fig. 7.1). (**a**) A clear hinge point corresponding to the original fracture line can be seen (*arrow*), attesting to preservation of the volar cortex length. The distal volar rim can be seen distal to the head of the ulna confirming the ulna minus variance (*stippled in black*). (**b**) The hinge point will be used as the rotation point of the distal fragment. (**c**) Pure rotation on the fulcrum will correct dorsal tilting preserving the radial length. (**d**) The result on this patient. A fine *grey line* has been drawn tangentially to the ulnar dome across all the radiographs. A fine *black line* marks the volar rim of the radius (From del Piñal et al. [21] with permission)

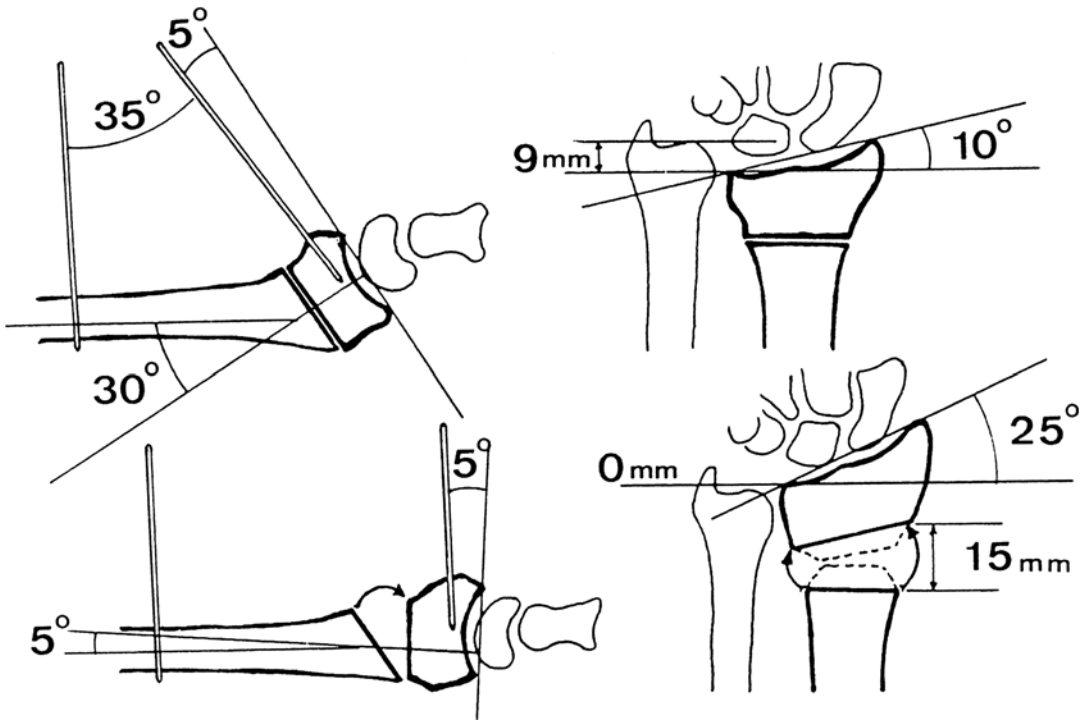


Fig. 7.4 Planning of the correction as advocated by Fernández. *Top left* demonstrates the K-wire placement to intraoperatively reference the correction of the dorsal tilt. *Bottom left* view shows the sagittal correction after the osteotomy with resultant dorsal bone gap. *Top right*, precorrection loss of radial inclination and radial shortening. *Bottom right*, postcorrection alignment demonstrates that the cortex is opened more on the dorsal radial than the dorsal ulnar side (In Ref. [8] with permission from the *Journal of Bone and Joint Surgery*)

radial tilt, I always use a volar approach as initially described by Lanz [24]. The time honoured dorsal approach, championed by Diego Fernández (osteotomy, cortico-cancellous interpositional bone graft and dorsal plating) [8], although compared to the former, is an easier technique, has the major drawback of requiring a plate to be applied on the dorsal surface. The latter runs an increased risk of extensor tendon damage. In addition, a second operation for plate removal is more likely.

Conversely, plating volarly is more technically demanding, as it allows no alterations once the plate is fixed distally. On the other hand, it rarely requires plate removal, as tendinitis is unusual (provided the appropriate length of screws is used). The initial problems with Lanz's technique, related to the implant size and the lack of stability of the construct when length was also corrected, have been overcome with modern fixed-angle plates. As a matter of fact, the exact application of these plates to the malunited epiphysis will automatically correct any malunion. Furthermore, the stability provided by the plate allows immediate rehabilitation, without the need of structural bone graft [25]. Last but by no means least, placement of the plate volarly will spontaneously correct any malrotation of the distal fragment in the axial plane.

Again when planning the procedure we would recommend a technique described by Fernandez [8]. Measurements are taken from the lateral radiographs. The key point is to fix the distal component of the plate, forming an angle ("correction" angle) with the diaphysis equal to the preoperative measured dorsiflexion plus 5°, in order to achieve some physiologic palmar tilt (Fig. 7.5).

The operation is performed under axillary block, on an outpatient basis. The arm was exsanguinated and a tourniquet applied. Bone

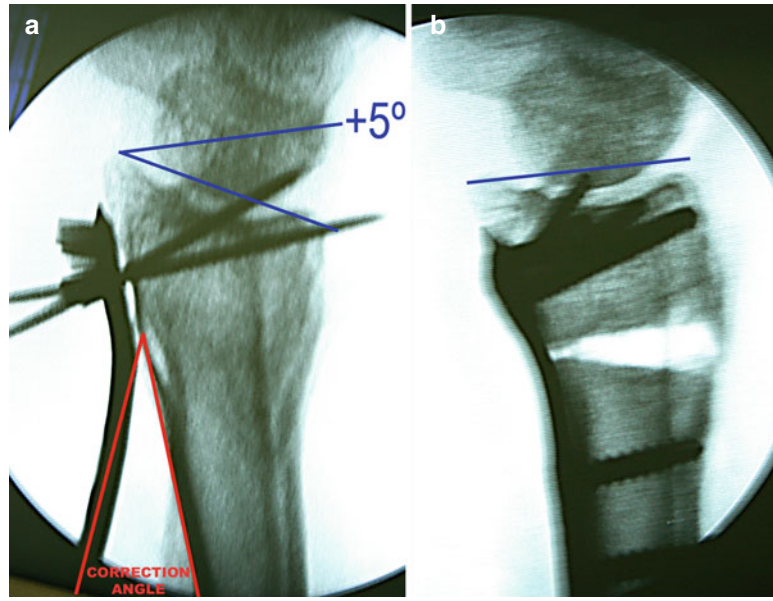


Fig. 7.5 (**a**) Correct placement of the plate can be assured intraoperatively by fluoroscopy when the stem of the plate creates an angle with the diaphysis of the same amount as (plus 5º) the dorsal tilt plus 5° (to restore the volar tilt). (**b**) In this setting "automatic" correction of the deformity will occur (Copyright Dr. Piñal 2011)

from the olecranon is harvested through a 2.5 cm transverse incision. The cavity is then filled with Surgicel® and the wound closed in a single layer, using a 3/0 subcuticular nylon.

Access to the malunion site is through a 6–8 cm incision, radial to the flexor carpi radialis sheath (FCR), with a 10 mm radially directed back cut at the proximal wrist crease. By dissecting with a knife on the radial aspect of the FCR sheath, the sheath can usually be preserved intact, but, more importantly, the radial artery will stay safely lateral. The space between the FCR and radial vessels is developed. A large constant branch from the radial vessels to the radial aspect of the pronator quadratus should be identified and coagulated. This muscle is then sharply elevated subperiostically and reflected ulnarly. Proximally, some fibres of the flexor pollicis longus are reflected ulnarly. This will expose the malunion site, the distal epiphysis and the radial shaft. Dissection now proceeds on the dorsum of the radius, going superficial to the brachioradialis, which is left undisturbed, except in cases of major radius shortening. The whole layer of thickened-scarred periosteum is elevated on block and then divided by making several transverse cuts, proximal to the extensor tendon compartments, until the tendons themselves are exposed. Unless this is done, proper reduction of the epiphyseal fragment would not be attainable, as this scarred tissue acts as a check-rein to volar tilting of the distal fragment.

At this moment the fixation plate is applied volarly, prior to the osteotomy, as recommended by Lanz [18, 24]. The transverse part of the plate is placed as distally as possible and always distal to the hinge point of the malunion. Guide K-wires are invaluable to ascertain that the distal pegs will be subchondral, as this will guarantee a strong hold. If the surgeon is satisfied with the fluoroscopy image, all the distal screws and pegs are inserted. As mentioned previously, at the end of this part of the operation, the plate should form an angle with the radial shaft (correction angle) equivalent to the amendment needed in the sagittal plane. To avoid the loss of any volar cortical bone by the cut of the saw, the osteotomy is performed using a 1 mm diameter K-wire. A series of perforations parallel to the articular surface, to the dorsal cortex, are made along the exact hinge point of the malunion (Fig. 7.6). Once the osteotomy is completed, the surgeon applies force dorsally to the distal-radius and plate to produce an osteoclasis of the weakened volar cortex. Quite frequently several attempts are needed and in some cases an oscillating saw is required to cut the very sturdy medial (ulnar) cortex. The distal-radius/plate block is then reduced to the shaft of the radius, by pushing volarly with the fingers on the distal

fragment, as in a closed Colles' reduction. The use of a lamina spreader to distract the collapsed dorsal space greatly helps achieve reduction. Other manoeuvres, such as the use of bone clamps to bring the stem of the plate to the shaft of the radius, are to be avoided as this may cause the screws to be pulled out of the bone. Once the plate adapts to the volar shaft cortex without undue force, it is held temporarily by two bone clamps. It is now critical to perfectly reduce the volar cortex, as otherwise shortening and incomplete volar tilt correction will ensue. Once this has been checked radiologically the rest of the screws are inserted and a bone graft applied. Thereafter pronator quadratus is closed, if possible, over the plate, by preplacing three resorbable stitches and tightening them in pronation. The skin is closed with subcuticular 3/0 nylon. In every case a standard exploratory dry arthroscopy follows the osteotomy [26]. Synovectomy, soft tissue procedures and/or ulnar styloid excision is undertaken as required (Fig. 7.7) (see "Ulnar side procedures" later).

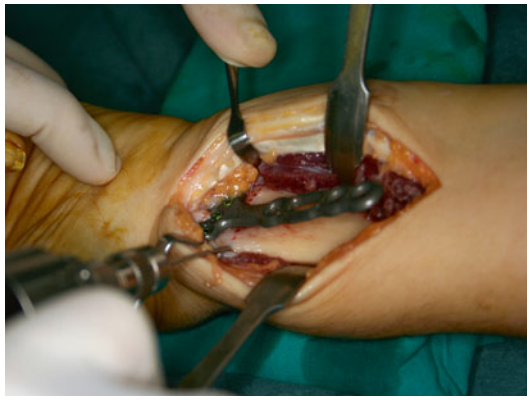


Fig. 7.6 The osteotomy is being performed with a 1 mm k-wire. Notice that the plate stands out of the diaphysis (correction angle) (From del Piñal et al. [21] with permission)

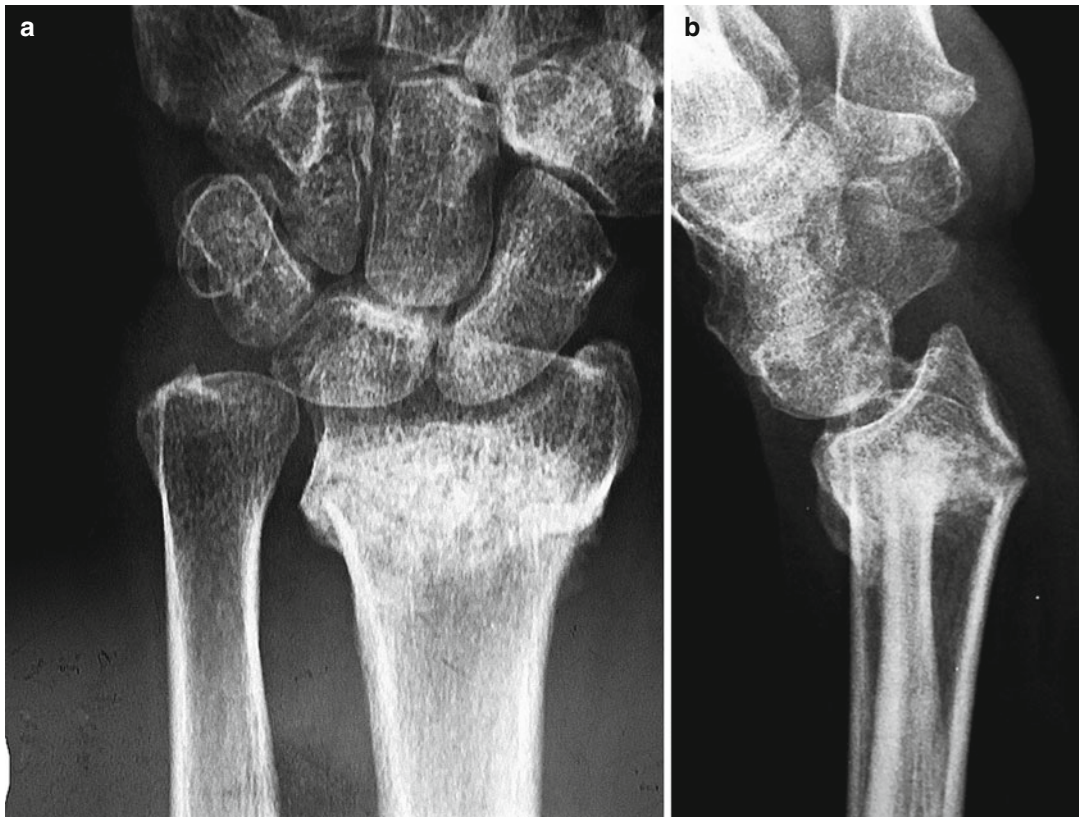
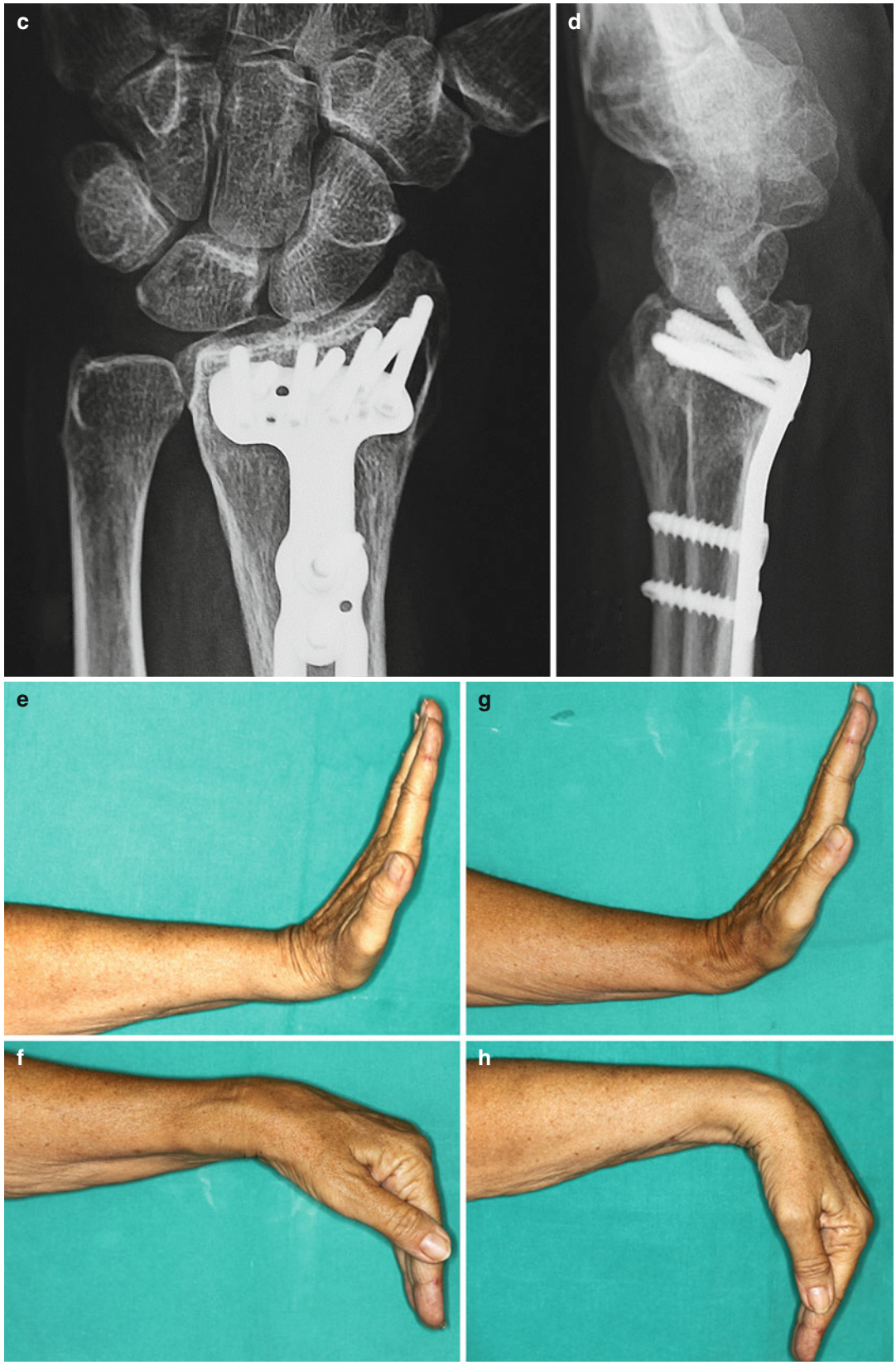


Fig. 7.7 (**a**, **b**) Despite the severity of the deformity and the apparent shortening of the radius the patient has a pure malrotation in the sagittal plane, without any shortening of the radius: notice the hinge point and the position of the volar rim distal to the head of the ulna in the lateral. (**c**, **d**) Pure derotational osteotomy fully corrects the deformity. (**e**, **f**) Lack of flexion is the most prominent complaint in most Colles' type malunion. (**g**–**j**) Final range of motion. Notice the improvement in flexion (Copyright Dr. Piñal 2011)



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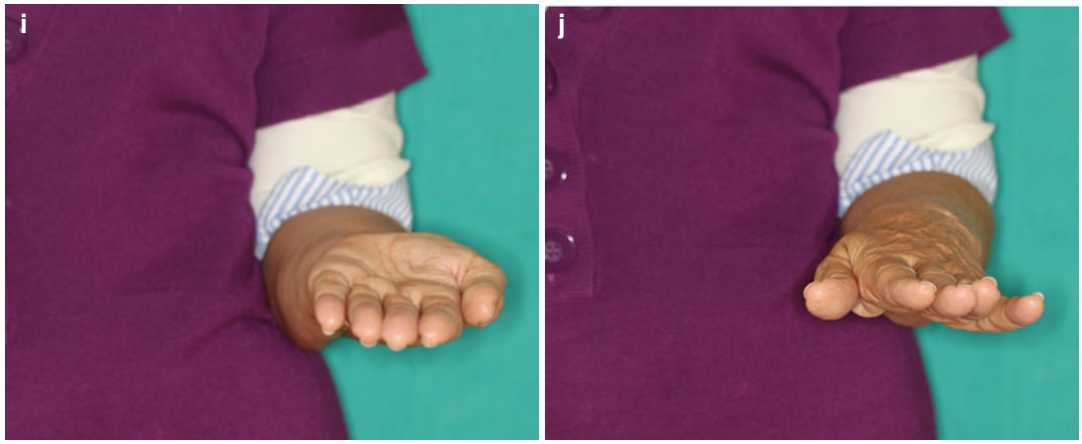


Fig. 7.7 (continued)

A bandage and volar splint are applied and then the tourniquet is released, maintaining some pressure over the surgical area for 5 min for haemostasis. At the first preoperative visit, 24–48 h later, a removable splint is applied, encouraging self-directed active range of motion exercises. After the 4th-6th week patients are weaned off the splint. No formal therapy is prescribed. Protection of the elbow for 4–6 weeks with skateboard splints is also recommended.

In cases of true concomitant shortening (and angulation) the technique is slightly modified or Wada's operation (see next section) is undertaken. The dorsal tilt is first corrected, with the plate inserted distally and then reduced to the shaft of the radius. With the help of a laminar spreader at the osteotomy site, the radius is distracted as required. Then, the screws are inserted into the longitudinal stem of the plate, to lock it. It is important, when shortening exists, to step-cut the brachioradialis as otherwise it will limit correction. The tails of the Z-plastied tendon are sutured at the end of the operation. The appropriate graft size is interposed; the graft itself, typically being cortico-cancellous, can be taken from the iliac crest. Modern plates allow the insertion of cancellous bone graft or even bone substitutes. It is, however, quite difficult to correct more than 10–15 mm of radial shortening by an open wedge procedure as proposed previously [8, 27] (see next section). Finally, severe shortening of the radius, more than 3 cm, requires distraction of the radius.

Closing Wedge Osteotomy (Wada's Procedure)

To avoid the need for bone graft and the risk of non-union, Posner and Ambrose [28] proposed a volar closing wedge osteotomy of the radius, combined with a Darrach procedure to address the distal radio-ulnar joint. The operation is less involved for the patient, as no bone graft is required and for fixation, the better-tolerated volar plate can be used. Despite good results reported at 5 years in a relatively young patient group (40-y-o), most hand surgeons would consider sacrificing a healthy/preservable distal radio-ulnar joint as a disservice to the patient. By the same token, however, when the DRUJ is irreversibly damaged, there is not much point in restoring the radial length. In these cases a Posner-Ambrose procedure, or a closing wedge radial osteotomy combined with a Sauvé-Kapandji, or an ulnar head replacement, are the options of choice.

Wada et al. in 2004 [29] popularized the concept of combining a closing wedge osteotomy of the radius and an open ulnar shortening, rather than a destructive procedure on the ulnar side. Initially they recommend it for the elderly or less demanding patients [29]. However, due to their good results they have now extended their indications to all ages [30].

The operation is again planned with the help of tracing paper on radiograms, in a very similar way as advised by Fernandez for the open wedge

osteotomy. The wedge to be removed from the volar radius is calculated from the lateral x-ray. Whilst the amount of ulna to be shortened is the sum of the preoperative ulnar variance, plus the amount of radius that will be removed from the volar cortex (Fig. 7.8). Although simple on tracing paper, in practice the operation is quite tricky; specifically minimal variations of the cutting angle on the volar surface of the radius with an incorrect sized wedge being removed. This, in turn, will affect the accuracy of the amount of radius removed. Intra-operative adjustments of the amount of ulna to be shortened must be made by fluoroscopy. To further complicate matters, the radius cannot be reduced until the ulna has also been cut. At one stage therefore, both bones have been divided and no fixation is in place. We have overcome this by stabilizing the distal radius fragment prior to the osteotomy, by preplacing the plate distally in the similar manner as advised by Lanz.

Despite these criticisms, Wada et al. [30] have shown a superior outcome in the closing-wedge osteotomy group as compared with the open-wedge group, with the added advantage of not requiring bone graft. On the other hand, an open wedge osteotomy allows for lengthening of the radius, without the need of an ulnar osteotomy. Ulnar shortening is not always a benign procedure.

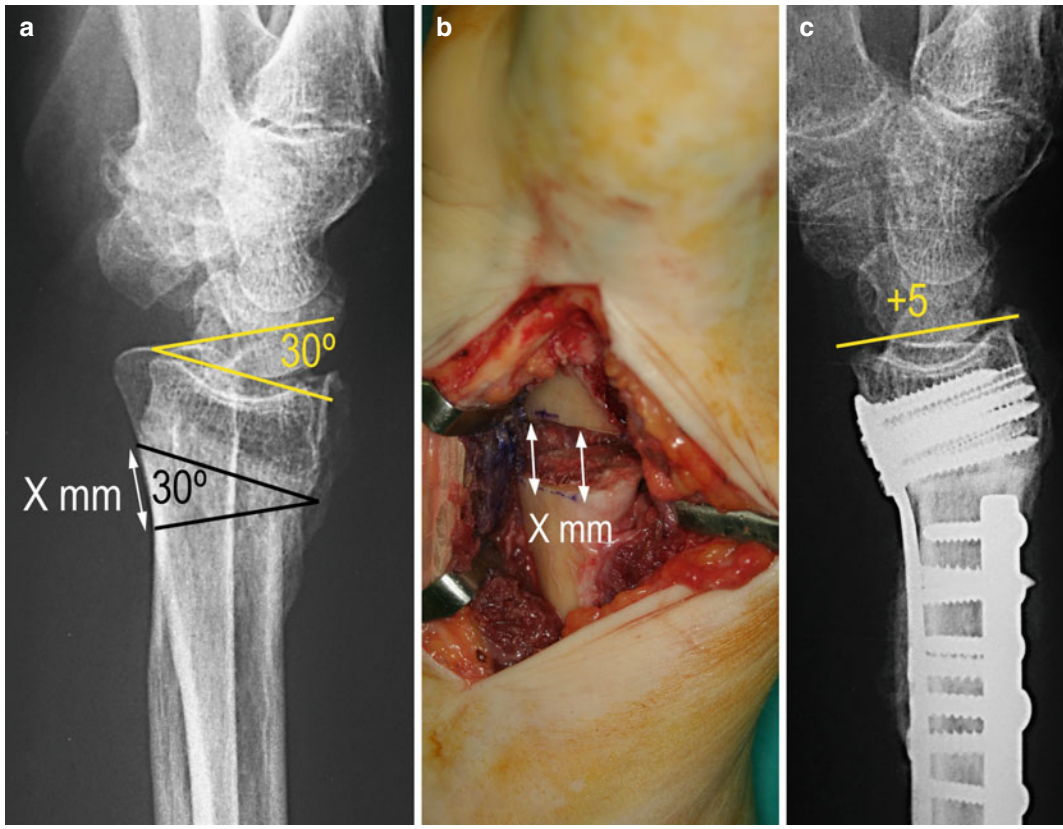


Fig. 7.8 Wada's osteotomy. (**a**) The correction angle is drawn at the planned osteotomy site in the lateral radiogram. The resulting value (named x) corresponds to the amount of volar cortex that has to be removed during the operation (**b**). (**c**) Correction of the dorsal tilt in the same patient after the osteotomy. The ulna has been shortened by the preoperatively measured ulnar variance (y mm) plus the amount of volar cortex removed (the "x" value). (**d**, **e**) Pre and postoperative P-A radiograms of the same patient (Copyright Dr. Piñal 2011)

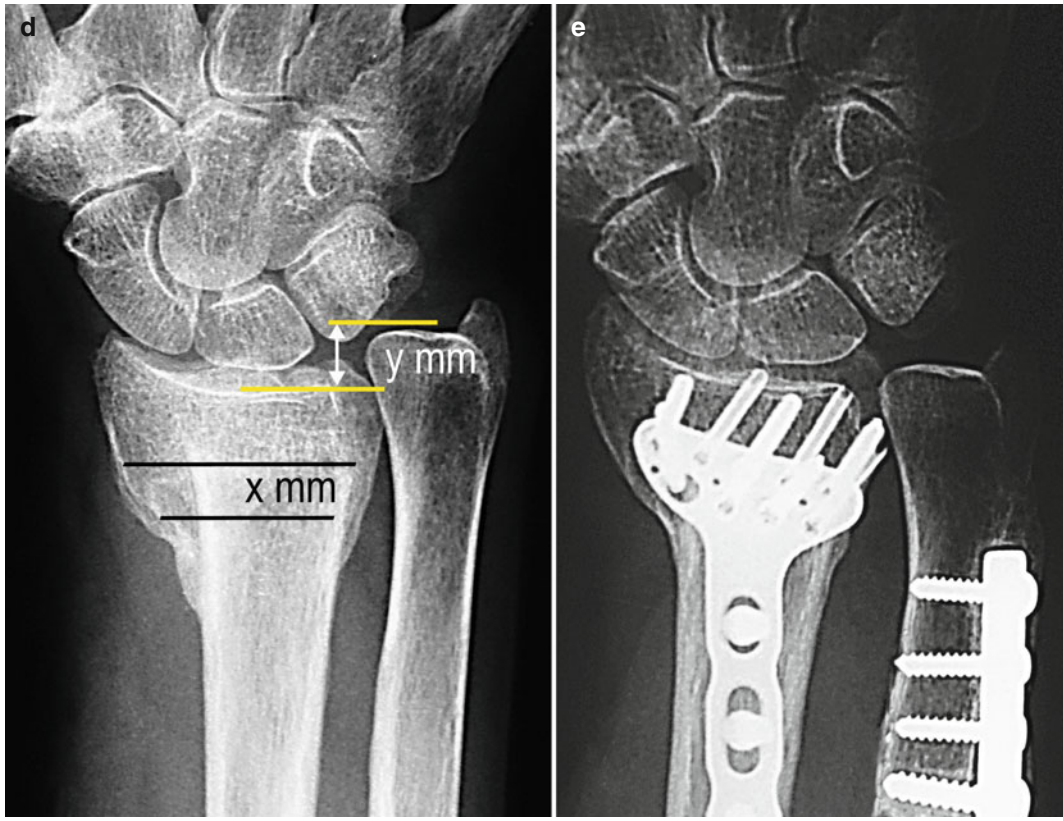


Fig. 7.8 (continued)

Clinical Pearl

It is critical to differentiate the simple rotational malunion from those that have true shortening. The former responds well to the much simpler de-rotational osteotomy. For the complex deformities I prefer now the Wada's approach, particularly in severe cases.

Volar Angulated Malunion

Increase in volar tilt of the radial epiphysis can occur as a result of a Smith type fracture, or an over reduced Colles type fracture. Limitations in extension and ulnar deviation are the norm. Nevertheless, most complaints are directed to the ulnar side, i.e. lack of supination, painful prominence of the ulna and volar sagging of the wrist (Fig. 7.9).

Fig. 7.9 Notice volar sagging and prominence of the right ulna in this volar malunion

The treatment of choice is volar open-wedge osteotomy and fixation with a volar fixed-angle plate [31]. The planning of the correction in the

sagittal plane is similar (but in reverse) to that undertaken for dorsal malunions. Several methods have been described to assess the malrotation in the axial plane, although the importance of this is limited: the flatness of the plate will correct any axial malrotation spontaneously. A critical issue when treating this deformity, is to avoid overcorrecting the volar tilt and converting it into a dorsal type malunion. Again, the use of volar precontoured plate will limit this risk.

The radius is approached as previously described for dorsally angulated malunions. The osteotomy is carried out, preferably at the malunion site, with the saw blade oriented parallel to the joint surface. Using a similar concept to the one referred to for pure sagittal malrotation presented previously, if no shortening of the radius exists, then the dorsal cortex is preserved to act as a hinge, hence performing a pure derotation (Fig. 7.10). Preservation of the dorsal cortex will prevent a common pitfall when correcting a Smith's type malunion, which is translation of the dorsal fragment instead of de-rotation, causing, at best, a partial correction. If this happens, the distal fragment becomes very unstable and the plate applied volarly will push the fragment dorsally, rather than derotating it. Reducing the distal fragment, by applying a laminar spreader and preliminary fixation by a Kirschner wire, will avoid this complication.

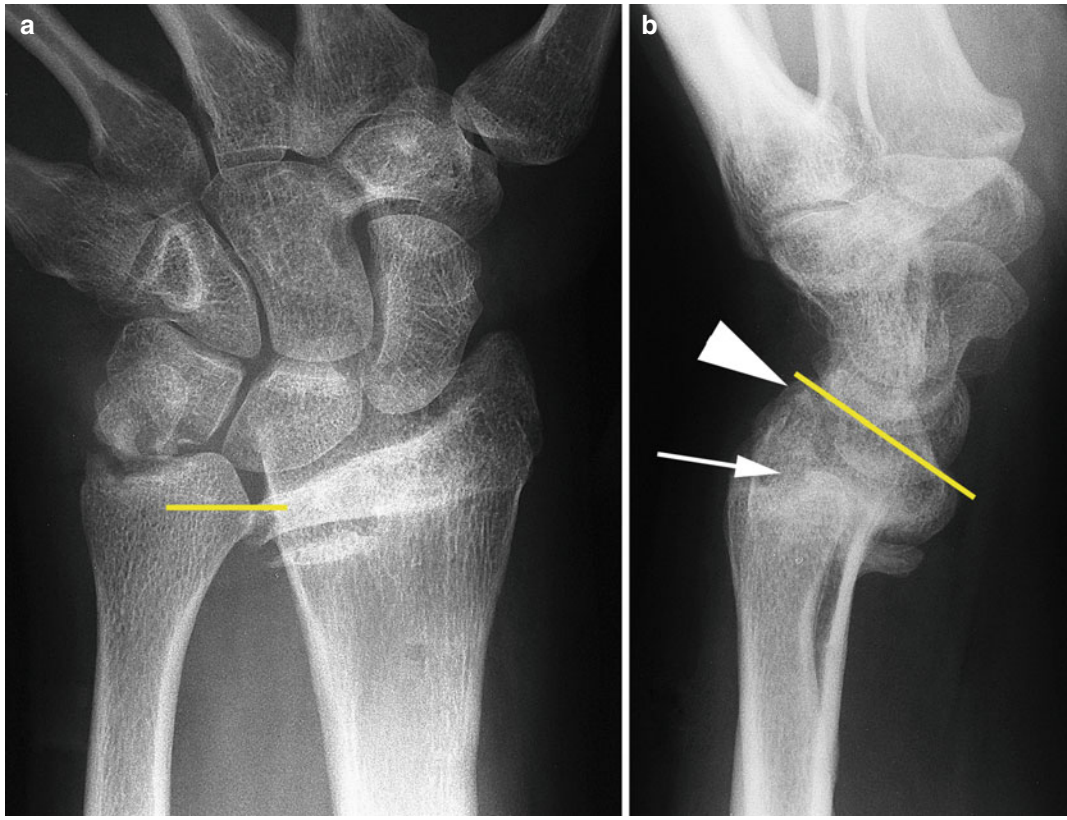


Fig. 7.10 (**a**, **b**) Pure volar malrotation in the sagittal plane. Notice that the dorsal cortex of the radius has been kept intact and that the ulnar head is proximal to the dorsal rim (*arrow*: level of the ulnar head; *arrow head*: distal-dorsal rim of the radius). (**c**) During the procedure great care was taken to maintain the dorsal cortex intact to act as a lid (*arrow head*). (**d**, **e**) The osteotomy has "lengthened" the radius despite being a pure derotational osteotomy (Copyright Dr. Piñal 2011)

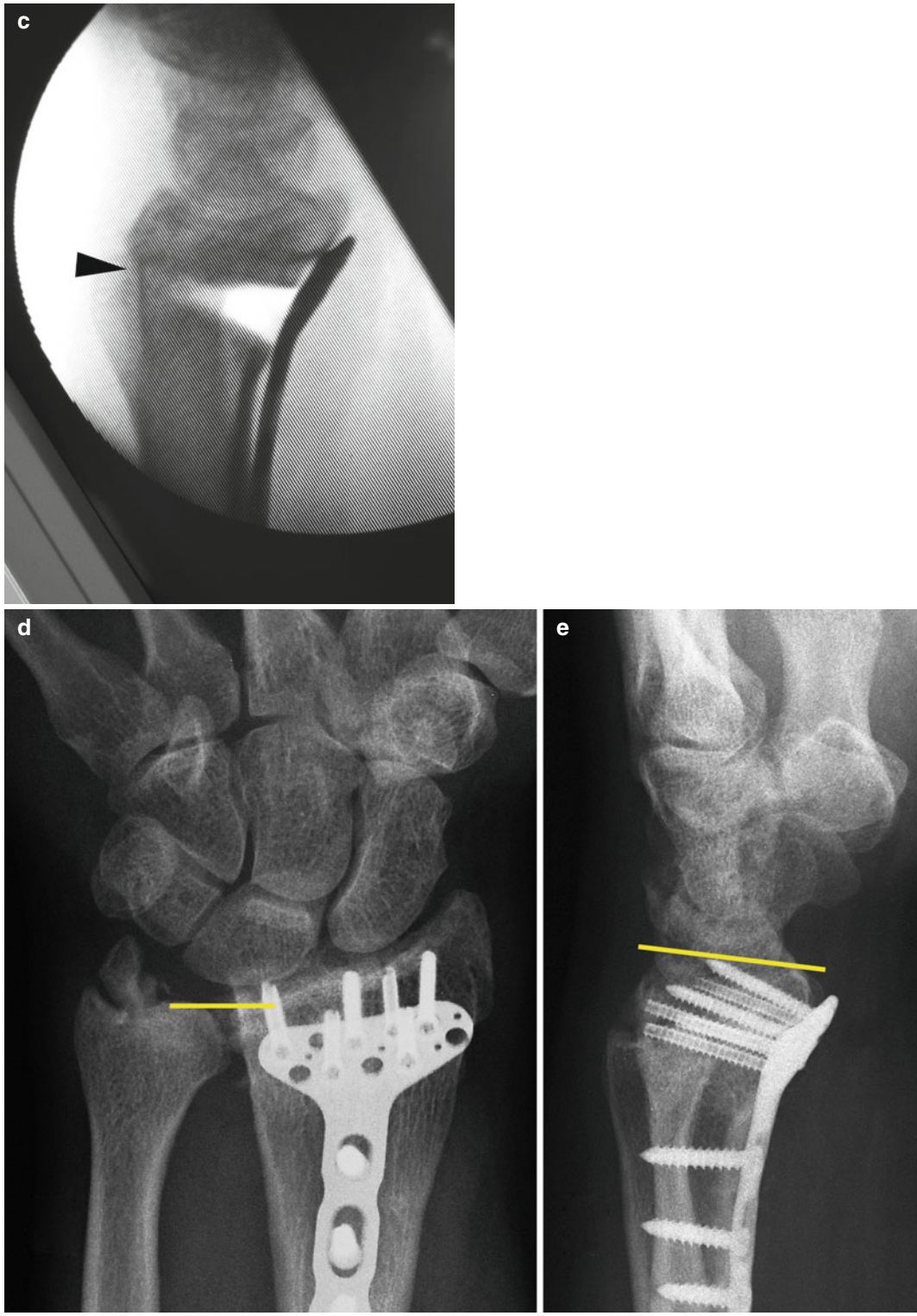


Fig. 7.10 (continued)

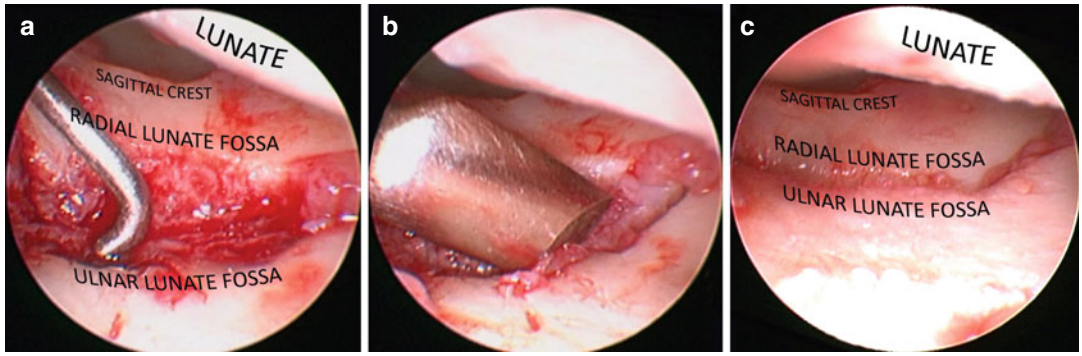


Fig. 7.11 (**a**) Correction of a 4 mm step-off on the lunate fossa (right wrist scope in 6R). (**b**) The osteotome (entering the joint through a dorsal portal) is separating the malunited fragments. (**c**) Corresponding view after reduction (Copyright Dr. Piñal 2010)

Intraarticular Malunion

Since pain and inferior results have been found in patients with an intra-articular step-off of more than 1 mm [32–34], we have defined any step-off larger than this 1 mm as intra-articular malunion, thus requiring surgery. Intra-articular malunion should be considered as a surgical emergency, since the cartilage of the radio-carpal joint can be irreversibly damaged very quickly. Poor prognostic factors for cartilage preservation are: location of the step-off (inter-facetary step-offs are much better tolerated than intra-facetary ones), height of the step-off and, above all, time and overzealous use of the hand during physical therapy. Time elapsed since the fracture further complicates the surgery (particularly open osteotomy), by making the fracture lines at the metaphysis barely identifiable.

The ideal candidate for an intraarticular osteotomy is a malunion of a single fragment in a young individual with a recent (less than 3 months) fracture. Single fragments, such as radial styloid, antero-ulnar or dorso-ulnar fragments, are the most amenable to correction, although four-part fractures and irregular malunion can also be addressed particularly if arthroscopy is used.

Correction of even the simplest intraarticular malunion is a difficult endeavour, whether either an open [35], or arthroscopic [36, 37] technique is used. A preoperative CT scan is invaluable for orientation purposes and the surgeon has to be experienced in this type of surgery. Arthroscopic guided osteotomy allows exact cutting through the fracture line within the joint, although considerable expertise in arthroscopy is required (Fig. 7.11).

Once cartilage has been damaged, osteotomy has no role and most will opt for some form of salvage, (see Chap. 4 in *Disorders of the Hand: Inflammation, Arthritis and Contractures*, vol 3). In younger active individuals, however, when the damage of cartilage is limited to the scaphoid or lunate fossae, reconstruction is possible by transplanting a vascularized osteochondral graft from the metatarsal [38, 39]. The mid-term results have proved very pleasing, but again, those are complex operations where expertise in microsurgery is required. Finally, in a group of patients where there is associated, but localized, cartilage loss, arthroscopic resection arthroplasty may be an option. At this time, we have experienced encouraging early results with this procedure [40].

Clinical Pearl

Intra-articular malunion should be considered as a surgical emergency, since the cartilage of the radio-carpal joint can be irreversibly damaged very quickly.

Ulnar Side Procedures

Ulnar sided pathology always accompanies the more obvious radius deformity and is a major

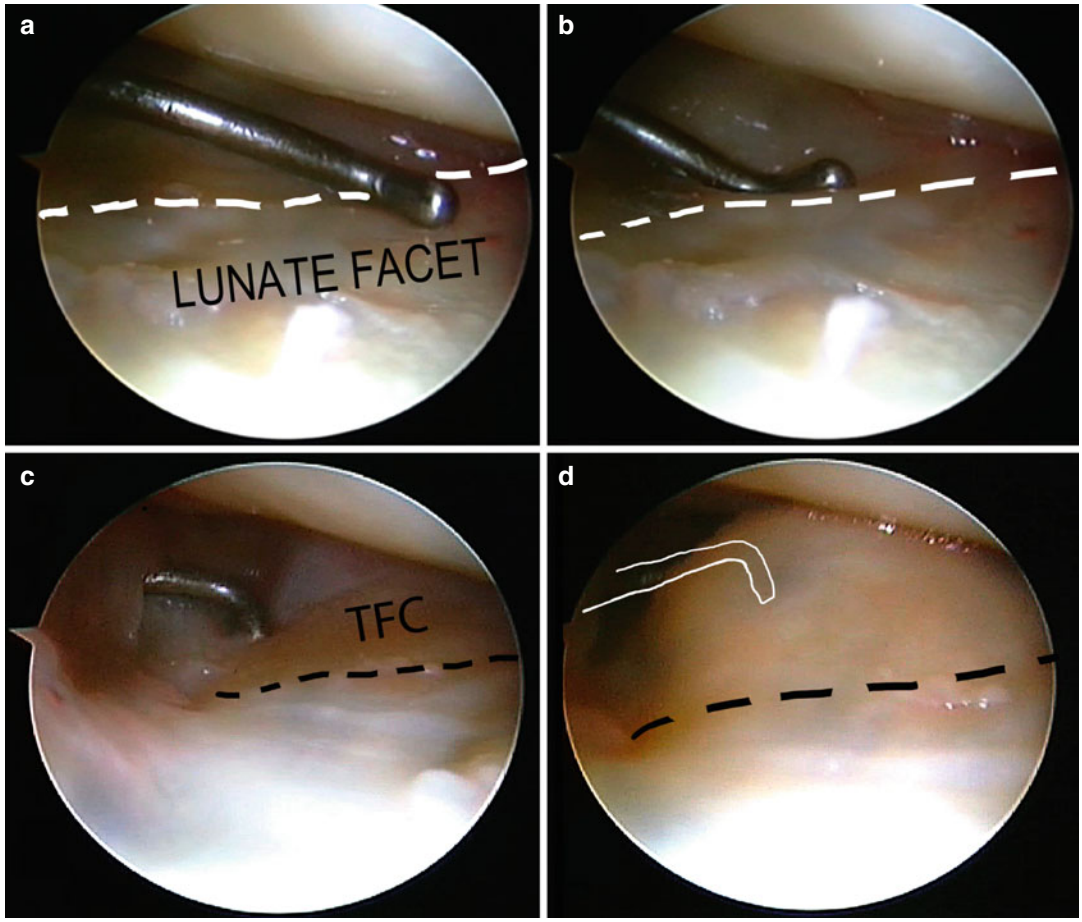


Fig. 7.12 Persistent signs of loosening and detachment of the TFC from the fovea are evident in this patient after radial osteotomy. (**a**, **b**) Positive trampoline test (the probe sinks into the TFC) (**b**). (**c**, **d**) Positive hook test (the TFC can be lifted from the fovea… in this case to the point of contacting the lunate) (left wrist, scope in 3–4, probe in 6R, dry arthroscopic technique) (Copyright Dr. Piñal 2011)

cause of persistent pain and secondary surgery. Impaction, incongruity, instability and stiffness can all be present together, or separately and be responsible, in differing degrees, for the patient's symptoms. By the same token, radial correction may, by itself, "spontaneously" deal with some of the ulnar problems, as the anatomic relations between the radius and ulna will be improved after the osteotomy. Specifically, symptoms related to impaction may not need to be addressed [3, 12]. Furthermore, symptoms related to instability may improve, once the sigmoid notch is repositioned and the ligaments retensioned. Conversely, if the TFC is detached from the fovea, the instability will persist and for such cases reinsertion into the fovea is needed (Fig. 7.12). It is therefore paramount that the surgeon carries out a re-exploration and an arthroscopy after the radial osteotomy in order to clarify the potential sources of any persistent pain. In brief, while the radius malunion is a two dimensional problem which can be addressed by osteotomy. Ulnar sided pain is usually more complex.

Ulnar head impaction symptoms should, as stated, be resolved by the radial osteotomy, as not only will the shortening, but also the dorsal tilt (both of which overload the ulnar head), will be addressed. In malunions where the radius has shortened with minimal tilting dorsally and

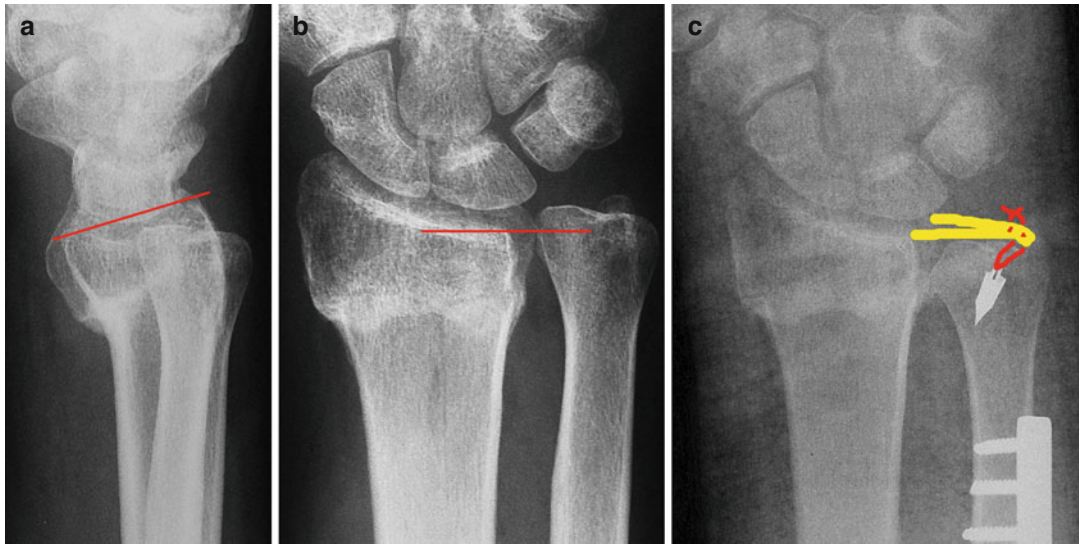


Fig. 7.13 (**a**, **b**) In a well aligned, in the sagittal and frontal planes, but shortened radius, an open ulna-shortening osteotomy is the best alternative to restore the anatomy of the DRUJ. (**c**) Instability of the ulna remained after the shortening due to TFC avulsion from the fovea. Arthroscopic reattachment of the TFC at the fovea was carried out (Courtesy of Dr. Piñal; Copyright by Springer 2010 in Ref. [42])

radially (less than 10º) and the major problem is of an ulnar impaction, the best option is an open ulnar shortening. We would not recommend an arthroscopic wafer procedure, as this can reduce the contact area within the sigmoid notch from 7 to 9 mm long [41] to 3–4 mm, causing an overload and again the potential for arthrosis. Conversely, ulnar shortening will restore the DRUJ relationships and will often retension the ligaments, improving any associated DRUJ instability. Arthroscopic examination of the TFC after shortening is recommended, as double lesions (impaction and TFC detachment) may occur [42] (Fig. 7.13).

Despite the lengthening achieved by a radial osteotomy, it may be insufficient to clear **impaction of the ulnar styloid** on the triquetrum. Patients with an ulnar styloid non-union are particularly at risk, due to the relative increase in length as a consequence of the non-union. Typically, the patient's symptoms are exacerbated while, with the forearm in supination, the wrist is brought into extension from radial to ulnar deviation; whilst minimal pain is produced when the same manoeuvre is carried out in pronation. During arthroscopy after the osteotomy, marked synovitis in the dorso-ulnar recess and sometimes the presence of the non-united styloid inside the radiocarpal joint are clues to the diagnosis. Resection of the offending styloid arthroscopically [43] (or via a mini-incision) is the treatment of choice. This adds very little to the procedure and clears what is, in my experience, a major cause of secondary pain (Fig. 7.14).

It is again important to remember that, if the foveal attachments of the TFC are torn, signs and symptoms of **distal radio-ulnar joint instability** will persist no matter what one does to the radius or the ulna. Excessive antero-posterior motion of the ulnar head, after the osteotomy, points to a major detachment. Arthroscopically, there are two clues for the diagnosis: loss of "trampoline effect" [44] and positive hook test [45] (Fig. 7.12). The former consists of loss of resilience, when the surgeon pushes down on the TFC with the probe in 6-R. Although quite popular, I find (perhaps because I always use the dry technique) the trampoline test misleading and prefer the reverse test, in which the TFC is hooked with the probe and lifted distally, attempting to stress its foveal insertion. Any lifting is diagnostic of rupture at the fovea and requires reattachment.

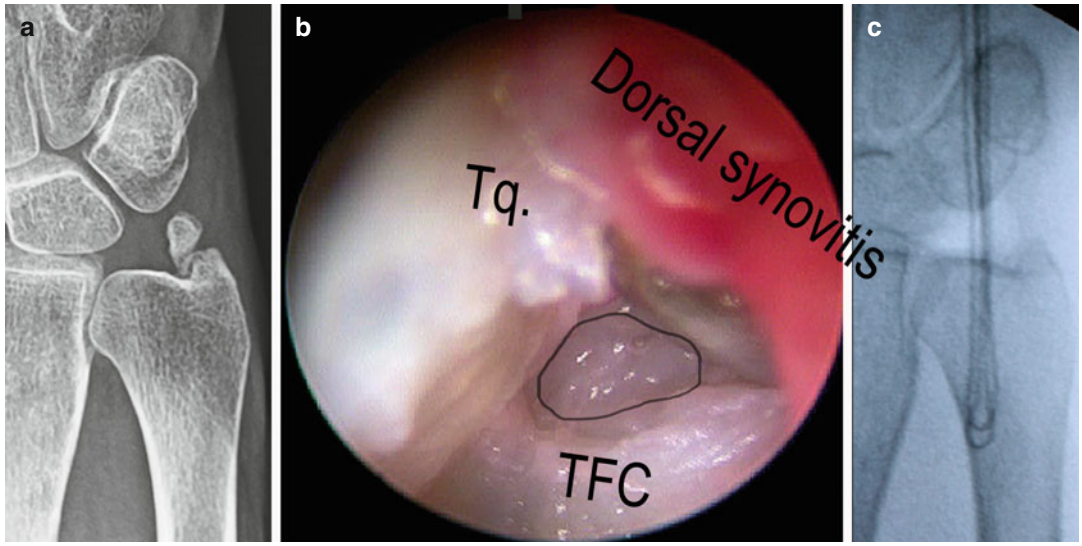
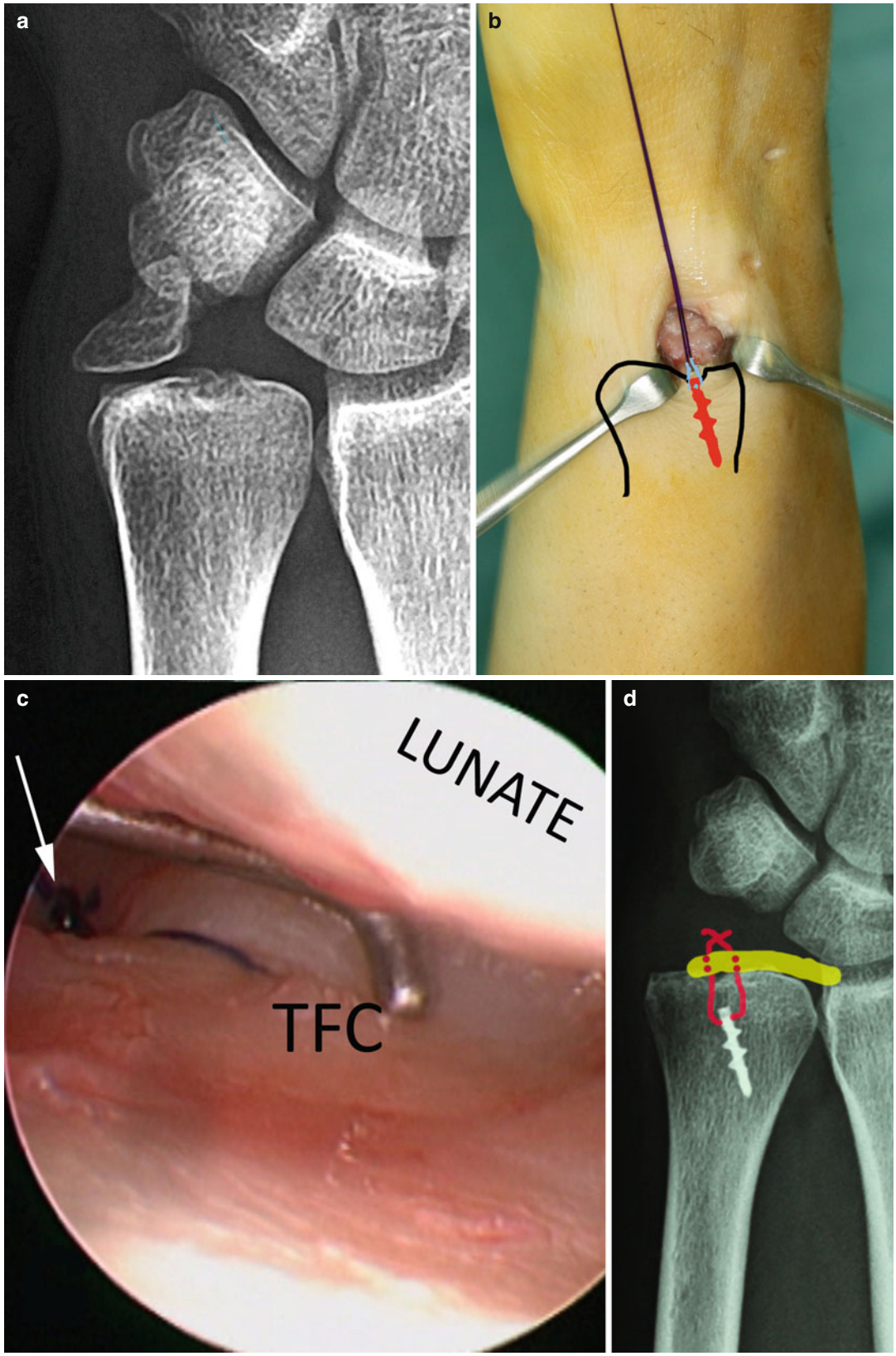


Fig. 7.14 (**a**) In a relatively well-aligned malunited (0° dorsal tilt) minimally shortened radius (less than 1 mm ulna plus) the "elongated" styloid seemed to be the main source of the patient's symptoms. (**b**) The tip of the non-united ulnar styloid is visible in the radio-carpal joint (encircled in the picture). This and the dorsal synovitis are the tell-tale signs of the aetiology of the patient's symptoms. *Tq* triquetrum, *TFC* triangular fibrocartilage (Dry arthroscopy right wrist: scope in 3–4 portal, probe in 6R). (**c**) Arthroscopic resection of the non-united fragment resolved the problem (Copyright Dr Piñal 2011)

My preferred technique for foveal reattachment is a modification of the arthroscopic assisted technique proposed by Atzei [46, 47]. Through a 1.5 cm incision, just volar to the ulnar styloid, a resorbable suture is placed in such a way as to leave the knot inside firmly bonding the TFC to the fovea (Fig. 7.15). Only under exceptional circumstances may one find insufficient tissue to reinsert on the fovea. For such cases, ligament reconstruction may be the only option either open [48, 49] or arthroscopically [46].

Symptoms caused by **joint incongruity** and/or **degeneration** at the DRUJ typically increase when the patient is asked to undertake active prono-supination, while the surgeon squeezes the wrist with his hand to increase compression at the DRUJ. In most cases the problem can be solved only by a "salvage" operation: Darrach, Bowers, Sauvé-Kapandji, or Ulnar Head prostheses are possible options. They all give similar results (never close to a normal DRUJ function) and the use may be more a matter of surgeon's preference, rather than the results of any scientific study. All these procedures share risks of instability of the ulnar stump, impingement, painful motion and some of them, non-union, loosening, breakage, etc. The technical intricacies and advantages of each of them are beyond the scope of this chapter. Nonetheless, as there is not a good alternative to a healthy distal radio-ulnar joint, the surgeon should make every effort to restore the sigmoid notch anatomy (Fig. 7.16).

Fig. 7.15 Technique for reattachment of the TFC with the knot placed inside. (**a**) The patient had an un-united ulnar styloid fracture with impingement and on exploration DRUJ instability after and old distal radius facture. The non-union was excised and a bone anchor inserted into the fovea (**b**). (**c**) After tightening the knot (*arrow*) the TFC is now tightly bonded to the fovea. (**d**) Schematic representation of the operation


a
b
c
LUNATE
TFC
d

Clinical Pearl

Although corrective osteotomy of the distal radius to reconstruct the natural anatomy is the foundation of treatment for this condition, malunion implies a major derangement of the wrist as a whole. Ulnar sided pathology always accompanies the more obvious radius deformity and is a major cause of persistent pain and secondary surgery. Impaction, incongruity, instability and stiffness can all be present together, or separately and be responsible, in differing degrees, for the patient's symptoms.

Outcome

The surgical correction of distal radial malunion is technically difficult. Even if accurate preoperative planning is performed, inability to achieve correction in one of the three key radiographic parameters (volar tilt, radial inclination and ulnar variance) can occur in up to 60 % of the cases [18, 29, 51]. This residual deformity has been held responsible for diminishing functional outcome after this type of surgery [18, 29, 51, 52]. Having said that, overall between 70 and 75 % of the patients obtain a good or excellent functional result after this type of surgery. Inability to fully correct the deformity, associated lesions, fixed carpal deformity and unattended ulnar side pathology can be the main cause for failure.

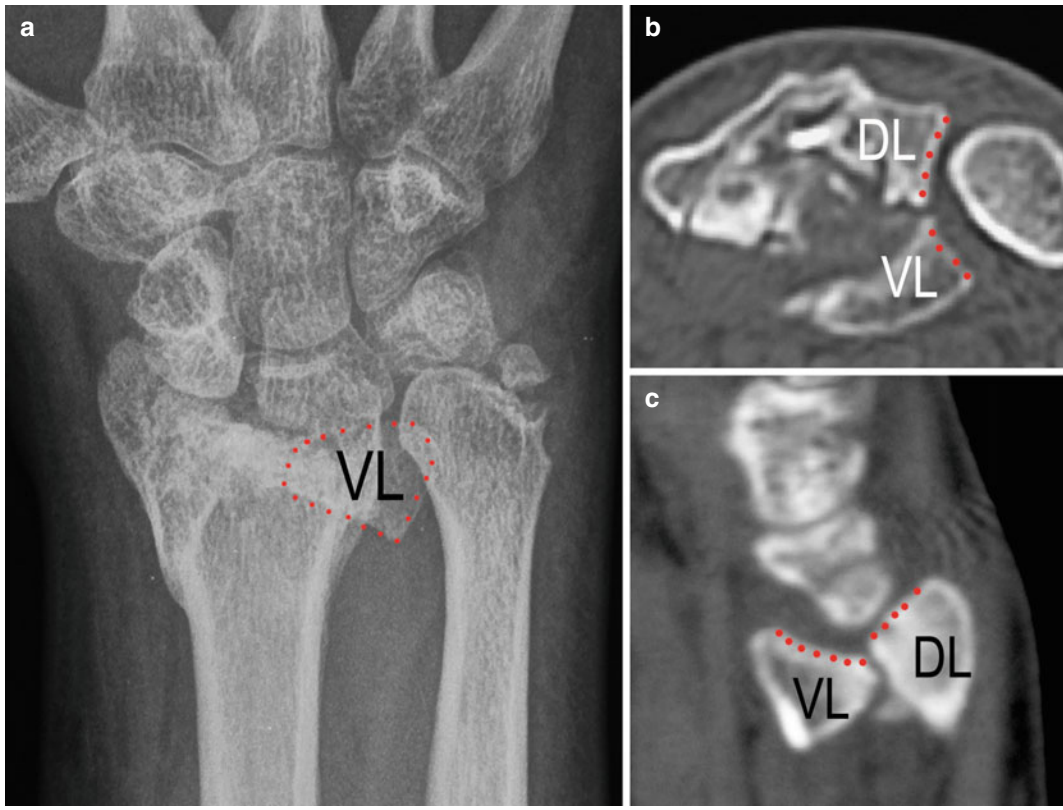


Fig. 7.16 Sigmoid notch reconstruction by osteotomy and reposition of two fragments. (**a**–**c**) In this 23-y-o patient, seen 4 months after his fracture, only the sigmoid notch was considered to be reconstructible. A volar-ulnar approach was used to osteotomize the volar-lunate facet fragment (*VL*) and a mid-dorsal approach was used to mobilize the dorsal-lunate facet fragment (*DL*). Each of those contained about half of the cartilage of the sigmoid notch. (**d**–**e**) Fixation was achieved with a buttress plate for the volar fragment and screws for the dorsal fragment. In order to achieve union in this severely scarred bed and to restore the sigmoid length, a vascularized cortico-periosteal graft from the medial condyle of the knee was inserted. (**f**–**g**) Nearly full pronation-supination was present at 2 years (From Del Piñal et al. [50]) (Copyright Dr Piñal 2011)

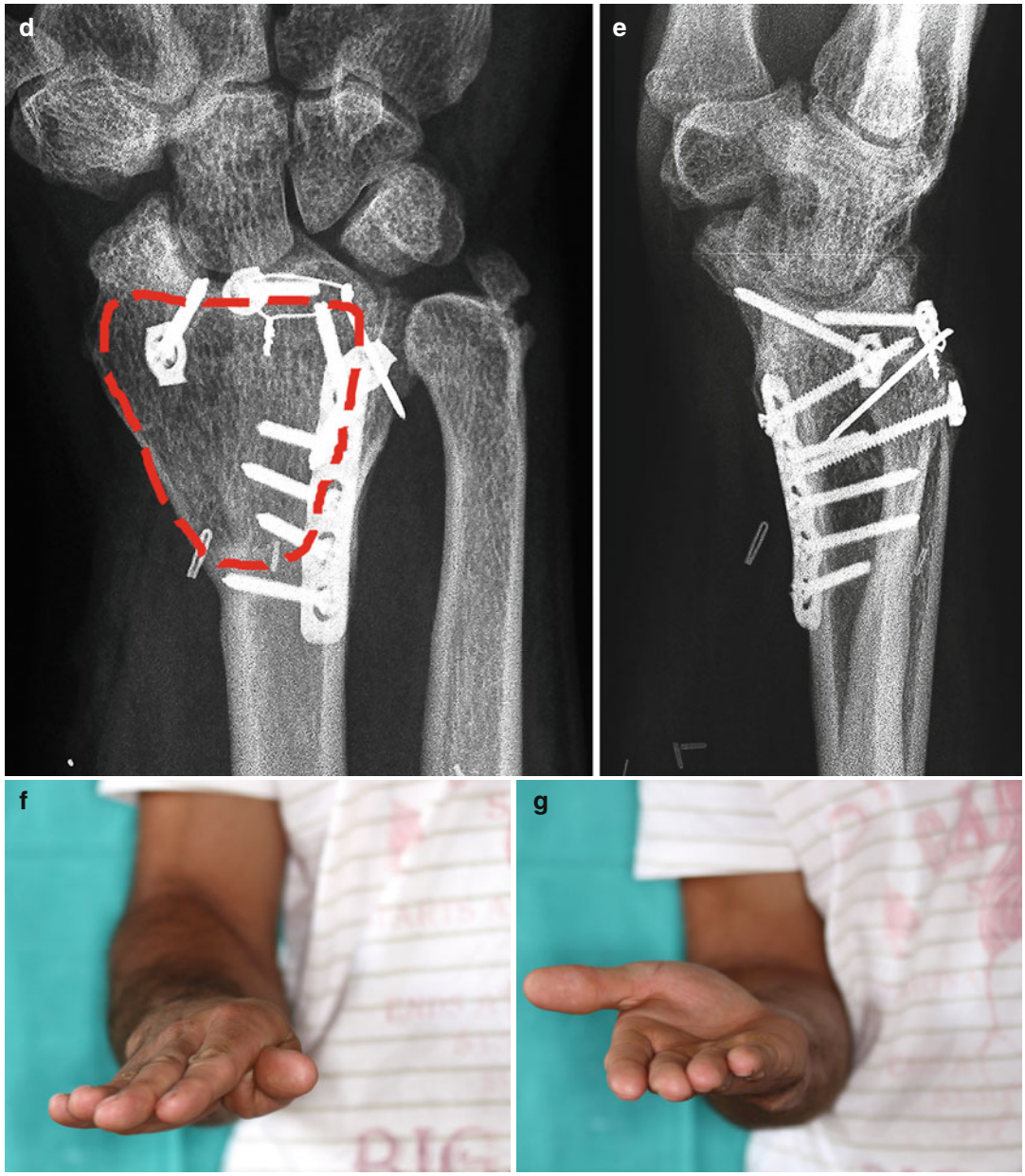


Fig. 7.16 (continued)

Conclusions

Malunion of the radius is a complex problem. It is much easier to prevent in the acute fracture, than later as a malunion. The radius, however, responds quite well to a carefully performed osteotomy, although a perfect correction is often difficult to obtain. Problems on the ulnar side, however, are more intricate and may require a variety of solutions. Finally, intra-articular malunion should be considered as a surgical emergency, in order to avoid irreversible damage of the cartilage surfaces.

Recently considerably progress in our understanding of the role of the distal oblique bundle and radial translation of the distal epiphysis in DRUJ instability after distal radius malunion has been made. The reader is referred to the references below to further expand this part.

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Local Vascular Bone Graft Reconstruction

8

Louisa N. Banks and Ian A. Trail

Keywords
Vascularised bone grafts • Pedicled bone grafts • Kienböck's disease • Preiser's disease • Intercompartmental supraretinacular artery • Extensor compartment artery • Corticocancellous graft • Avascular necrosis carpus • Microvascular • Non-union

Introduction

Huntington first described vascularised bone graft in 1905 (pedicled fibular to cover a tibial defect). Since then, pedicle grafts have been reported using the rib, clavicle, iliac crest, scapula, medial femoral condyle, distal radius, greater and lesser trochanter, pisiform, humerus and second metacarpal. Vascularised bone grafts (VBGs) have been used in the treatment of many pathologies including: those involving the carpus; intercalary defects of long bones; lower limb osteomyelitis; in the spine and also for reconstruction (of the mandible) [1–4].

It is thought that VBGs allow the transfer of living bone, maintaining cell viability and allowing primary bone healing without creeping substitution (leading to mechanical weakness). There is less osteoclast resorption and production of reactive bone, so less extensive remodelling of VBGs takes place. VBGs are thought to maintain near normal viability of osteocytes and offer "superior biologic and mechanical properties" over their non-vascularised counterparts [5].

Background/Aetiology

The use of VBGs has been described in the management of carpal pathology, such as scaphoid non-union avascular necrosis of the lunate (Kienböck's disease) and the scaphoid (Preiser's disease) since the 1980s [6]. VBGs can be local, pedicled or free grafts and offer the advantage over non-vascularised grafts (having some osteogenic potential), in that they retain live, functional osteocytes and osteoblasts. The literature shows that there are three sites at the

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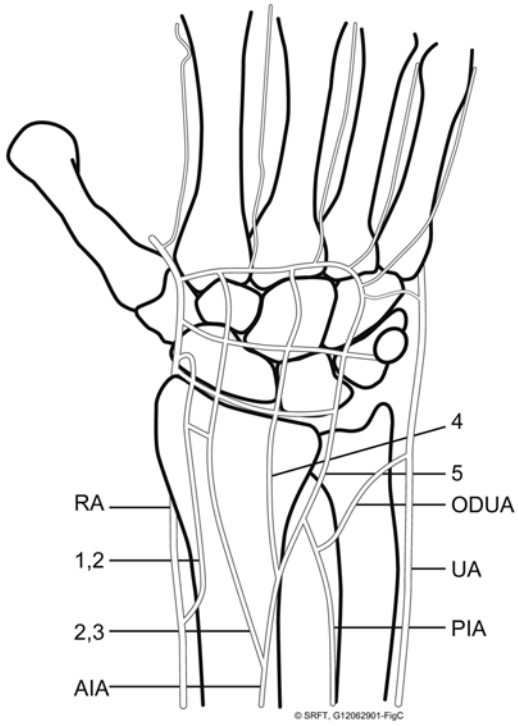

Fig. 8.1 Illustrates the dorsal wrist with potential pedicle sites and their corresponding arterial donors. 1, 2 ICSRA; 2, 3 ICSRA; 4th ECA; 5th ECA. *RA* radial artery, *UA* ulnar artery, *PIA* posterior interosseous artery, *AIA* anterior interosseous artery, *ODUA* oblique dorsal artery of the distal ulna

dorsal, distal radius and one at the dorsal, distal ulnar that are commonly used for VBGs. On the volar side of the wrist, the use of pronator quadratus has been described, which can be elevated in either an anterograde or retrograde fashion. Amongst the carpal bones themselves, the pisiform and part of the capitate have been harvested as pedicled VBGs. The metacarpals can also be used as donors. However, the most commonly used donor grafts for wrist pathology are taken from the dorsal aspect of the wrist. These are the 1,2 intercompartmental supraretinacular artery (ICSRA) the 2,3 ICSRA, the fourth extensor compartment artery (ECA) and the fifth ECA (See Fig. 8.1). These can be combined to provide a longer length of pedicle (for example, 4th plus 5th ECA combination graft) [6] (see Fig. 8.3). Kuhlmann described a graft that can be harvested from the anteromedial part of the distal radius and Zaidemberg described one from the posterolateral part of the distal radius [7, 8].

Vascular bone grafts taken from distant sites, such as the iliac crest, distal medial femur and fibula have also been described [6, 9]. These grafts have the advantage of having a greater volume of bone available for transportation and, as such, can "obliterate dead space; enhance bone healing and resist infection by ensuring blood supply" [2].

Scaphoid Injuries and Vascularised Bone Grafts

The scaphoid is the most commonly injured bone of the carpus (60 % of all carpal injures) and 5–15 % of these go on to non-union [10]. Fractures involving the proximal pole of the scaphoid are particularly susceptible to avascular necrosis, due to the tenuous blood supply of the scaphoid entering from its distal, dorsal aspect. Scaphoid fractures may also go onto non-union if there is fracture displacement (≥2 mm), or associated carpal instability. Pedicled (pronator quadratus), vascularised bone grafts for the non-union of scaphoid fractures have been reported since the early 1980s [11, 12]. Since then, several other sites have been used to provide VBGs – including 1,2 ICSRA, palmar carpal artery, iliac crest with implantation of the second dorsal vascular bundle, dorsal metacarpal arteries and free vascularised bone.

VBGs may also be of use in revision surgery, where primary fixation has failed and a non-union is clinically present, in proximal pole fractures and in AVN of the proximal pole. Even when VBG are used for scaphoid pathologies, non-union remains a problem in smokers, females and when significant carpal collapse is present [13].

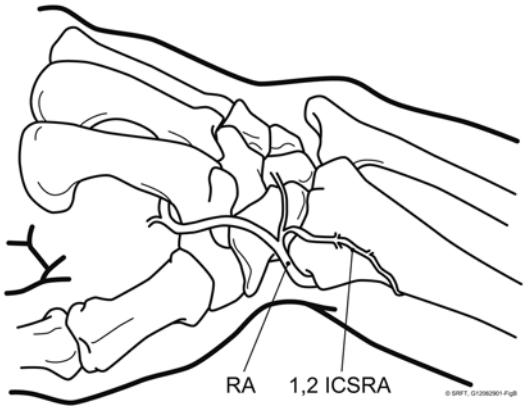

Fig. 8.2 Illustrates the 1, 2 intercompartmental supraretinacular arterial (1, 2 ICSRA) pedicle. *RA* radial artery

The 1,2 – intercompartmental supraretinacular artery graft (1,2 – ICSRA) is a pedicled, distal, dorsal radius vascularised bone graft based on a consistent and reliable arterial network, initially described by Zaidemberg et al. and further delineated and defined by Sheetz et al. [8, 14] (see Fig. 8.2). Waitayawinyu et al. in a recent study noted that the origin of the 1,2 – ICSRA from the radial artery was approximately 2 mm proximal to the tip of the radial styloid and gave a mean pedicle length of 22.5 mm (range 15–31 mm) [15]. The origin of the 1,2 – ICSRA was often noted to be separate, but could be combined or shared with the origin of the dorsal scaphoid branch. As the 1,2 – ICRSA courses on top of the extensor retinaculum between the first and second extensor compartments, there is an average of five to six perforating branches.

Pedicle grafts from the volar aspect of the distal radius have also been used [7]. These grafts are based on the palmar carpal artery, which lies between the palmar periosteum of the radius and the distal part of the superficial aponeurosis of the pronator quadratus. The bone graft is harvested from the ulna aspect of the distal radius and incorporates a 5 mm wide strip of fascia and periosteum. This has the advantage of allowing a corticocancellous graft to be harvested, which can be used to correct flexion deformity to the scaphoid.

Avascular Necrosis of the Scaphoid (Preiser's Disease) and Vascularised Bone Grafts

Preiser's Disease is idiopathic AVN of the scaphoid and is postulated to be due to a fault with the blood supply to the scaphoid, as yet unknown. Similarly to using VBGs for scaphoid fractures, VBGs for Preiser's disease were also described by Braun in the 1980s, again from the volar distal radius (pronator pedicle). Patients with localised AVN, as opposed to global AVN do better with VBG [16].

As with scaphoid fractures, VBGs for Preiser's disease are not recommended in the presence of carpal instability and radiocarpal osteoarthritis [17]. At the time of surgery, as with scaphoid non unions, if significant degenerative change is present, salvage procedures, such as proximal row carpectomy, or scaphoid excision with four-corner fusion, should be considered.

Kienböck's Disease and Vascularised Bone Grafts

Kienböck's disease is AVN of the lunate bone and is more common than Preiser's disease. The exact aetiology is still unknown. However, both mechanical (such as negative ulnar variance) and biological abnormalities (tenuous blood supply) have been implicated. In the earlier stages of the disease, that is when there is little collapse or fragmentation of the lunate, reconstructive surgery is an option. This would take the form of procedures to unload the lunate, such as radial or capitate shortening, together with vascularised bone graft. VBGs for the lunate were described 40 years ago (pedicled bone graft using the scaphoid tubercle on the abductor pollicis brevis muscle) [18].

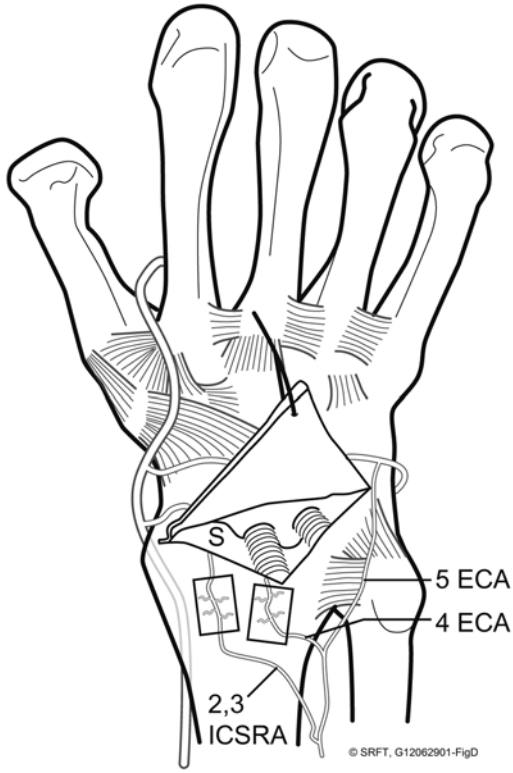

Fig. 8.3 Illustrates the position of the second and third intercompartmental supraretinacular arterial (2, 3 ICSRA) pedicle and the fourth and fifth extracompartmental (*ECA*) pedicle. *S* scaphoid

Subsequently, many other types of vascular bone graft have been used, although the most common is the 4 and 5 ECA (see Fig. 8.3).

Surgical Techniques and Rehabilitation

Pedicled VBGs can be a technically demanding procedure. Any surgeon undertaking this particular operation should have a thorough knowledge of the microvascular supply around the wrist and, obviously, access to magnification. Of greatest concern, is that vessels can easily be damaged during insertion and kinking of the pedicle, or vessel damage can lead to the bone graft effectively becoming devascularised. The graft needs to be fixed in a stable fashion (for example, with a Kirschner wire) when it reaches its final destination.

Free VBG (such as iliac crest) allows for a larger volume of bone to be transported and this may be of more use when treating problems such as severe humpback deformities in the case of chronic scaphoid pathologies. Evidence has suggested that the incidence of humpback deformity is significantly greater in VBGs that go onto non-union [13].

If capitolunate wear is identified at the time of surgery, then VBGs are probably not suitable and a salvage procedure should be considered instead. However, if degenerative changes are isolated to the radial styloid, then a radial styloidectomy can be undertaken in conjunction with a VBG. Finally, any inter-osseous scaphoid cysts need to be fully excised prior to the introduction of any graft.

Moran and Shin (2007) described the technical difficulties in the use of VBG for the treatment of Preiser's disease [19]. Perforation of the proximal part of the scaphoid can occur when attempting to remove the necrotic/sclerotic bone. This, in itself, can also present a problem as significant defects may be left once necrotic bone is removed, necessitating large volumes of VBG.

In Kienböck's disease, the use of dorsal VBG may be the most simple as a single incision can be utilised, without compromise to the volar carpal ligaments. An unloading procedure, such as capitate shortening, may aid in revascularisation, as it is thought that the initial stages of bony healing increase osteoclast response and bony absorption and may in turn lead to initial weakening, causing further collapse [20].

Clinical Pearls

Ensure knowledge of the microvascular supply around the wrist and that access to magnification is available prior to embarking on VB grafting.

Ensure that the pedicle is not kinked.

Make sure that the graft is stably fixed (may need adjuncts to aid this).

Try and utilise a single incision for both harvesting of the bone graft and treatment of the pathology.

Outcome Including Literature Review

Scaphoid Pathology

Reports describing the use of the pronator muscle as a pedicle from the volar radius for bone grafting of (chronic) scaphoid non-unions were first described in the 1980s and "excellent" results with good bony union were reported [11, 12]. A Brazilian study (number (n) = 80) compared distal radial VBGs with non-vascularised bone graft from the iliac crest for treating scaphoid fracture non-union. Functional results (in terms of range of motion, grip strength) and radiographic findings were compared, with similar results and negligible difference in the mean time to union (9 months in the vascularised group versus 8 in the non-vascularised group) [21]. Another Brazilian group concluded that VBG (from the dorsal, distal radius) had superior results (in terms of function and time to union) for the treatment of scaphoid non-union (n = 86) in the presence of a sclerotic, poorly-vascularised proximal pole [22]. The literature often represents small patient numbers, so it may be difficult to ascertain their significance, but Boyer (n = 10) reported a 40 % non-union rate for AVN of the scaphoid proximal pole when treated with a 1,2 ICSRA VBG [23]. Other series (using the 1, 2 ICSRA VBG) have reported more ambitious rates of 90–100 % with regard to union, but again numbers of patients have been small (n = 11, 22) [8, 24]. Gras and Mathoulin (2011) have reported their use of the pedicled volar carpal artery graft in the treatment of 111 scaphoid waist or proximal pole non-unions, of which 73 were primary procedures and 38 were secondary, with a mean non-union period of 26 and 33 months respectively [25]. Sixty-four patients had no instability or malalignment (Alnot grade IIA), 42 had a mobile non-union with anterior defect resulting in DISI deformity (Alnot grade IIB) and five had a mobile non-union and DISI deformity with isolated radioscaphoid arthritis (Alnot grade IIIA). None of the patients had proximal pole AVN. Seventy of seventy-three patients (96 %) treated primarily and 34 of 38 patients (90 %) treated secondarily, achieved union at a mean of 9.7 weeks (range, 6–24 weeks) and 10.8 weeks (range, 6–24 weeks), respectively. Although statistical values were not reported, patients in both the primary and secondary groups had improved grip strength (26.8–42.3 and 20.4–39.5 kg, respectively), improved range of motion (18° and 28° increased flexion-extension arc, respectively) and most had complete resolution of their pain (96 and 56 % of patients respectively). In the primary treatment group, 95 % of patients had excellent or good results, based on the Mayo wrist score, compared with only 74 % of patients in the secondary treatment group. Radiographic carpal measures and arthritis were not reported.

A bone graft from the thumb metacarpal pedicle on the first dorsal metacarpal artery has been used as a volar interposition graft to treat persistent scaphoid non union. Bertelli and colleagues (2007) used this graft, harvested and fixed through a single volar incision, to treat ten patients with persistent scaphoid non-union of at least 2 years duration. In this group nine patients went on to non-union with good clinical results [26].

Merrell et al. (2002) carried out a meta-analysis on scaphoid non-unions and found that unstable non-unions, treated with screw fixation and bone grafting, had a 94 % union rate when compared to Kirschner wiring and wedge grafting (77 % union) [27]. Eighty-eight per cent of patients with AVN of the scaphoid proximal pole treated with a VBG went onto union, compared with 47 % of those treated with screw and wedge fixation. They felt that:"established, unstable non-unions should be treated with screw fixation and wedge grafting". They also suggested that a VBG may be preferable for patients with AVN of the scaphoid's proximal fragment, or after a previously failed surgery.

Evidence for VBG in Preiser's disease is also limited with small patient numbers (n = 8 in Moran's study; n = 9 in Kalainov's), but indicates that although the disease may progress (eventually leading to wrist fusion), pain and functional scores may improve with VBG, even though there is often incomplete vascularisation of the scaphoid following the procedure [16, 28]. At this time it is not known whether any additional procedure

to unload the scaphoid post-operatively (for example, using an external fixator, or Kirchsner wires) affords any additional benefit [19].

Lunate Pathology

There is evidence that replacing the necrotic lunate (in stages Lichtman's/Ståhl's II to IV) with a vascularised pisiform transfer gives good results in terms of pain and wrist function over the long term (10–12 years) [29]. Other studies, however, have suggested that the results don't last in the medium to long term and that collapse is common, leading to fair-to-poor outcome [30, 31]. This has been shown with both pedicled and free VBG (iliac crest) [32]. The most common VBG used for the lunate, according to the literature, seems to the 4th + 5th ECA (see Fig. 8.3). As Moran & Shin observe, this pedicle has a large diameter and length and is ulnar based, allowing for arthrotomy, without compromising the vessels [19].

Again, there is little or no evidence to date, comparing one VBG with another, or VBGs in combination with such procedures as capitate or radial shortening. It has been shown, however, that isolated capitate shortening for early stages of Kienböck's, (stages II, IIA) can allow for revascularisation of the lunate [33]. Waitayawinyu reported that, in combination with VBG (n = 14), capitate shortening is effective in significantly improving grip strength (58–78 % of the normal side), satisfaction score and arc of motion [34]. They found that the average time from osteotomy to healing of the capitate was 48 days and felt that it was an effective treatment of ulnar-positive Kienböck's disease prior to the onset of radiocarpal arthrosis.

Complications of Treatment

Local pedicled VBGs are the most commonly used and are technically less demanding than free VBGs, with less associated morbidity [35]. As mentioned earlier, large volumes of necrotic bone within the scaphoid shell need to be removed in Preiser's disease and a similar volume of VBG required in it's space. If the VBG is local (for example from the dorsal distal radius), the patient may need long periods (2–3 months) of wrist immobilisation, which can obviously lead to stiffness. There is also the post-operative potential that the scaphoid can fragment and collapse further following revascularisation.

Pedicled grafts may become kinked, or the vessels damaged in insertion in the scaphoid or lunate, which can lead to devascularisation. The grafts themselves may increase osteoclast activation, which can lead to early collapse (secondary to bone reabsorption), which is why offloading the carpal bone in question may be of benefit.

Conclusions

Vascularised engineered bone has been created and demonstrated in a laboratory environment by implantation of vascular bundles into bone fragments, or demineralised bone matrix with subsequent bone formation and remodelling [36, 37]. This new technology may well be the way forward for the future of carpal pathology, considering how far we've come in the last 30–40 years.

Concurrently, however, there is a general paucity of level I or II evidence in the literature comparing the different sources of VBGs and pedicled versus free grafts. With regard to non-vascular grafts, however, meta analysis, whilst showing variable outcome does seem to indicate more favourable results with VBG, in terms of pain, function and bony union. As to the VBG used, it is often down to surgeon choice and experience. Currently, local VBGs remain a popular tool in the armament of the hand surgeon for treating particular difficult carpal bony pathology.

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Chronic Instability of the Fingers and Thumb

9

Carlos Heras-Palou

Keywords
Base of thumb • MCP • Interphalangeal • Chronic instability • Surgery • Rehabilitation

Introduction

The human hand explores a hostile environment. Injuries are common and frequently involve the joints of the thumb and fingers. The impact of injuries to these small joints can be surprisingly significant. Partial injuries to the ligaments, or sprains, often heal well, but have a tendency to stiffness if not mobilized soon. Complete ruptures of ligaments have a tendency to instability, a less common but more disabling outcome.

It is considered that the thumb represents 50 % of the function of the hand. Instability at either the CMCJ or MCPJ can therefore cause significant impairment. Injuries to the finger metacarpophalangeal joint are rare and probably under diagnosed. Often, initially considered to be "a sprain" patients may present months after the injury, when repair is not possible and a reconstruction is required. The proximal interphalangeal joint has been called "the epicentre of the hand" [1] because of its functional importance, and injury to this joint often results in a poor outcome. Perhaps more than anywhere else in the body, a clear understanding of the anatomy of these joints is required for successful treatment.

Instability of the Carpometacarpal Joint of the Thumb

The CMCJ is a biconcavo-convex universal joint, formed by two saddle shaped surfaces that allow flexion-extension, abduction-adduction and pronation-supination between the trapezium and the first metacarpal. The combination of these movements allows the most important function in the hand: thumb opposition. The geometry of the joint, however, offers no inherent stability. Forces across the CMCJ of the thumb are approximately 12 times the pinch force generated during opposition of the thumb [2]. As a consequence, when the pinch between thumb and index finger, generates a force of 7 kg, the resultant force across the base of the thumb is 84 kg. The combination of the lack of inherent stability and huge forces

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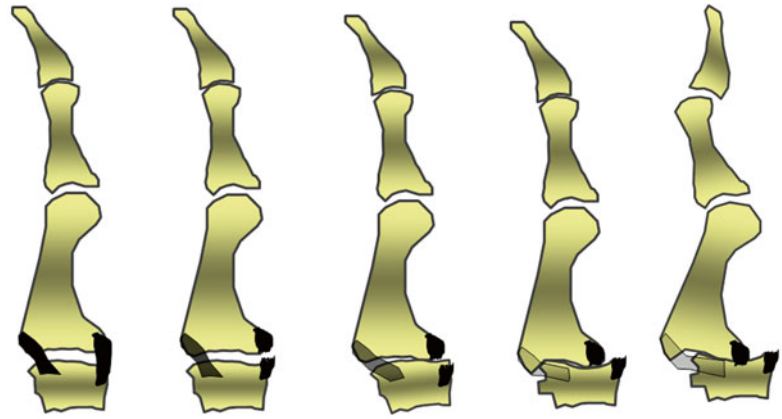


Fig. 9.1 Injury of the beak ligament of the CMJC, with stretching of the dorsal ligaments, may results in subluxation of the joint, adduction of the metacarpal, hyperextension of the MCPJ. In time, degenerative changes will develop

across this joint makes instability at the base of the thumb a significant problem.

Chronic instability of the CMCJ of the thumb can be primary or secondary. Primary instability is caused by three different factors: shape of the trapezium, laxity of the ligaments and muscular imbalance. A trapezium that is small (hypoplastic) or with increased obliquity will predispose to dorso-radial displacement of the metacarpal. This can be exacerbated by congenital ligament and capsular laxity. Muscle imbalance can also increase the instability, for example, lack of insertion of one of the slips of abductor policis longus onto the trapezium, will result in the total force of this tendon acting on the metacarpal causing a shearing force at the CMCJ [3]. Conversely, contraction of the first dorsal interosseous muscle will tend to stabilise the metacarpal onto the trapezium, preventing dorso-radial displacement when loading the thumb. Secondary instability can be due to ligament injury, fractures of the trapezium or the first metacarpal or surgical removal of the trapezium.

The anatomy of the ligaments of the CMCJ has been the focus of several studies, with the ultimate description of 16 different ligaments. The key factors in understanding the anatomy of this joint are the beak of the thumb metacarpal, the trapezial recess where the beak ligament inserts and the dorsal ligament complex [4]. As the thumb moves into opposition, it pivots around the volar beak of the thumb metacarpal, tightening the dorsal ligament complex. In this position the joint becomes congruent and stable in its position of function. This explains why the CMCJ can appear to be very lax when examined with the metacarpal extended and abducted, but allows strong pinching and grasping without yielding.

Ligaments also have an important role in proprioception, crucial for joint stability. The dorsal ligaments are very rich in sensory nerve endings, while the beak ligament is histologically more akin to joint capsule with scarcely any nerve endings [5]. It seems that these dorsal ligaments inform the muscles around the thumb, to make them effective dynamic stabilizers.

Patients with CMCJ instability typically present with pain at the base of the thumb, worse during or after activity, weakness of the hand in pinch and grip, often intermittent swelling and sometimes clicking during movement. Hyperextension at the metacarpophalangeal joint will cause a tendency for the metacarpal to adduct and for the base of the metacarpal to displace in a dorsal and radial direction, increasing the instability; therefore significant MCPJ hyperextension (more than 30°) will need to be addressed as part of the treatment of CMCJ instability.

There may be a history of trauma, often the injury happens with the thumb in opposition, for example holding the steering wheel of the car during a road traffic accident. In this position, the ligaments are tight and any force in a dorso-radial direction will tend to damage them. Alternatively, patients may present without a clear history of trauma, but with a long history of pain of insidious onset. CMCJ instability is an etiological factor in osteoarthritis of the joint, a very common condition, and there is a spectrum of pathology between pure instability and pure joint degeneration (Fig. 9.1).

Radiographs of the thumb are often of normal appearance. However, on occasion they can show a dorso-radial displacement of the metacarpal. Stress radiographs may show displacement of the base of the metacarpal, [6], which correlates with joint laxity. MRI has been recommended to image the beak ligament [7]. Arthroscopy of the CMCJ can show ligament damage and also, importantly, assess the joint surface for any degenerative changes that may be present.

In the acute situation, CMCJ instability can be treated conservatively with activity modification and splints with the metacarpal in palmar abduction-extension for 4–6 weeks. However in chronic cases, these measures often only help in the short term and symptoms recur later on. Recommended surgical treatments include metacarpal osteotomy or ligament reconstruction. Currently, there is good experimental evidence for the use of either.

Extension osteotomy of the thumb metacarpal by 15° decreases joint laxity in radio-ulnar, dorsal-volar and pronation-supination directions, and may stabilise the thumb to a degree or at least similar to that of a standard ligament reconstruction [8].

There is also evidence that stabilisation of the joint decreases symptoms and slows down the development of degenerative changes. Several techniques to stabilise the CMCJ of the thumb have been described, most of them are effectively a tenodesis, using either palmaris longus, extensor carpi radialis longus, APL or flexor carpi radialis. The commonly used techniques are those described by Eaton and by Brunelli.

After a meticulous anatomical study, Brunelli described a method of stabilisation of the CMCJ using just one of the tendons of abductor pollicis longus (98 % of thumbs have several APL tendons) which is passed through a drill hole placed in the base of the first and second metacarpals to reconstruct the intermetacarpal ligament. This new ligament provides a pivot point for the joint allowing movement, but preventing radial displacement of the base of the metacarpal.

In the classic technique described by Eaton and Littler, a modified Wagner approach is used, taking care to protect the branches of the radial nerve. The radial border of the thenar muscle is incised and the muscles elevated extraperiosteally to expose the CMCJ capsule. Blunt dissection is extended dorsally to expose the metacarpal cortex. The capsule is incised and the joint can be inspected. If there are degenerative changes, it may be better to proceed to an arthroplasty procedure rather than a stabilisation.

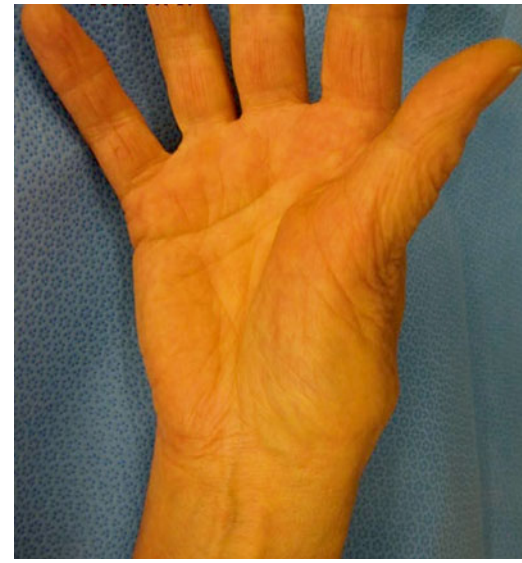


Fig. 9.2 Hyperextension of the MCPJ can be secondary to adduction of the metacarpal. This in turn accentuates the dorsal subluxation at the CMCJ, contributing to degeneration of the joint and thumb collapse

Half the FCR tendon is harvested for a length of 9 cm. A tunnel is drilled from dorsal to volar in the thumb metacarpal, 1 cm distal to its base and the hemi-tendon is passed from palmar to dorsal through the tunnel and sutured to the metacarpal periosteum. The graft is then passed under the APL tendon, then sutured to the APL, before passing underneath and around the ulnar half of the remaining FCR tendon. The graft is sutured as it loops around. If there is enough length, the graft can be brought back dorsally and sutured to APL again. A Kirschner wire is then drilled from the metacarpal into the trapezium and kept for 5 weeks until the soft tissues have healed.

If on lateral pinch there is 30° of hyperextension at the MCPJ, either a MCPJ capsulodesis or arthrodesis is recommended (Fig. 9.2).

After surgery, the thumb is kept immobilised for 5 weeks in a thumb spica splint. Active range of movement exercises are started after removal of the wire, and strengthening exercises can start 2 months after surgery.

Clinical Pearl

Stability of the CMC joint of the thumb is uncommon. Generally, a significant force is required to de-stabilize this joint. Diagnosis is based on clinical suspicion, but can be aided by stress x-rays. MRI scans can also be increasingly useful. Treatment by tendon reconstruction is generally effective.

The outcomes are generally good, with only 17 % of patients reporting pain with activities of daily living, and 54 % reporting pain with strenuous activities, 10 years after surgery. When tested against stress, 87 % demonstrated joint stability at the CMCJ.

MCPJ Thumb Instability

The MCPJ of the thumb has little intrinsic stability and depends on its ligaments and muscle attachments. The range of motion is the most variable of any joint in the body, with a wide spectrum; some thumbs can flex to 90° degrees, while others have hardly any movement.

The collateral ligaments arise from the lateral condyles of the metacarpal and pass obliquely to insert on the volar third of the proximal phalanx. They are tight in flexion and loose in extension. The accessory collateral ligaments originate from a more volar site on the metacarpal head and insert onto the volar plate and sesamoid bones on each side of the joint, being tight in extension and loose in flexion. The volar plate has a strong insertion into the proximal phalanx, and with the collateral and accessory collateral ligaments forms three sides of a box, stabilising the joint. In contrast to the PIPJ, the volar plate of the MCP has not got strong check rein ligaments.

The adductor pollicis muscle inserts on to the ulnar sesamoid, and the flexor pollicis brevis and abductor pollicis brevis insert onto the radial sesamoid. These muscles have secondary insertions into the extensor mechanism via the abductor and adductor aponeurosis, providing additional stability.

Injuries to the ligaments of the MCPJ of the thumb are common, often related to sport (50 %) or to work (38 %), and more prevalent in the young male population. The ulnar collateral ligament is injured more often (84 %) than the radial collateral ligament. There is some evidence that joints with a lesser range of movement are more prone to injury [9].

Ulnar Collateral Chronic Instability

Ulnar collateral chronic instability, often referred to as skier's or gamekeeper's thumb, may represent as a pure avulsion of the ligament; or an avulsion of a fragment of bone, usually from its distal insertion in the proximal phalanx; or it may represent a midsubstance rupture or attenuation. The term gamekeeper's thumb was used initially to describe the chronic instability of the MCPJ caused by repetitive trauma as seen in Scottish gamekeepers that often stressed this joint undertaking a manoeuvre to kill a wounded rabbit by twisting its neck [10].

Skier's thumb reflects the incidence of acute UCL injuries associated with a fall while grasping a ski pole in the hand, forcing the thumb into radial deviation; this term is now often used for acute injuries.

Stener described the displacement of the avulsed edge of the ligament, with interposition of the adductor aponeurosis preventing healing of the ligament (Fig. 9.3). If in doubt, an ultrasound scan can often demonstrate the presence of a Stener lesion.

Failure to diagnose or treat a complete tear of the UCL often results in chronic instability. Patients present with pain in the thumb, often exacerbated by activity. The pinch and grip are weak and some tasks, like opening a jar or holding a large bottle, become increasingly difficult. There is often a history of intermittent swelling of the joint and the patients may report a feeling of instability. Crepitus of the joint on movement indicates degenerative changes are present.

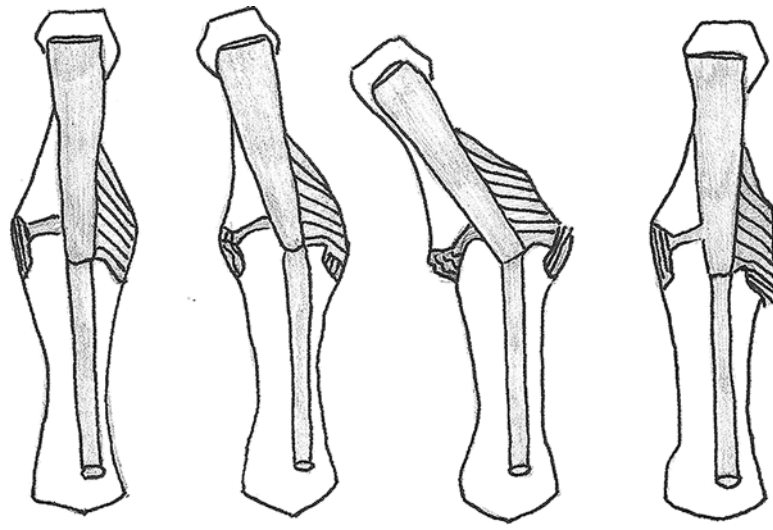


Fig. 9.3 Stener lesion. The adductor aponeurosis covers the ulnar collateral ligament. When the MCPJ is pushed into radial deviation, it can rupture this ligament, and the proximal end of the ruptured UCL can become superficial to the aponeurosis, while the distal insertion is deep to it, preventing healing and causing chronic instability

On examination, it is important to inspect the thumb for volar subluxation of the MCPJ and for radial deviation of the thumb. Pushing the phalanx in a radial direction will cause deviation at the joint, indicating instability (Fig. 9.4). When this is done in flexion, the true collateral ligament is tested, when done in extension, the accessory collateral ligament and the volar plate are tested. Increased deviation compared to the contralateral thumb, and lack of a firm end point are diagnostic of instability. In contrast with an acute injury, in most patients with chronic instability it is not necessary to inject local anaesthetic before examination to elicit instability.

Radiographs may show volar subluxation and radial deviation. It is, however, important to assess any degenerative changes in the joint. Further imaging is not required, since the indication for treatment is based on symptoms and degree of clinical instability.

Treatment

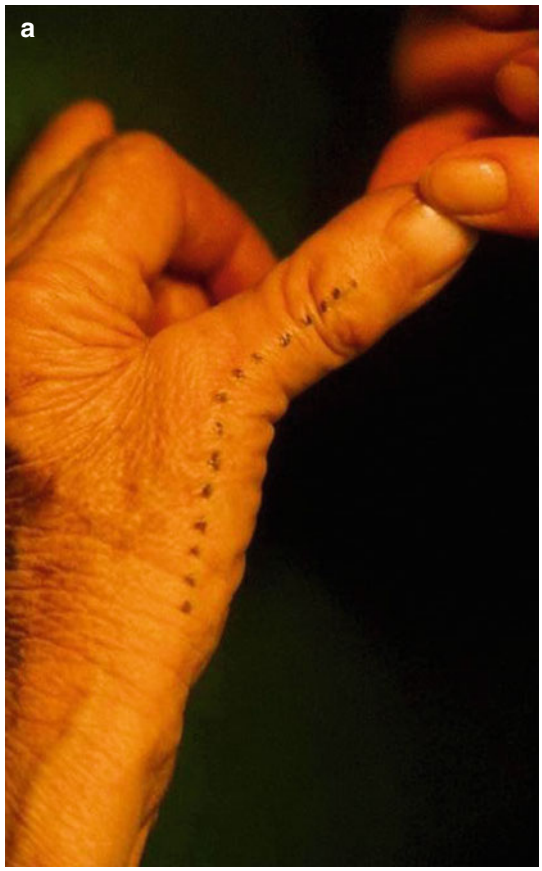
In most cases of symptomatic chronic UCL insufficiency, surgery is indicated. As the period since injury increases, it is less likely that a direct repair is feasible, although if the delay is less than a few weeks it may be possible to identify and repair or reattach the ligament. Degenerative changes or a MCPJ that had very little movement before the injury, would be indications for joint arthrodesis rather that a reconstruction.

Commonly used techniques recommended for MCPJ instability without degenerative changes include reconstruction using a tendon graft or adductor advancement. There are also reports of bone-ligament-bone reconstruction. Some patients, however, may prefer a MCPJ arthrodesis because it is more durable and predictable, although at the cost of losing movement.

Adductor pollicis inserts on the ulnar side of the MCPJ and is a dynamic stabiliser of the joint. Neviaser proposed the advancement of the insertion of the adductor onto the ulnar side of the base of the proximal phalanx, combined with suture of the ligament, to act as a dynamic tendon transfer [11]. The advantage of this technique is that it provides a strong dynamic stabiliser during pinch that prevents radial deviation at the MCPJ. This technique involves an incision on the ulnar side of the MCPJ, protecting the sensory branches of the radial nerve, exposing the ulnar side of the joint. The adductor aponeurosis is incised and reflected volarly and after identifying the scarred ligament and capsule, the joint is exposed. The adductor pollicis tendon is then detached from its insertion in the ulnar sesamoid.

Provided there are no degenerative changes, the ligament and capsule are reefed. The adductor tendon is attached to the base of the proximal phalanx with the use of a bone anchor or a pull-out wire. In the postoperative period, the thumb is immobilised for 4 weeks in a cast or splint, thereafter range of movement exercises are started.

An alternative procedure is reconstruction using a free tendon graft. The MP joint is exposed through a mid-axial incision to expose its ulnar aspect. The adductor aponeurosis is mobilised and a hole is drilled on the ulnar side of the base of the proximal phalanx, in the area of insertion of the UCL. A tendon graft is obtained, usually palmaris longus, and a figure of eight suture is placed at the end of the graft. The two ends of the suture are placed on needles and passed through the tunnel at the base of the proximal phalanx and through the radial cortex where sutures are tied over a button. Alternatively, a bone anchor or an interference screw can be



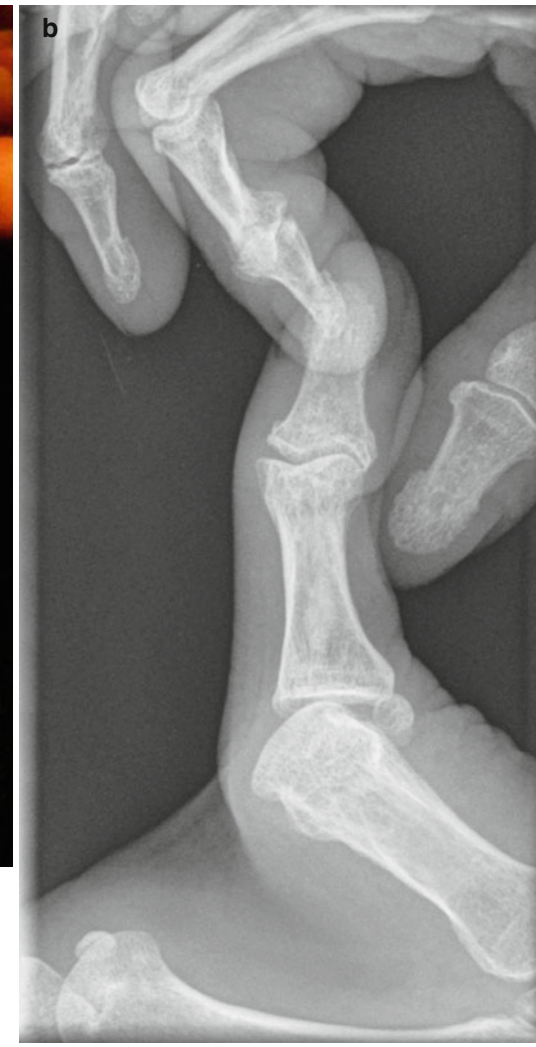


Fig. 9.4 (**a**) Testing the UCL of the MCPJ clinically shows increased radial deviation, without a firm end point. Stressing the MCPJ under fluoroscopy shows the displacement in the left thumb, diagnostic of ulnar collateral ligament insufficiency (**b**). Compare with the right side, with a normal competent ulnar collateral ligament (**c**). It is important when testing under fluoroscopy to try to avoid radiation to the fingers of the examiner, as far as possible

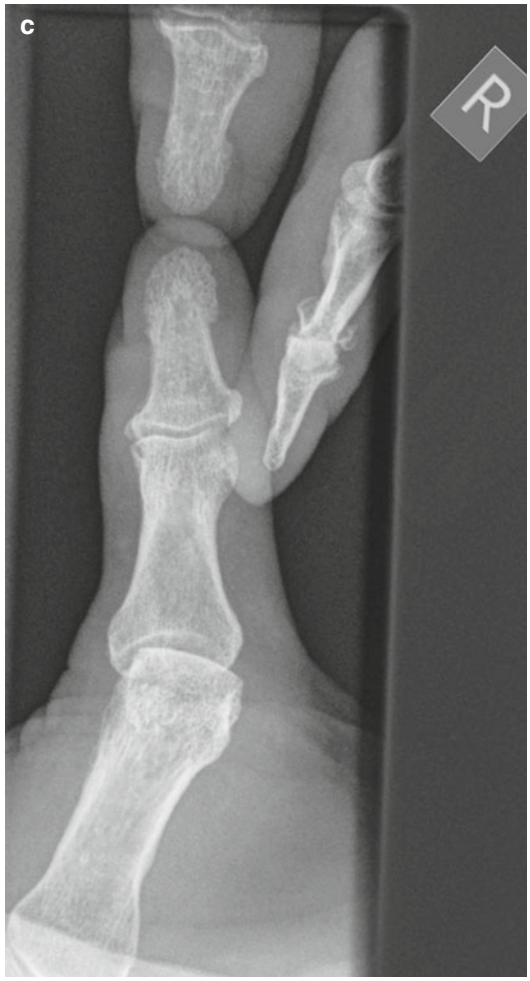


Fig. 9.4 (continued)

used. The ligament is identified and dissected and used to suture the tendon graft to the metacarpal. If the remains of the ligament are not strong enough, the graft can be passed through a tunnel in the metacarpal head (Fig. 9.5). The adductor aponeurosis is sutured over the repair. A thumb spica is recommended for 4 weeks and a splint for a further 5 weeks.

The main contraindication to these reconstructive procedures is osteoarthritis of the joint. A relative contraindication is marked volar subluxation or supination of the joint, which may not be improved by these procedures. On occasions MCPJ instability is diagnosed as an incidental finding, with very few symptoms; no surgical treatment is required in these cases.

Radial Collateral Chronic Instability

Although injuries to the RCL of the MCPJ are less common (16 %), they can present with similar symptoms and disability. They are caused by forced adduction of the thumb or by a torsional force on the flexed thumb. The principles of diagnosis and management are similar to those described above.

There are some anatomical differences between the radial and the ulnar sides of the joint. On the radial side, the abductor aponeurosis is wider, and there is little potential to have a lesion, similar to the Stener on the ulnar side of the joint. The RCL is torn in about equal frequency from its proximal and distal insertions and, it is more common that it presents as a mid-substance tear. A rupture of the RCL allows the joint to rotate, pivoting around the intact UCL, giving the typical appearance of a dorso-radial prominence of the metacarpal head. The tear often extends into the dorsal capsule.

The clinical and radiological assessment is similar to that described for the UCL. Plain radiographs are important to assess joint alignment, volar subluxation and any signs of joint degeneration.

Treatment

RCL tears seem to develop chronic instability more often than UCL tears. In the chronic situation, the surgical options include repair of the ligament, reconstruction or arthrodesis. Repair is often possible up to 6 weeks after injury, but becomes increasingly difficult after that.

Reconstruction can be achieved with abductor advancement, similar to the technique describe by Neviaser for the UCL (Fig. 9.6), or again with a free tendon graft.

Chronic Volar Instability of the Thumb

Chronic instability of the thumb into hyperextension is often related with generalised joint laxity. These patients can maintain the MCPJ flexed

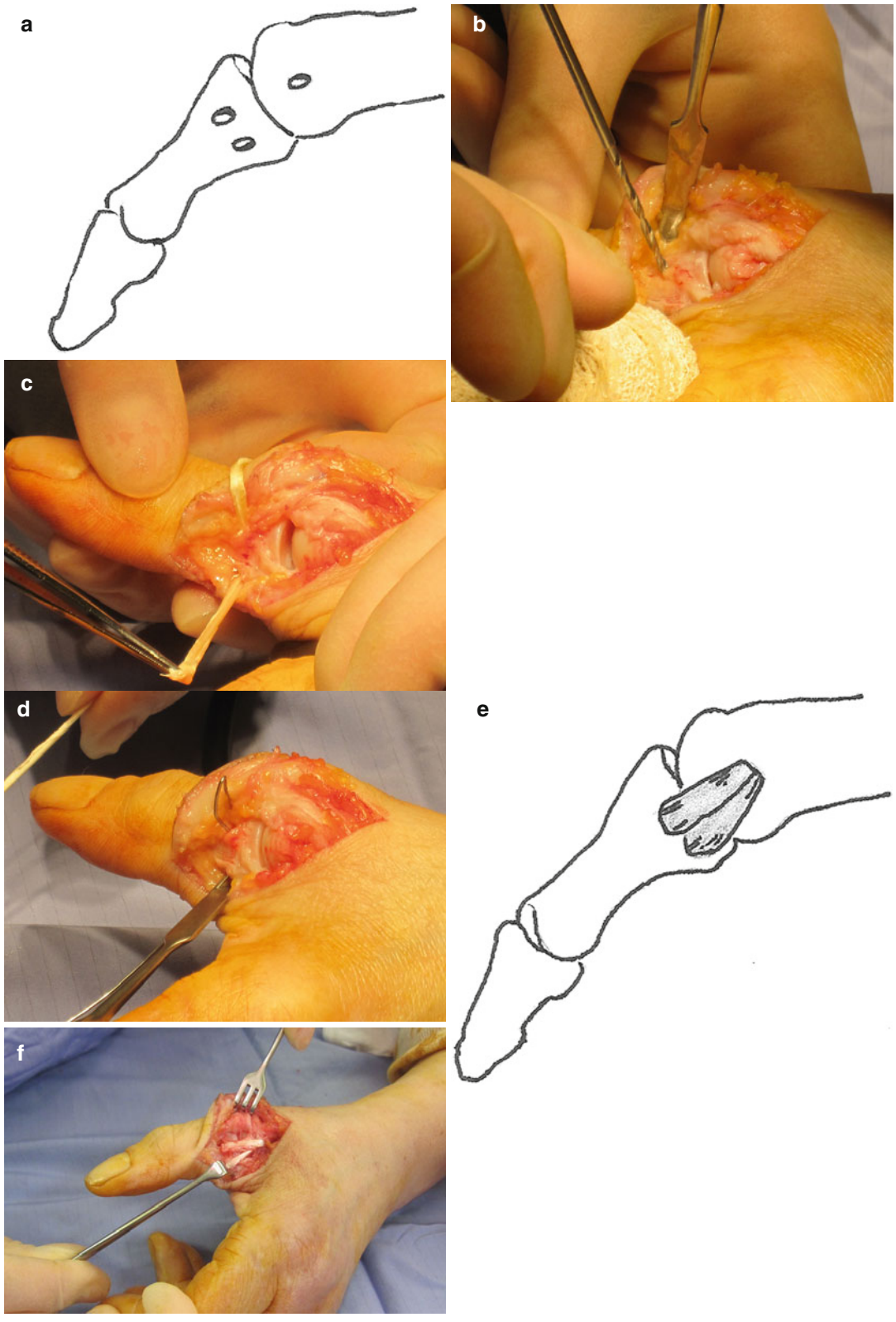
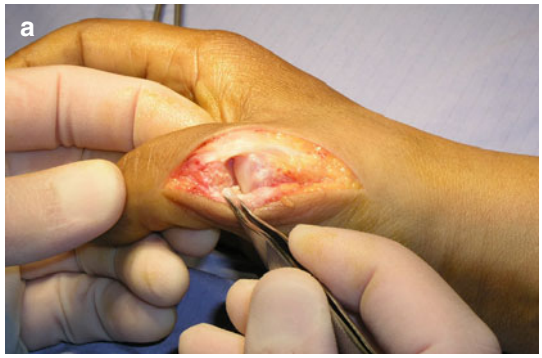


Fig. 9.5 (**a**–**f**) Stabilisation of MCPJ using a tendon graft. *Two holes* are drilled in the proximal phalanx to make a tunnel to pass the tendon graft, and *one transverse hole* is drilled into the metacarpal to anchor the graft. The graft is passed through the tunnel in the phalanx. The graft is anchored in the metacarpal using an interference screw



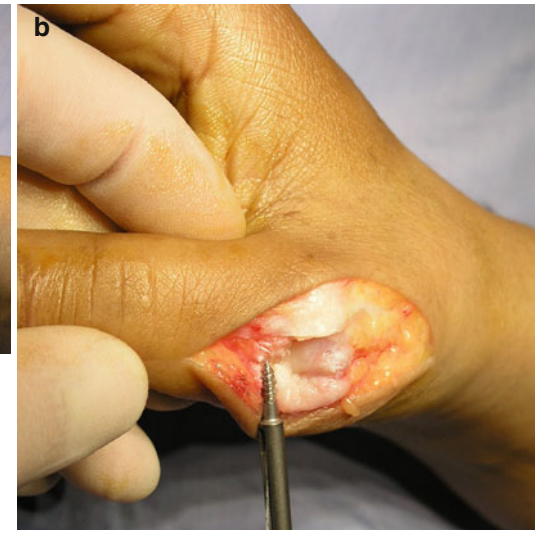


Fig. 9.6 Exploration of the radial collateral ligament of the MCPJ shows a chronic avulsion (**a**). In this case the ligament could be reattached using a bone anchor, and an advancement of the abductor pollicis tendon was carried out to provide dynamic stability (**b**)

during use, but they are able to subluxate the thumb by hyperextending this joint, often voluntarily. Rarely volar instability of the thumb can be associated with systemic conditions e.g. collagen disease, such as Ehlers Danlos, neuromuscular disorders or secondary to osteoarthritis at the base of the thumb.

Post-traumatic instability presents with a history of injury, pain and swelling of the MCPJ, tenderness on the volar side of the joint and pain on hyperextension of the joint. Patients report pain when pinching or grasping.

Treatment

Eaton reported volar capsulodesis for patients with MCPJ hyperextension of more than 30° in the context of osteoarthritis of the CMCJ [12]. Sesamoid arthrodesis to the metacarpal head achieves the same aim of limiting extension and good results have been reported in patients with osteoarthritis and with cerebral palsy [13]. In addition, a technique to advance the insertion of abductor pollicis brevis and flexor pollicis brevis by 1.5 cm onto the proximal phalanx has been reported with good results [14]. Finally, formal arthrodesis is also an option.

Clinical Pearl

Instability of the thumb MCP joint is probably the commonest chronic dislocation in the hand. Diagnosis is made by clinical examination, although stress x-rays can be useful. It is also important to exclude secondary osteoarthritis. Surgical reconstruction using tendon augmentation, often results in a good outcome.

Chronic Instability of the MCPJ of the Fingers

Although a comparatively uncommon injury, radial collateral ligament injuries of the metacarpophalangeal of the fingers are more common than injuries to the ulnar collateral ligament, which are very rare. Chronic instability refers, for the most part, to insufficiency of the radial collateral ligament, occurring gradually after an injury. It may also contribute to the development of degenerative changes within the joint.

Patients present with pain, worse on gripping, and often intermittent swelling. The finger may

appear deviated towards the ulnar side, with some rotational deformity towards pronation. Clinical testing reveals excessive lateral deviation without a firm end point. Radiographs may show a ligament avulsion. It is also important to assess any degenerative changes that may be responsible for some of the symptoms. Ultrasound and MRI scans may confirm the diagnosis and the extent of the injury. Finally and occasionally, arthroscopy of the MCPJ can be helpful to confirm the diagnosis and inspect the joint surface for any degenerative changes (Fig. 9.7).

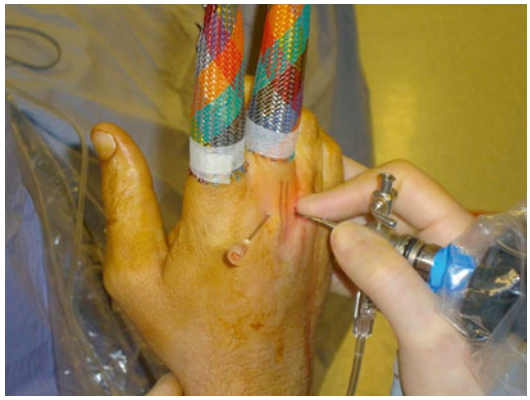


Fig. 9.7 Arthroscopy of the MCPJ is useful to assess the ligaments, and determine if a ligament is avulsed from its proximal or its distal insertion. Further to that assessment of the joint surface is important to exclude degenerative changes

In the acute situation, conservative treatment by splinting the MCPJ in extension may allow the ligament to heal. In cases of gross instability or deviation of the finger at rest, surgical treatment is recommended. In chronic cases, that is 3 months post-injury, it is very unlikely that splinting will eliminate the symptoms, and surgery is preferred (Fig. 9.8).

On some occasions, it may be possible to reattach the ligament, but this is often not possible and a ligament reconstruction, similar to those used for the collateral ligaments of the MCPJ of the thumb, is required. This is very applicable to the radial collateral ligament of the

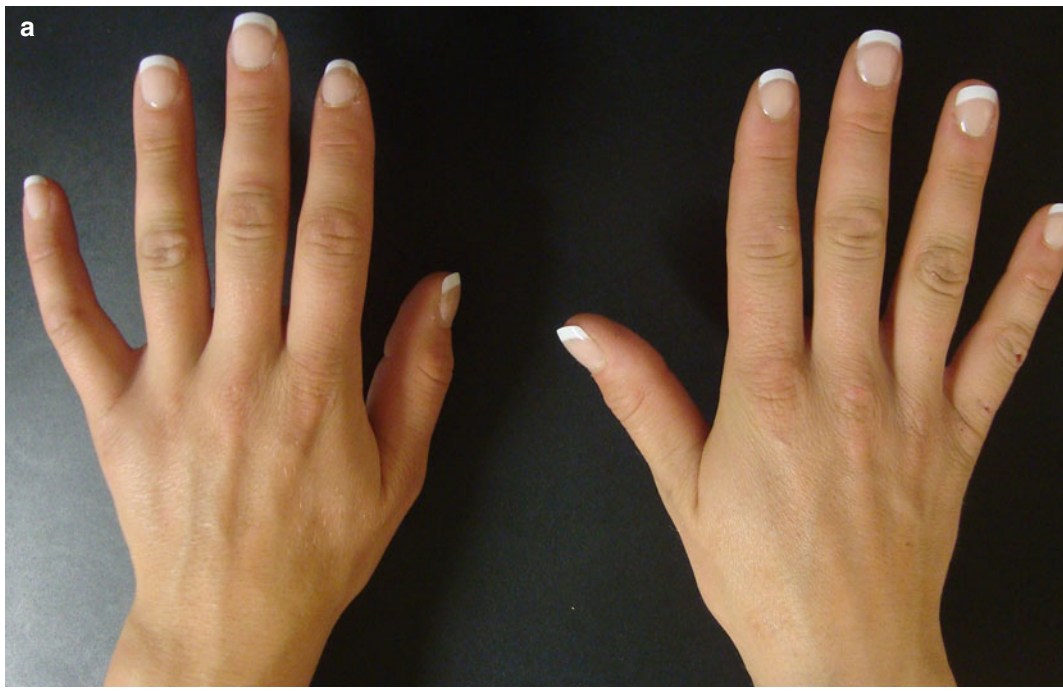
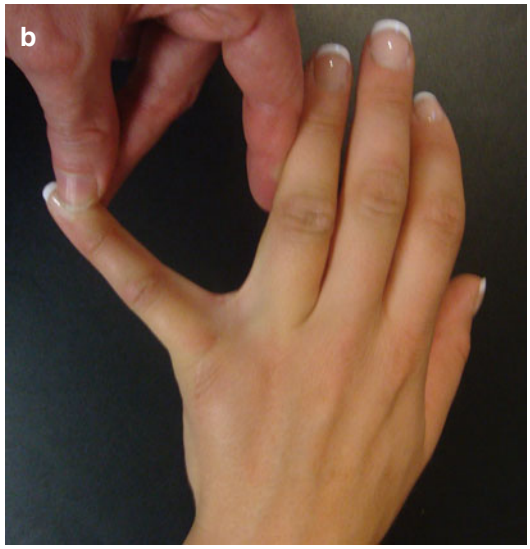


Fig. 9.8 This 27 year old lady sustained an injury to the MCPJ of her left little finger 6 months previously. Note the slight increased abduction of the little finger (**a**). Testing the radial collateral ligament shows increased ulnar deviation of the little finger without a firm end point, diagnostic of radial collateral ligament insufficiency (**b**). At surgery, avulsion of the ligament was confirmed (**c**)



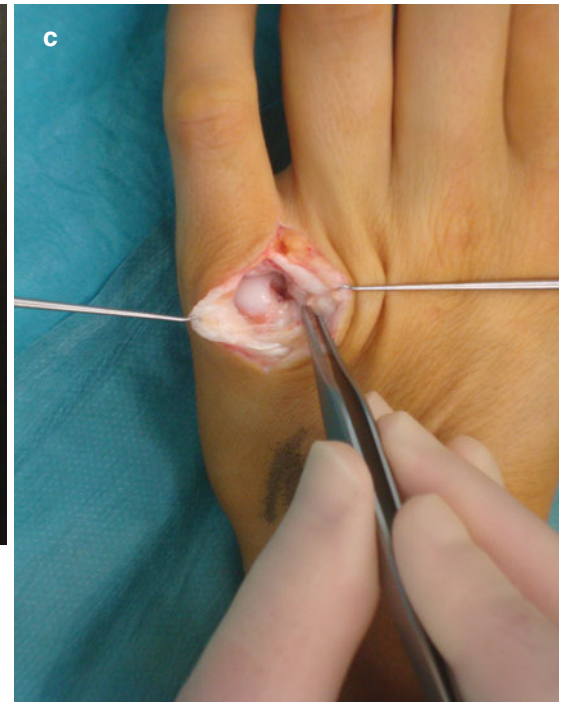


Fig. 9.8 (continued)

index finger, but technically more difficult in the other digits because access is restricted by the adjacent digit.

The Interphalangeal Joints

The PIPJ is a hinge joint (ginglymus) with a normal arc of flexion-extension of approximately 100°. The bicondylar geometry makes it quite stable to a shearing lateral force, even in the absence of lateral capsular restraints. The joint is further stabilised by the collateral ligaments, accessory collaterals and volar plate. The main stabiliser, however, are the collateral ligaments, inserted eccentrically on the concave area of the lateral face of the condyle of the proximal phalanx, and shaped like a fan, attached distally on the volar three-quarters of the sides of the base middle phalanx (Fig. 9.9). As well as wide, this

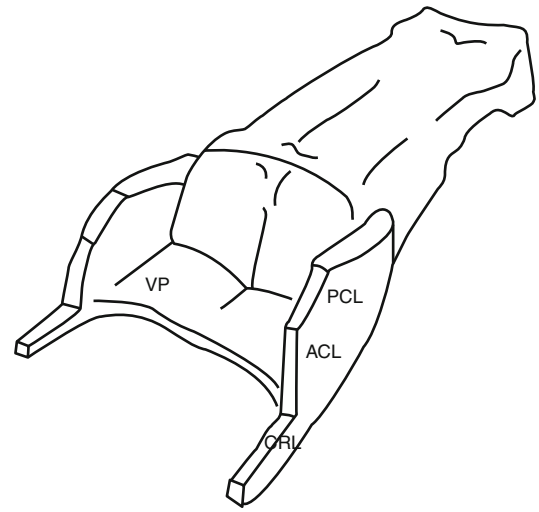


Fig. 9.9 Ligaments of the PIPJ. The proper collateral ligament (*PCL*), accessory collateral ligament (*ACL*) and the volar plate (*VP*) of the PIPJ form three sides of a box, in continuity with the middle phalanx, to contain the condyles of the proximal phalanx. The insertion into the proximal phalanx is through the check rein ligaments (*CRL*)

insertion onto the middle phalanx is long, extending into the metaphysis. The collateral ligament is a strong and thick structure, measuring 35 % of the total width of the joint [15], and its fibres are parallel to the middle phalanx in every position of the joint [16].

The volar plate is a complex capsular structure which acts as a volar restraint, limiting interphalangeal extension. In addition, it contributes to lateral stability of the joint in full extension. Distally, the volar plate inserts strongly on the volar lateral corner of the middle phalanx. The central 80 % is meniscoid, and forms a volar extension of the joint surface of the middle phalanx. Proximally, the volar plate forms the check rein ligaments that insert in the proximal phalanx, as well as joining the A2 pulley [1].

The accessory collateral ligament joins the volar side of the true collateral ligament, to the lateral side of the volar plate. Its fibres are tight in full extension, and relax in flexion to cup the volar part of the condyles of the proximal phalanx, which is wider than the dorsal side as a consequence of the trapezoid cross section of the distal part of the proximal phalanx.

Ligamentous injuries in the hand most often involve the PIPJ. Frequently these injuries are caused by ball handling sports like cricket, basketball, rugby or baseball, but are also common at work, in the home and as part of hand polytrauma. Patients present with a painful, bruised, swollen finger, following trauma. The finger will look clearly deformed in cases of dislocation. Appropriate radiographs must be obtained of the finger (Figs. 9.10, 9.11 and 9.12).

Dorsal Chronic Instability

Dislocations of the PIPJ can be dorsal, lateral or volar. Dorsal dislocations are usually caused by hyperextension with a component of axial compression [17]. The volar plate is usually avulsed from its insertion on the middle phalanx in a pure dislocation, but more often there is an avulsion fracture of the base of the middle phalanx.

Dorsal dislocations have been classified into three groups:

I. A hyperextension deformity with subluxation (joint surfaces still in contact)
II. With a bayonet deformity (joint surfaces not in contact) (Fig. 9.12)
III. Fracture dislocation.

Most type I or type II dislocations when treated within the first few days of injury can generally be reduced by closed methods and are usually stable after reduction.

Chronic dislocation or instability of the PIPJ require release of the contractures around the joint, together with reduction and reconstruction of damaged structures. Due to the technical difficulties of achieving this in one procedure, some authors recommend staged treatment with the application of an external fixator to distract the joint for a few weeks, followed by open reconstruction and stabilisation. If the treatment is carried out in one stage, it is done surgically through a volar approach, raising the A3 pulley to one side. Thereafter, the release of all tight structures, including the collateral ligaments is required in order to reduce the joint. A repair of the volar plate may be required to prevent re-dislocation [18]. On occasions a small dorsal approach may be necessary to release adhesions between the extensor tendon and the proximal phalanx.

In late presenting cases of a fracture dislocation, it may not be possible to fix the fracture of the middle phalanx due to its size or comminution. In these cases, a volar plate arthroplasty, replacement arthroplasty or reconstruction with an osteochondral graft have all been advocated. Using an osteocartilaginous graft taken from the proximal part of the hamate bone of the same hand to reconstruct the volar aspect of the base of the middle phalanx seems to provide a stable joint with a good range of movement [19]. However, recurrent instability can be a very frustrating problem for both the patient and the surgeon (Fig. 9.13).

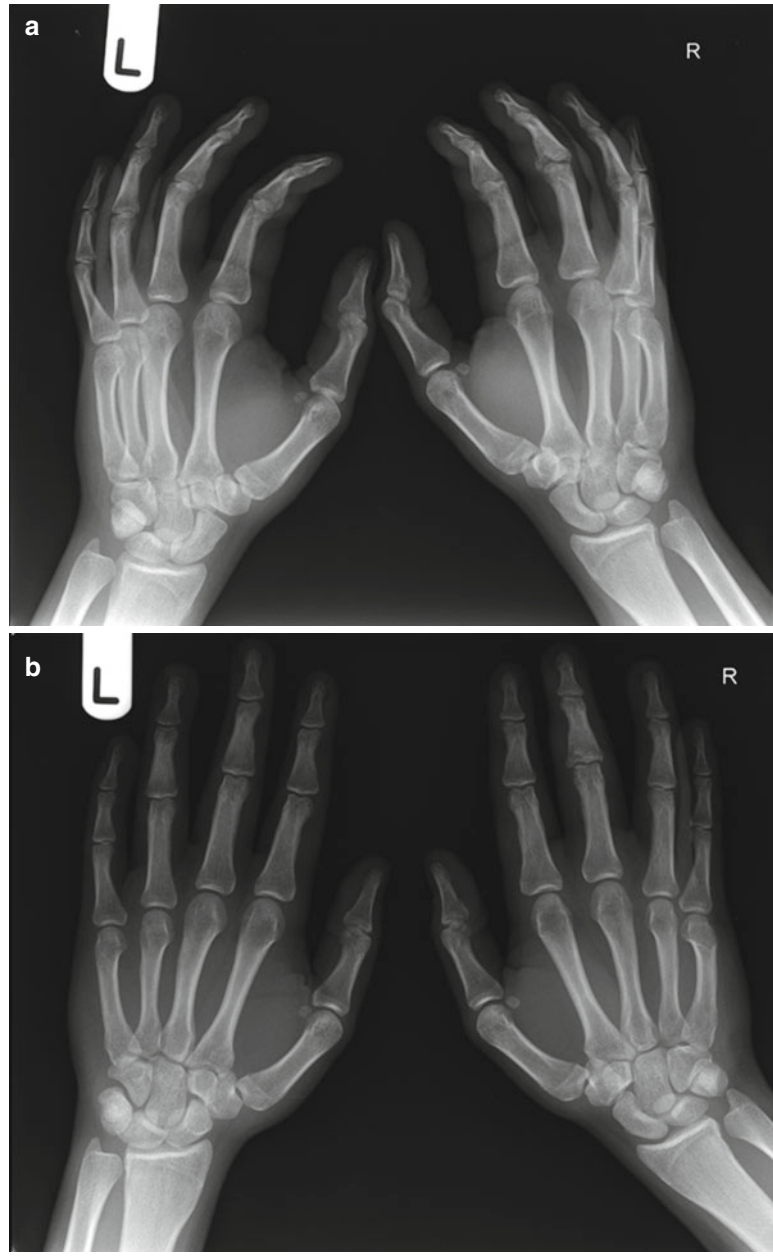


Fig. 9.10 A 23 year old man presented with pain and swelling in his right middle finger PIPJ after a fall onto both hands. Oblique (**a**) and posterior-anterior (**b**) radiographs of the hand were obtained. Can you diagnose the problem?

Hyperextension Deformity

Injuries to the volar plate of the PIPJ may result in hyperextension at his joint, causing a tendency to "swan necking", which can present with clicking of the lateral bands, and occasionally with a history of the finger locking in extension (Fig. 9.14). Most surgical treatments attempt to avoid hyper extension by creating a volar restraint, for example using half of the flexor digitorum superficialis

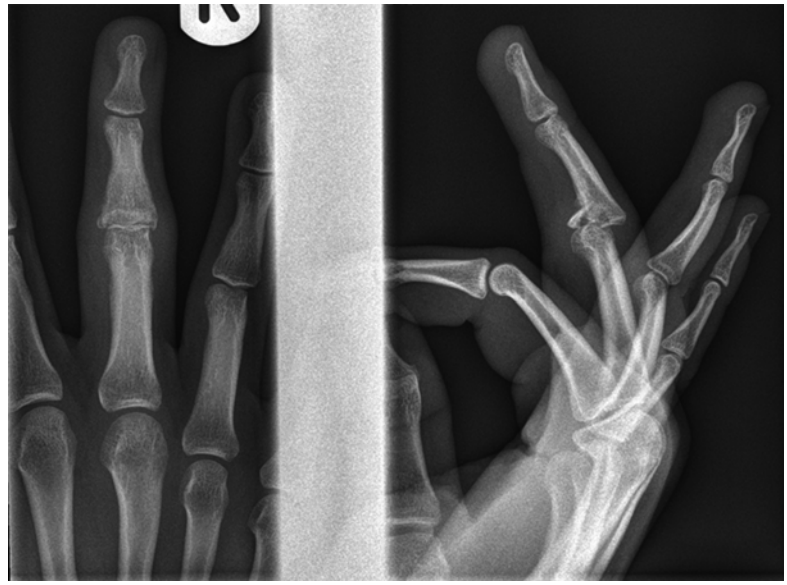


Fig. 9.11 Same patient as in Fig. 9.10. Now with radiographs of the painful and swollen right middle finger. It is now easy to diagnose a dorsal fracture-dislocation of the PIPJ. The diagnosis was missed initially and the patient underwent surgery 6 weeks post injury

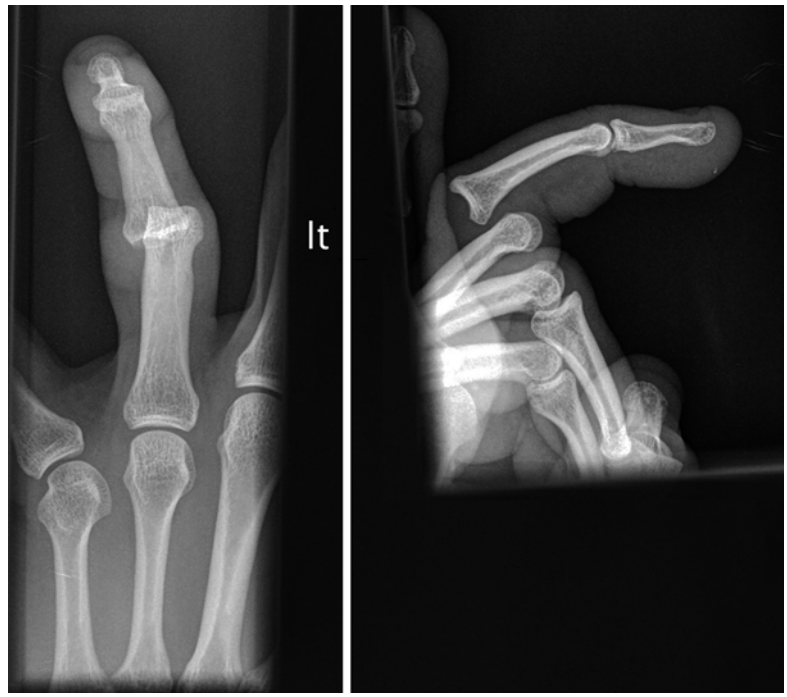


Fig. 9.12 Dorsal dislocation of the PIPJ. There is no contact between the joint surfaces, and no visible fracture. This is therefore a type 2 dislocation

as a tenodesis, or by rebalancing the forces in the finger by transferring one of the lateral bands volar to the centre of rotation of the joint, as described by Zancolli. Good results have also been reported with reattachment or advancement of the volar plate [20].

Lateral Chronic Instability

Pure lateral force applied to a finger can cause a sprain of the ligaments, or if excessive and prolonged, a rupture of the ligaments and a dislocation, although this is a rare injury. On

examination of the joint, more than 20° of deformity with lateral stress, indicates complete lateral collateral ligament disruption and probably partial avulsion of the insertion of the volar plate [21].

Screening under fluoroscopy after local anaesthetic infiltration is extremely useful, together with a comparison to the same joint in the other hand.

Volar Chronic Instability

Volar dislocation of the PIPJ is an uncommon injury. It can be caused by volar directed force, but more often by a rotational force to the finger, for example when the finger gets caught in a tumble dryer that is still spinning, or when leading a horse holding the reins between two fingers, and the horse suddenly pulls. As rotation is applied, the condyle of the proximal phalanx ruptures the space between the central slip and the lateral band of the extensor mechanism. The central slip is usually avulsed. The collateral ligament ruptures and the volar plate is also avulsed (Fig. 9.15). The PIPJ tends to be very unstable, even after reduction.

The management of chronic volar dislocation of the PIPJ again requires the release of a contracted joint and reconstruction of the damaged extensor mechanism, as well as the repair or reconstruction of the collateral ligament.

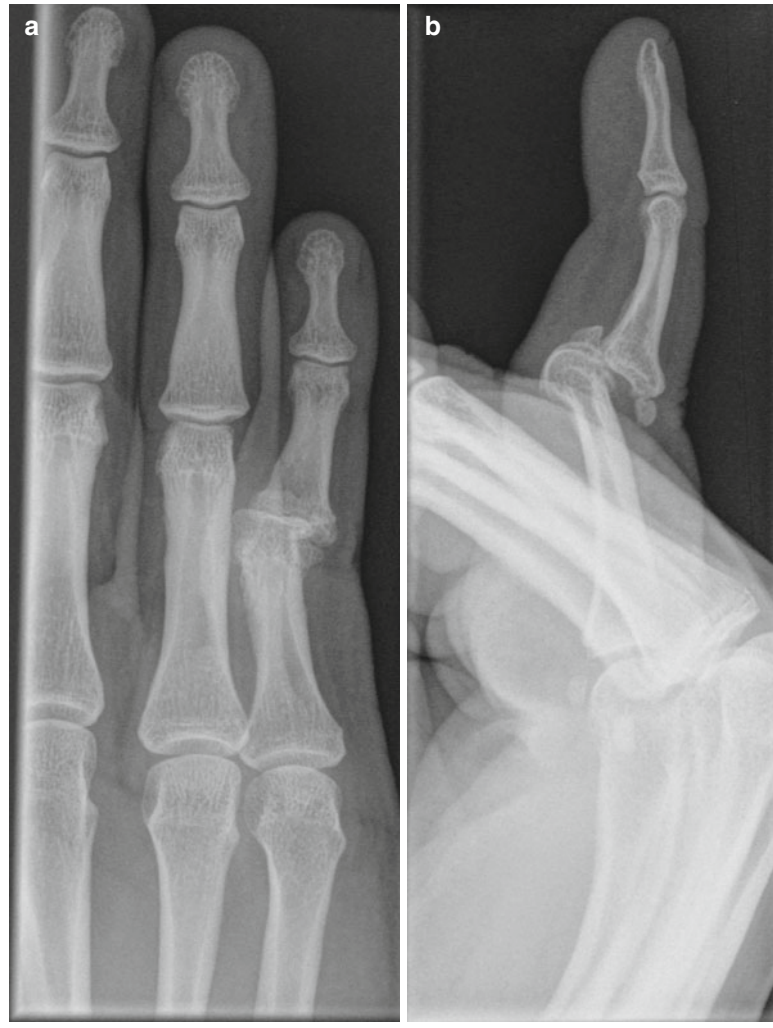


Fig. 9.13 Recurrent instability. This patient presented after an acute injury with a dorsal dislocation of the PIPJ of his little finger. He had a previous injury to the same finger 1 year previously (**a**, **b**). Initially this was treated conservatively a dorsal splint with slight flexion (**c**). Shortly after removal of the splint the PIPJ dislocated again (**d**). Stabilization with a volar plate reattachment using a bone anchor was carried out. Unfortunately the instability recurred again shortly after the surgery (**e**, **f**)

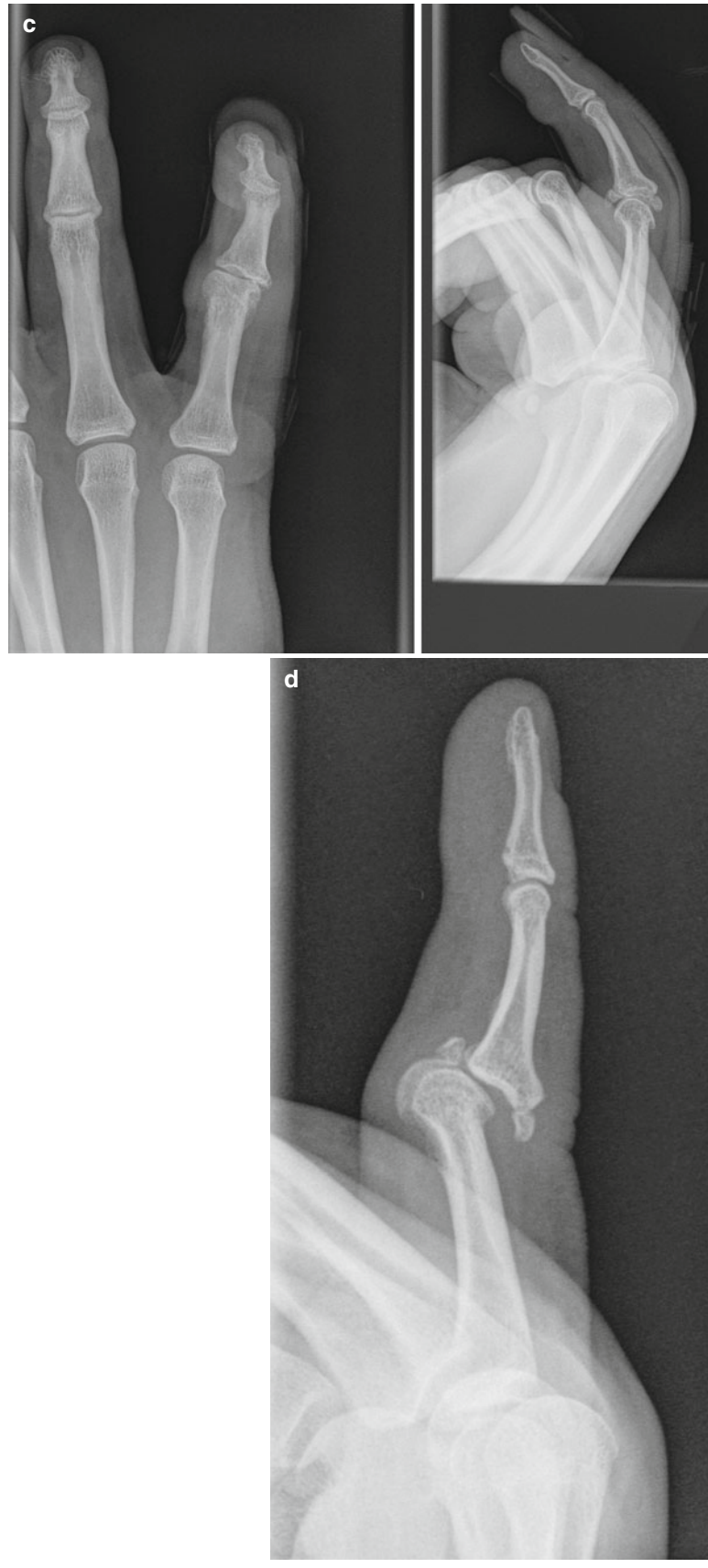


Fig. 9.13 (continued)

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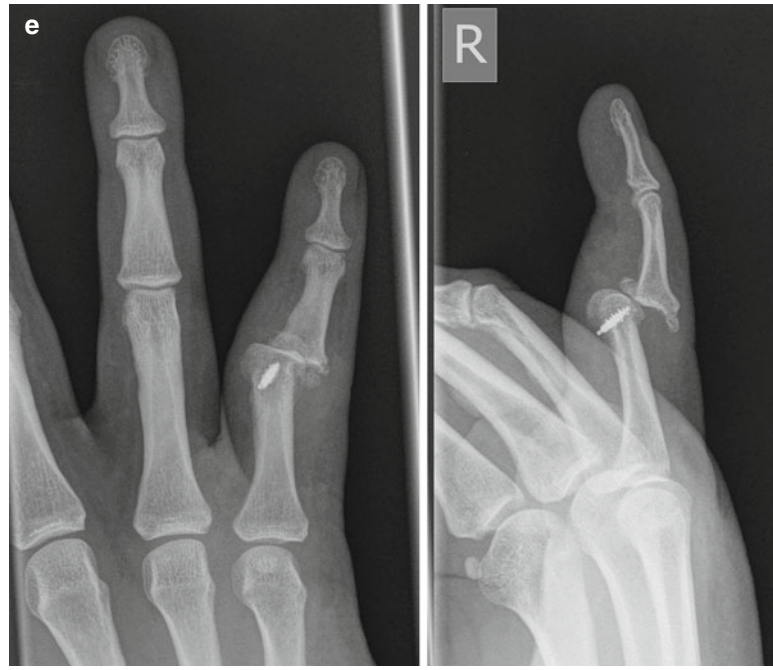

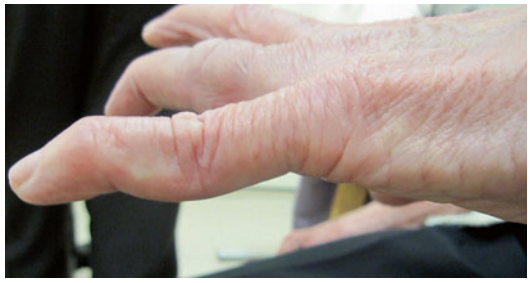

Fig. 9.14 Swan-neck deformity secondary to a volar plate injury. This patient complained of clicking. The dorsally subluxated lateral bands of the extensor mechanism snap as they go from dorsal to volar

Clinical Pearl

Injuries to the proximal interphalangeal joint are relatively common, particularly among sportsmen. Despite that, chronic instability is relatively rare. Treatment would involve both reduction and stabilization, either as a one or two stage procedure. Results, however, can be quite variable. Long term complications including secondary osteoarthritis can be treated by arthrodesis.

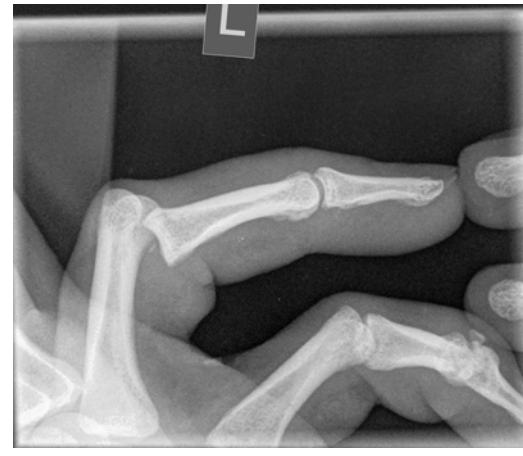

Fig. 9.15 Volar dislocation of the PIPJ. These are very unstable injuries

Distal Interphalangeal Joint and Interphalangeal Joint of the Thumb

Isolated soft tissue injury of the DIPJ is uncommon. Instability of the distal interphalangeal is most often related to degenerative changes in the joint due to osteoarthritis. At the distal segment of the finger, sensation and stability are more

important than movement. As a consequence, for most patients, the commonest procedure performed is an arthrodesis of the joint.

Summary

Chronic instability of the joints of the finger and thumb are fortunately not common. More often they are seen in the proximal interphalangeal joint of the finger and metacarpophalangeal joint of the thumb, where diagnosis can be easily made on clinical examination. Instability at the carpometacarpal joint of the thumb and metacarpophalangeal joint of the fingers are more difficult to diagnose and often require a degree of suspicion.

Stress x-rays can be useful, however, increasingly MRI scans are becoming more sensitive which as well as confirming the diagnosis, can actually identify the structures that are torn.

Optimum treatment should be undertaken at the time of the initial injury. This often results in the best outcome. The results of secondary reconstruction can be variable, particularly at the proximal interphalangeal joint of the finger. Results at the MCP joint of the thumb are more reliable.

Long term complications include continuing instability and secondary osteoarthritis. These are best treated by arthrodesis or occasionally a joint replacement with ligament reconstruction.

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Chronic Carpal Instability

10

Marc Garcia-Elias

Keywords
Carpus • Instability • Scapholunate • Lunotriquetral • Wrist • Kinematics • Ligament injury • Carpal dysfunction • Osteoarthritis • Grip strength • Range of motion • Ligament reconstruction • Midcarpal instability • Tendon reconstruction • Capsulodesis

Introduction

Most wrists with an unsolved bone or ligament injury evolve into permanent, dysfunctional and often painful malalignment. When progressively loaded, a malaligned carpus no longer dissipates its internal stresses across central, cartilage covered joint surfaces, but tends to shift loads towards the periphery of the joint, inducing progressive osteoarthritis. This often results in capsular retraction and further wrist dysfunction. When an unstable wrist moves, sudden changes in bone positioning may occur (wrist clunking), which can also result in painful synovitis and muscle protective contractures. Indeed, the chances of an unstable wrist developing painful osteoarthritis are high, not only as a result of abnormal transfer of loads (dyskinetics) but also because the presence of dysharmonic motion (dyskinematics) [1, 2].

Carpal instability may be the consequence of a large spectrum of injuries or diseases. Congenital anomalies, avascular necrosis, infections or inflammatory arthritis, may all generate wrist instability. In fact, any condition in which the shape and/or position of the carpal bones is modified, may result in carpal instability.

There are four major patterns of carpal instability: (i) carpal instability dissociative (CID), where there is a major disconnection of bones within the same carpal row, either by fracture or ligament disruption; (ii) carpal instability non-dissociative (CIND), where there is no disruption between bones of the same row, but a dysfunction between the radius and the proximal row and/or between the proximal and distal rows; (iii) carpal instability complex (CIC), where features of both CID and CIND types are present in the same wrist; and (iv) carpal instability adaptive (CIA), where wrist malalignment is not caused by an injury within the wrist, but by a problem outside the wrist [3]. The carpal

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malalignment often seen as an adaptation to a malunited distal radius fracture is a good example of a CIA pattern. In this chapter, only the dissociative type of carpal instability (CID) will be discussed.

Dissociative instabilities are frequent conditions resulting from the rupture of the linkage between bones of the same row. Within the proximal row, the disconnection may predominantly affect the scaphoid and lunate bones, but it may also derive from lunotriquetral dissociation, from an unstable scaphoid fracture or from an advanced Kienböck's disease [4]. In this chapter, only the chronic, posttraumatic carpal instability, secondary to ligament rupture between bones of the proximal row without osteoarthritis will be discussed. The acutely injured wrists inducing instability, as well as the osteoarthritic wrists secondary to an unsolved ligament injury, are covered elsewhere.

Scapholunate Dissociation

The term "scapholunate dissociation" (SLD) has long been used to describe one of the most frequent carpal instabilities, the one that results from the loss of the linkage between the scaphoid and the lunate. With time, if the initial lesion is not properly solved, SLD may become a more extended wrist instability problem [3, 5]. The condition may emerge from either an isolated injury or associated with other local trauma, such as a distal radial fracture or a displaced scaphoid fracture. When the ligaments attached to both ends of the scaphoid have failed, and the bone has collapsed into flexion and pronation, the condition is known as rotatory subluxation of the scaphoid [6, 7]. In less advanced cases, where only the proximal ligaments are disrupted, the scaphoid may remain normally aligned with the distal ligaments preventing collapse of the bone [8]. In between the two extremes, a spectrum of conditions are possible, some exhibiting carpal malalignment, some appearing as normally aligned wrists [7, 9].

Presentation and Diagnosis

Post-traumatic SLD may be diagnosed acutely, immediately after the accident, or in the chronic phase. The first option usually comes after violent trauma, such as a fall from a height or a motorcycle accident, while in the chronic phase the patient may not even recall a specific trauma responsible for the symptomatic wrist. In the first case, the diagnosis may be obvious; whilst in the second, identification of a precise carpal dysfunction may not be easy [10]. Except for open dislocations, the external appearance of most wrist with SLD is seldom remarkable. Swelling is moderate and the overall range of motion may not be substantially decreased. Grip and pinch strength may be reduced by actual loss of muscle strength, or by inhibition caused by pain.

Palpation for areas of tenderness is most useful in the diagnosis of chronic instability [10]. If sharp pain appears by flexing the wrist and pressing the area distal to Lister's tubercle, the probability of either scapholunate (SL) injury or localized synovitis is high.

Passive mobilization of the SL joint may establish the presence of abnormal motion or crepitus. A positive "Scaphoid shift test", as described by Watson, is not diagnostic but it may help in suspecting an SLD [7]. If the SL ligaments are torn or substantially lax, pressure on the palmar scaphoid tuberosity, while the wrist is moved from ulnar deviation to radial deviation, forces the proximal pole to subluxate dorsally out of the radius. What makes this test positive is not the induction of pain on the dorsoradial aspect of the wrist, but the presence of a dorsolateral scaphoid subluxation. When pressure is released, a clunk may indicate self-reduction of the scaphoid over the dorsal rim of the radius.

Summary

The diagnosis of scapho-lunate ligament injury is principally by suspicion.

The most useful diagnostic took is localized tenderness, supported by a "positive scaphoid shift test" and ballottement.

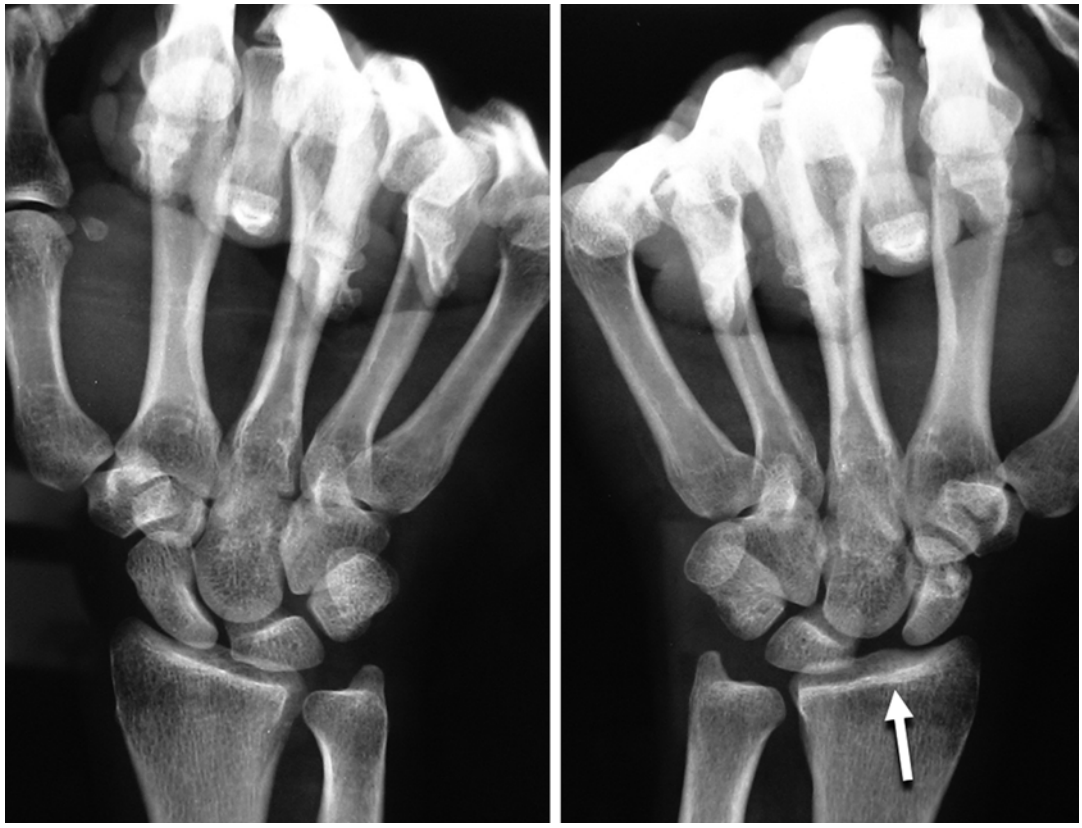

Fig. 10.1 If the SL ligaments are normal, closing the fist should not cause separation of the scaphoid and lunate bones. As compared to the contralateral side, any asymmetrical SL gap (*arrow*) greater than 5 mm is diagnostic of SLD

SLD can be suspected by careful analysis of standard radiographs. Three essential features are usually found: (1) increased SL gap, the so-called Terry Thomas sign: As compared with the contralateral side, any asymmetrical gap greater than 5 mm is said to be diagnostic of SLD (Fig. 10.1), (2) Scaphoid ring sign: When the scaphoid has collapsed into flexion (rotator subluxation of the scaphoid), the scaphoid tuberosity is projected in the posteroanterior X-ray, in the form of a radiodense ring over the distal two thirds of the scaphoid, (3) Increased SL angle: If in the lateral view, the SL angle is greater than 60°, an SLD is likely [11, 12].

Injecting dye in the joint and analyzing the wrist with tomography or MRI scans may be useful in further defining partial tears of the SL ligaments or osteochondral defects. Notwithstanding, the gold standard technique in the diagnosis of intracarpal derangements, particularly when the diagnosis is still unclear, is arthroscopy [9, 13]. Information for a correct arthroscopic diagnosis of SLD dissociation is provided elsewhere in this book.

Treatment of SLD

Treatment of chronic SLD is not always predictable and often unsatisfactory [10, 11]. On the one hand, because the SL ligaments sustain considerable tension, most ligament reconstructions will deteriorate with time. In addition by the time the injury is recognized and treated the secondary stabilizers of the scaphoid may have also degenerated, thus limiting the chances for a successful surgical stabilization. Needless to say, treating the injury in the acute phase is more

Table 10.1 Staging of scapholunate dissociations **According to Garcia-Elias et al. [16]**

SLD stage	1	2	3	4	5	6
Is there a partial rupture with a normal dorsal SL ligament?	Yes	No	No	No	No	No
If ruptured, can the dorsal SL ligament be repaired?	Yes	Yes	No	No	No	No
Is the scaphoid normally aligned? (normal ≤45°)	Yes	Yes	Yes	No	No	No
Is the carpal malalignment easily reducible?	Yes	Yes	Yes	Yes	No	No
Are the cartilages at both RC and MC joints normal?	Yes	Yes	Yes	Yes	Yes	No

rewarding than dealing with a chronic case [13–15]. What follows is the description of the staging system we recommend to use in such cases [16], followed by a review of surgical alternatives proposed for the chronic, non-arthritic SLD.

When deciding treatment for an SLD, aside from the patient's age, health status, and professional and/or vocational demands, five factors are to be considered:

1. Integrity of the dorsal SL ligament: It is important to establish whether the dorsal SL ligament is intact. Arthro-CT scans or MRI's may be of help, but most often arthroscopy is the one that verifies the best the status of the important dorsal SL ligament.
2. Healing potential of the disrupted ligaments: If there is a mid-substance ligament rupture, with irregular ends, a poor healing potential is likely. By contrast, if there is an avulsion type of rupture with the ligament intact, the injury may heal if properly reattached.
3. Status of the secondary scaphoid stabilizers: It is important to evaluate whether the secondary scaphoid distal stabilizers (STT and SC ligaments) are functional or not. A radioscaphoid angle beyond 60° is indicative of a distal ligament disconnection.
4. Reducibility of carpal malalignment: Chronic SL dissociations often develop fibrosis at the level of the disrupted ligaments and capsular contractures, making reduction of its malalignment difficult. Reducibility, therefore, is an important parameter to consider. If the SL joint can be reduced with minimal force, the case is considered reducible. By contrast, if reduction can only be obtained by applying substantial force using "joy-sticks" wires, the case is to be considered irreducible. In general, no irreducible malalignment is likely to obtain good results by soft-tissue reconstruction.
5. Status of cartilage. It is important to note that if there is a cartilage defect, reactive chondrolysis, or joint degeneration in association to SL dissociation

By investigating these five parameters, each case can be classified into six stages (Table 10.1). The fact that the number of "no" answers increases from left to right, indicates a progression of the severity and/or chronicity of the problem from mild (Stage 1) to severe (Stage 6). The following is a description of the six stages [16].

SLD Stage 1 (Partial SL ligament injury): The SL ligaments are only partially ruptured and wrist alignment is normal. If there is pain, this is usually due to abnormal shear stress by the

increased SL motion. Usually diagnosed by arthroscopy, these patients do well by debriding any unstable fragment of the disrupted proximal SL membrane, and percutaneously pinning the SL joint under arthroscopy control. In some instances, K-wire fixation may be supplemented by a dorsal capsulodesis [17–22]. After surgery, all these patients should follow an adequate program of proprioception, re-education of the muscles that may dynamically stabilize the scaphoid, namely the flexor carpi ulnaris, extensor carpi radialis longus and brevis, and the flexor carpi radialis (FCR) [23].

SLD Stage 2 (Complete SL ligament injury, repairable): This stage is characterized by complete disruption of the three components of the SL ligamentous complex, with a ruptured dorsal SL ligament still repairable, with good healing potential. The carpal bones are normally aligned, without cartilage degeneration. The secondary scaphoid stabilizers are still functional, and therefore there is no rotator subluxation of the scaphoid. Acceptable recovery of function is likely if the dorsal SL ligament is properly reattached in the early stage [21, 22].

Following this type of surgery therapy should include proprioception to re-educate the scaphoid dynamic stabilizers [23].

SLD Stage 3 (Complete SL ligament injury, non-repairable, normally aligned scaphoid): In this stage there is complete, non repairable rupture of all three components of the SL ligamentous complex. Carpal alignment is still normal owing to the integrity of the secondary stabilizers [6]. In these cases, either a bone-ligament-bone graft or a tendon reconstruction would be indicated. This will be the main subject of this chapter

SLD Stage 4 (Complete SL ligament injury, non-repairable, with carpal collapse): This stage is defined by a complete loss of the SL linkage, plus insufficiency of the distal scaphoid stabilizers (STT and SC ligaments). The carpus has collapsed, the radioscaphoid angle is larger than 60°, and the lunate may be abnormally ulnarly translocated and in DISI. To be included in this stage however, the malalignment needs to be easily reducible [16]. Cases in this stage may be treated by a tendon reconstruction [24–26], or by a reduction-association of the SL joint (RASL procedure) [27, 28] as described below.

SLD Stage 5 (Complete SL ligament injury with irreducible malalignment, but normal cartilage): Chronic malalignment may become fixed, either by bone deformity, by capsular contracture or both, thus preventing an easy reduction. In such cases, even if there is no cartilage damage, no soft-tissue procedure can achieve long lasting stability. In most instances, these cases are better treated by a partial intracarpal fusion [29–34] than by a soft tissue procedure.

SLD Stage 6 (Chronic SL ligament injury with degenerative osteoarthritis) : Chronic SLD tends to develop cartilage degeneration following a specific pattern called "Scapho-Lunate Advanced Collapse" (SLAC) [7]. These cases are usually treated by motion preserving, salvage procedures, such as proximal row carpectomy or a scaphoidectomy plus midcarpal ("four corner" or lunocapitate) fusion.

Summary

Stage 1	Debridement and stabilization
Stage 2	Repair
Stage 3 & 4	Reconstruction
Stage 5	Intercarpal fusion
Stage 6	Salvage

Surgical Techniques Used for the Treatment of SLD

When deciding a treatment for a patient with SLD, it helps to take into account the staging system described above, as well as the individual requirements that each patient may have. The following is a description of the most commonly utilized surgical operations for the chronic SLD. Salvage procedures for the degenerative osteoarthritis caused by the unsolved SLD (stage 6) will not be discussed in this chapter.

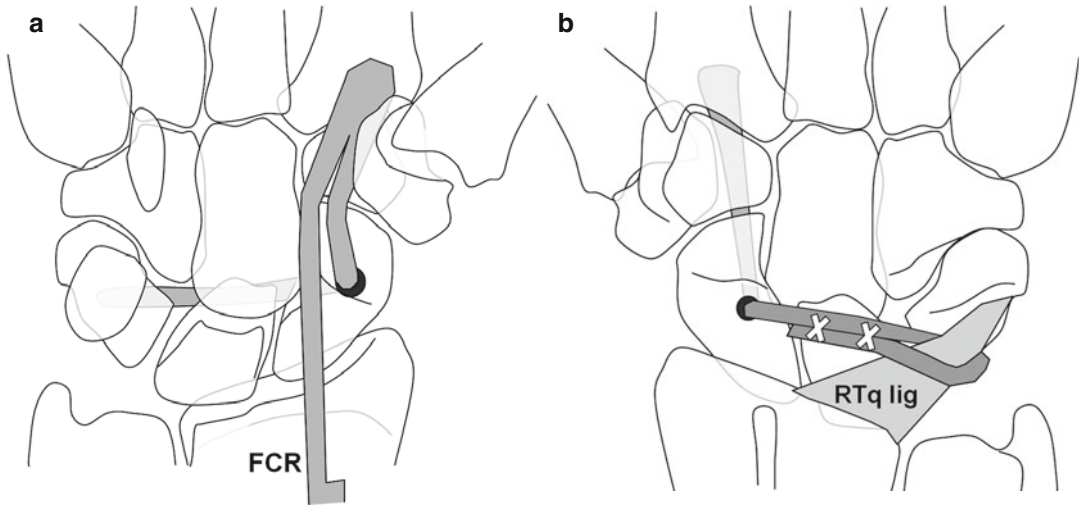

Fig. 10.2 (**a**, **b**) Schematic representation of the "Three-ligament tenodesis" technique to reconstruct unrepairable dorsal SL ligament, seen from a palmar (**a**) and dorsal (**b**) perspectives [16]. The FCR tendon is passed through a tunnel across the distal scaphoid aiming at the point where the dorsal SL ligament inserts. The tendon is then tensioned using the dorsal RTq ligament as an anchored point. Once around the ligament, the tendon is sutured onto itself. Unlike the original Brunelli method, this method does not attempt to cross the radiocarpal joint with the graft

Tendon Reconstruction of SL Stability. The 3LT Method

The first suggested treatment for SLD was described in 1975 by Dobyns and Linscheid [11], and consisted of passing a strip of tendon through anteroposterior tunnels in the proximal pole of the scaphoid and the lunate. Immediate stability was obtained by tightly suturing the tendon graft around the joint. Unfortunately, the drill holes across poorly vascularized areas of bone often fractured, inducing early joint osteoarthritis. In 1995, Brunelli and Brunelli recommended using a strip of the FCR tendon to reconstruct both the proximal and distal connections of the scaphoid, in cases with a SLD with carpal collapse [24]. The strip of tendon, which was left attached distally, was passed through a transverse drill hole, across the distal scaphoid and anchored to the dorsal-ulnar corner of the distal radius by transosseous sutures. The method was subsequently modified by Garcia-Elias, Lluch and Stanley [16], who suggested using a strip of FCR tendon to augment the palmar distal connections of the scaphoid, to reconstruct the dorsal SL ligament, and to prevent the lunate from sliding ulnarly (Fig. 10.2a, b). Because this tendon reconstruction replicates the action of three ligaments, it has been named "three-ligament tenodesis" or "3LT" procedure. It is principally indicated in cases of SLD Stage 4, that is cases with non-repairable SL ligament injury, very easily reducible and without cartilage degeneration.

The surgical technique for the 3LT procedure is as follows. The dorsum of the scaphoid and lunate are exposed using a standard dorsal approach, as described by Berger and Bishop [35]. Alternatively, a nerve sparing capsulotomy, as recommended by Hagert et al. [36] may be used. Using a cannulated drill, a 2.7 mm diameter tunnel is made across the scaphoid entering at the point of insertion of the dorsal SL ligament. The drill hole is made along the longitudinal axis of scaphoid, aiming at the palmar convexity of the scaphoid tuberosity. Through small transverse incisions a distally based, 8 cm long, strip of FCR tendon is obtained and passed through the scaphoid tunnel using a wire loop. A transverse trough or channel over the dorsum of the lunate is then made with a rongeur. This trough needs to be deep enough to uncover cancellous bone. To ensure intimate contact between the tendon strip and the decorticated dorsal aspect of the lunate,

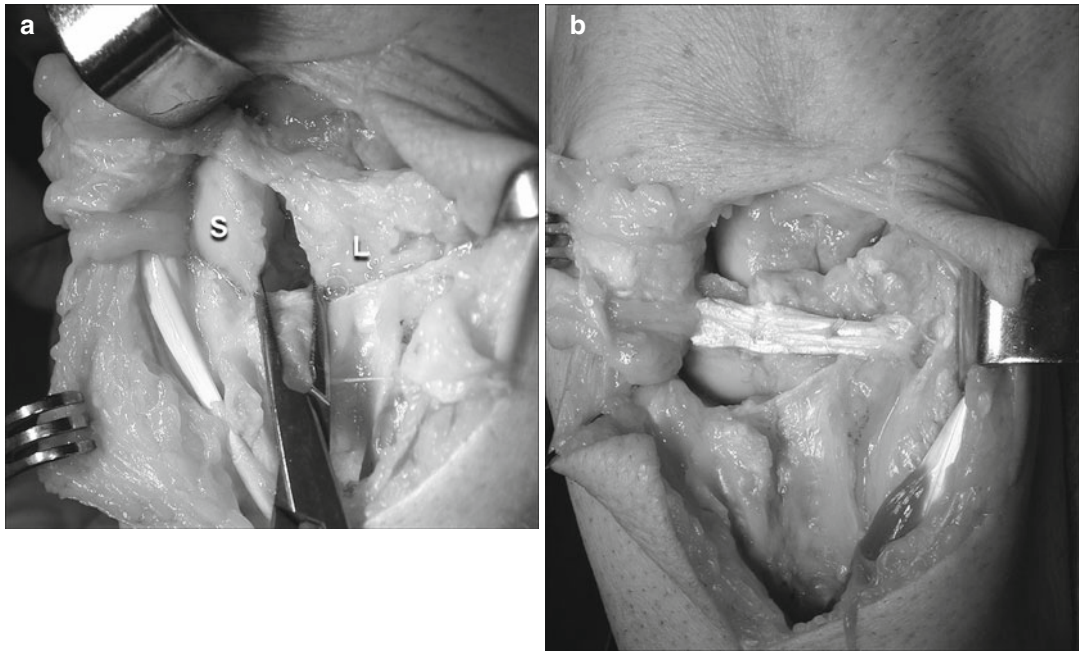

Fig. 10.3 (**a**, **b**) Chronic, reducible SLD, without osteoarthritis (Stage 3). (**a**) Surgical picture of the SL joint, with a forceps demonstrating complete rupture of the SL ligaments. Only an elongated distal portion of the dorsal SL ligament is present, probably one of the reasons why the carpus had not collapsed yet. (**b**) Dorsal aspect of the "three-ligament tenodesis" before closing the capsulotomy. Note the tendon graft sutured onto itself, connecting the scaphoid to the dorsal radiotriquetral ligament

an anchor suture is placed into the floor of the trough. The dorsal radiotriquetral ligament is then identified and a slit is created in its distal end, just proximal to its triquetral attachment. The tendon strip is passed through that slit, and the ligament is used as a pulley to tension the ligament strip (Fig. 10.3a, b). Before tightening the tendon, the scaphoid, lunate and capitate are reduced and stabilized with K-wires. The tendon graft is finally sutured onto itself, and the anchor suture in the lunate is used to ensure close contact of the tendon graft to lunate cancellous bone.

Complete wrist immobilization is maintained for 6 weeks, first in a short arm thumb spica cast for 4 weeks and a dorsal splint for another 2 weeks, when wires are removed. After this, a protective removable splint, allowing supervised rehabilitation, is retained for an additional 4 weeks. Contact sports are to be avoided for 6 months after surgery.

In a recent review of 38 patients with symptomatic SL dissociation who had undergone the 3LT procedure with an average follow-up of 46 months (range 7–98), pain relief at rest was obtained in 28 patients, with 8 complaining of mild discomfort during strenuous activity, and 2 having pain in most activities of daily life [16]. Twenty-nine resumed their normal occupational-vocational activities. Average ranges of motion at follow-up were for flexion 51 (74 % of contralateral), extension 52 (77 %), radial inclination 15 (78 %) and ulnar inclination 28 (92 %). Average grip strength relative to the contralateral normal side was 65 %. There were no signs of scaphoid necrosis. Seven patients showed mild signs of degenerative osteoarthritis at the tip of the radial styloid, while two developed subsequent global arthrosis (SLAC), although none were substantially symptomatic.

According to the more recent publications, the 3LT tenodesis appears to be a quite reliable solution for the unstable SLD, with less morbidity than partial fusions, and acceptable functional results [25, 26]. Whether or not the stabilizing

efficacy of this technique deteriorates with time, however, is not known.

Bone-Ligament-Bone Grafts

In an attempt to replicate the positive results of replacing knee ligaments with bone-ligament-bone grafts, Weiss suggested transferring a bone-retinaculum-bone autograft harvested from the region of Lister's tubercle to the dorsal SL ligament [37]. Harvey et al. [38] recommended the use of the third metacarpal-capitate ligament. The surgical technique is straightforward. Once the SL joint is reduced and transfixed by wires, a deep trough is carved at both sides of the joint and the bone-ligament-bone graft is buried and fixed with mini screws. In theory, the implanted ligament has similar elasticity and strength to the original structure. Unfortunately, the proximal scaphoid is poorly vascularized, making difficult the consolidation of the graft. Furthermore, the technique requires prolonged immobilization, which will result in the mechanical properties of the ligament deteriorating and ultimately potentially failing under the torques that will be exerted on it. However, the early clinical results are encouraging in the dynamic instabilities with normal secondary stabilizers [38]. By contrast, the rate of complications when used in static instabilities is still very high [37].

Reduction-Association of the SL Joint (RASL Procedure)

In the late 80's, several authors attempted to fuse the SL joint, as the primary solution for this type of carpal instability [27, 28]. Interestingly enough, the ones that failed to obtain fusion did clinically better than those that achieved fusion. This led Herbert and colleagues [27], as well as Rossenvasser et al. [28] to propose a novel approach to static SL instabilities. The method consists of repairing the SL ligaments, and protecting the repair, by internally blocking the SL joint with a transverse headless screw for 8 months or more. The goal was to achieve wrist stability by inducing fibrous union of the SL joint. The early results were surprisingly good, most patients exhibiting substantial range of motion and a strong grip [28]. The long term results of creating so much stiffness in one of the most mobile intercarpal joints is not known. Certainly, caution is recommended when using this experimental method.

Scaphotrapezoid-Trapezial Arthrodesis

Fusing the scaphoid to the distal row has been one of the most popular methods of treatment of SLD [30–33]. The aim of the procedure is to bring the proximal pole of the scaphoid back to its natural articulating position at the center of the scaphoid fossa. With this, the radioscaphoid congruency is secured and the possibility of developing later degenerative changes is reduced. Yet, STT fusion is a technically demanding procedure. It is important that the external dimensions and alignment of the resultant STT fused block be the same as the external dimensions of the bones in the normal wrist. Under-reduction (scaphoid flexed) fails to close the SL gap, whilst over-reduction (scaphoid extended) may induce rapid radioscaphoid osteoarthritis. Furthermore, the average rate of non-union is not low, pain relief is not uniformly predictable and radioscaphoid impingement pain is a frequent problem [31, 32]. To alleviate that, Watson recommends performing a dorsolateral styloidectomy in association with an STT fusion [33]. Of course, any pre-existing cartilage defect in the radioscaphoid joint is a formal contraindication to STT fusion.

Radio-Scapho-Lunate Fusion Plus Distal Scaphoidectomy

In recent years the functional importance of the midcarpal joint has been emphasized. Certainly, in most activities of daily living the wrist moves mostly along the so-called dart-throwing plane of rotation (from an extended-radial deviated position, to a flexed-ulnar deviated position), a particular type of wrist rotation that has proved to occur almost exclusively in the midcarpal joint (Fig. 10.4). Based on this, if a painful SLD is not reducible and needs to be stabilized by an arthrodesis, fusing the radioscapholunate joint rather than the midcarpal joint may be a reasonable choice [34]. That option would be particularly indicated if, aside from the SLD, there are abnormalities in the radiocarpal joint. Fusing the radiocarpal joint eliminates pain induced by local synovitis, while stabilizing the proximal component of the

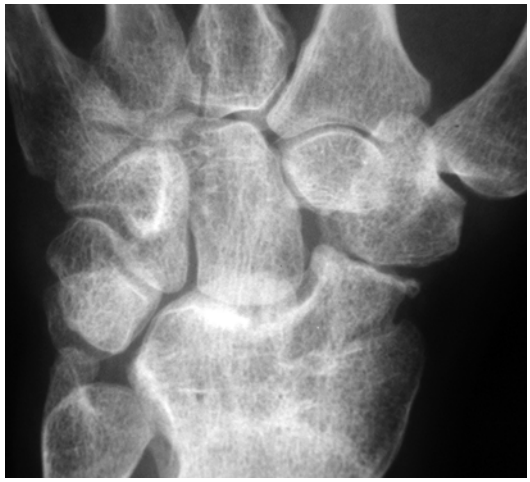

Fig. 10.4 PA radiological view of the wrist of a 47 years of male who underwent a radioscapholunate fusion plus distal scaphoidectomy 7 years ago for a chronic hardly reducible SLD. Note the integrity of the central portion of the midcarpal joint, allowing 70° of "dart-throwing" motion, and 85 % grip strength

midcarpal joint. In order to avoid the STT joint becoming symptomatic, as the consequence of localized radioscaphoid impingement, routine excision of the distal third of the scaphoid has been suggested [34]. With this technique, the midcarpal "ball-and-socket" articulation is liberated from its lateral constraint and allows more than 50 % of the overall wrist motion. The early results published so far are very encouraging.

Lunotriquetral Dissociation

Post-traumatic lunotriquetral (LTq) instability usually results from injury to the LTq supporting ligaments, a condition often missed at presentation [39–41]. A high proportion of LTq ligament ruptures result from a fall backwards on the outstretched hand, the arm being externally rotated, the forearm supinated and the wrist extended and ulnarly deviated [40]. If in that position the hand hits the ground, most of the blow is taken by the pisiform, which displaces dorsally against the triquetrum. Because the lunate is blocked dorsally by the radius, substantial shear stress to the LTq joint appears. This is the most likely explanation for the frequent tears of the LTq ligaments. The same mechanism may also explain some peripheral tears of the triangular fibrocartilage complex, and avulsion of the ulnocarpal ligament; so it is not rare that a combination of all these injuries appear in the same patient [41, 42].

Ulnarside wrist pain and weakness are the most common symptoms of patients with LTq instability [43]. As stated for SLD, there is a spectrum of clinical presentations involving varying degrees of LTq joint disruption, ranging from a partial tear of its proximal membrane to complete dissociation with static carpal collapse [42]. In the latter stage, the carpus usually collapses into a static VISI, and the wrist presents with a forklike appearance, with the ulnar column sagging palmarly, relative to the prominent distal ulna. Often there is painful crepitation in ulnar deviation, point tenderness over the dorsal aspect of the LTq joint and a giving way sensation in full supination, ulnar deviation and extension. Ulnar nerve paresthesias are usual.

A positive "ballottement test" is pathognomonic of this problem [39]. Whilst blocking the lunate with one hand, the triquetrum and pisiform are passively mobilized back and forth with the other hand. If there is painful asymmetric displaceability of the joint, with crepitation, the test is positive. The so-called "Derby test" is also helpful [43]. The pisiform is pushed dorsally while the wrist is in extension and radial deviation. With this, the LTq joint is reduced, the feeling of instability disappears and the grip strength becomes normal, until the compression to the pisiform is released.

X-rays are normal if there is only the intrinsic LTq ligaments damage [41]. In fact, for a VISI pattern of collapse to appear, there is a need for a more extended rupture or attenuation of the LTq joint supporting ligaments, this includes the dorsal radiotriquetral and the palmar midcarpal ligaments. In such static CID-VISI cases, the lunate has a moon-like configuration on PA views, the scaphoid is foreshortened, and there is disruption of the so-called Gilula's line 1, in the form of a step off between the lunate and the triquetrum [10]. In such cases, it is not unusual to find a slightly increased SL gap and a positive ring sign of the flexed scaphoid. This does not represent an SL rupture, but the result of the capitate loading the most palmar SL ligament fibers, which are longer than the dorsal ones, thus allowing separation

of the two bones. When the diagnosis offers some difficulty, arthroscopy is strongly recommended [44]. The joint is best seen through the radial midcarpal portal; a normal LTq joint is tightly closed and does not allow introduction of the trocer in the joint unless the ligaments are abnormally lax.

Treatment of LTq Instability

In general, vascularity of the palmar LTq ligament is good, for acute tears of the LTq ligaments tend to heal well with immobilization alone. If unusually unstable, percutaneous K-wire fixation for a period of 6–8 weeks often leads to excellent results [44]. Open repair of the thin dorsal LTq ligament is not easy and probably unnecessary.

Long lasting dynamic instability of the LTq joint is currently treated with splints that stabilize the Ltq joint by pushing dorsally the pisotriquetral joint. Avoidance of actions reproducing the painful crepitation is also important, as well as a supervised therapy program to re-establish adequate proprioceptive control of the extensor carpi ulnaris (ECU) and flexor carpi ulnaris (FCU) on the ulnar column of the wrist. Isometric contraction of these two muscles generates a dorsally directed force by the pisiform, onto the triquetrum that helps controlling the overall alignment of the wrist [10]. Certainly, inadequate neuromuscular control and functional overuse is what precipitates the onset of symptoms of most dynamic LTq instabilities.

When conservative treatment has failed in controlling symptoms, different surgical strategies have been proposed. LTq partial arthrodesis has been, by far, the most frequently utilized alternative to the point of being considered the gold standard [45]. Surprisingly enough, the technique is recommended, even though most publications insist on these fusions not being very effective in eliminating pain, particularly when the ulna is long [32, 46]. Indeed, LTq fusions are to be associated with a partial resection of the distal ulna (open or arthroscopic "wafer" procedure) or to an ulnar shortening if there is an ulnar plus variance. Furthermore, localized fusion is contraindicated if the LTq instability has evolved into aVISI carpal malalignment [10]. Tendon reconstruction of the LTq ligaments using a strip of ECU, recreating a loop around the Ltq joint is another alternative, pioneered by the group from the Mayo Clinic [47] (Fig. 10.5a–c). A third alternative has been proposed by the colleagues at the Wrightington Hospital in UK. It consists of creating a vertical tether to the triquetrum, with a strip of ECU tendon passed through a bone tunnel on the dorsal aspect of the triquetrum, and triangular fibrocartilage [48] (Fig. 10.6). Both tenodesis alternatives have found promising results, certainly better than localized fusions. Yet no publications, other than the ones from the first proponents of these techniques, are available.

When the LTq instability has evolved into a massive carpal collapse in VISI, no soft tissue procedure has proved to be capable of solving the problem. In those cases, either fusion of the LTq joint plus a radiolunate fusion, or a midcarpal ("four corner") fusion is recommended. When the chronic LTq instability is associated to SL instability, the case is categorized as a perilunate instability, and is to be treated by means of a proximal row carpectomy, or with the most complex "spiral tenodesis" procedure. In this latter case, a strip of FCR tendon is used not only to reconstruct the dorsal SL ligament, as in the "3LT procedure", but also both LTq ligaments, palmar and dorsal, as well as the palmar radiotriquetral ligament (Fig. 10.7a–c). Although yet unpublished, this technique has shown promising results in this author's experience. As with most tendon reconstructions analyzed in this chapter, long term results have not yet been published, for caution is always recommended.

Summary

Localised tenderness and bolotment is pathognomonic

X-ray may be normal

Diagnosis confirmed by arthrogram or arthroscopy

Treatment with either ligament reconstruction or fusion

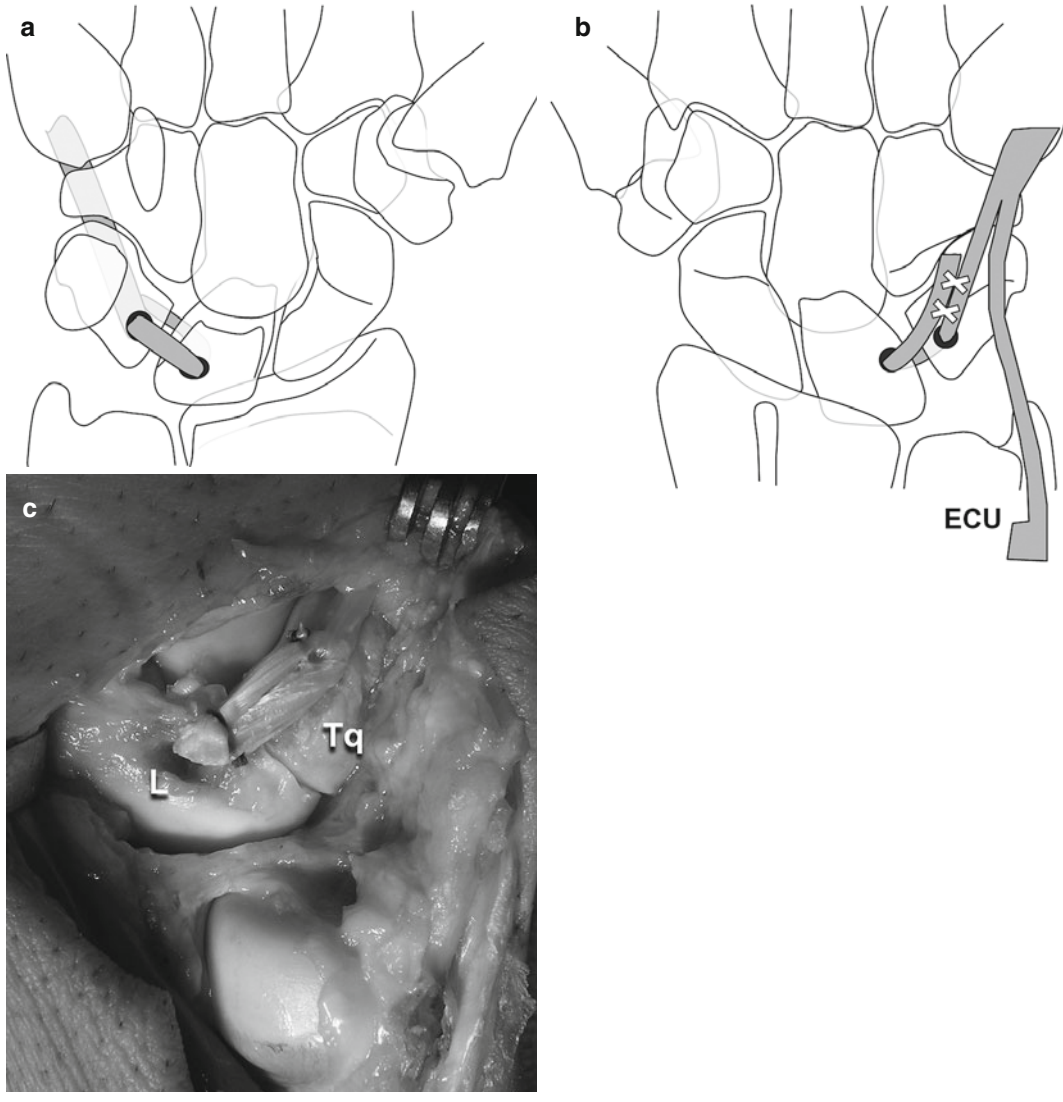

Fig. 10.5 (**a**, **b**) Schematic representation of the Mayo Clinic ligament reconstruction of chronic LTq dissociation [47]. A strip of Extensor Carpi Ulnaris (*ECU*) is looped around the joint across two tunnels converging onto the two insertion sites of the volar LTq interosseous ligament. (**a**) Volar view. (**b**) Dorsal view. (**c**) Surgical picture of the dorsum of the LTq joint after an ECU tendon stabilization, (According to Shin et al. [47])

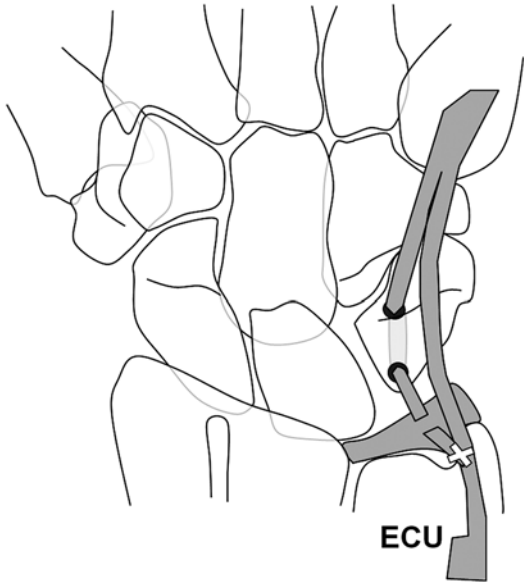

Fig. 10.6 Schematic representation of the Wrightington method of stabilization of dynamic LTq dissociations [48]. Using a strip of the Extensor Carpi Ulnaris (*ECU*) tendon, the unstable triquetrum is tethered proximally to the dorsal edge of the triangular fibrocartilage, and distally to the hamate. It is particularly indicated in chronic dynamic LTq instability. In patients who have associated problems in the wrist, the results are less predictable

Summary

The term "carpal instability" describes a wrist dysfunction where there is a loss of the ability to bear physiologic loads without yielding (kinetic instability) and/or the ability to move without sudden changes of carpal alignment (kinematic dysfunction). A biomechanically unstable wrist should not be considered a clinical entity, until the dysfunction is associated with symptoms, such as pain, loss of grip strength or a giving-way sensation when performing hand activities. Treatment decision in post-traumatic dissociative instabilities is based on five prognostic factors: extent and nature of the ligament rupture, healing potential of the disrupted ligaments, status of the secondary stabilizers reducibility and finally the indemnity of the articulating surfaces. Some chronic instability may be successfully treated by solving the underlying injury (ligament disruption or bone fracture) plus adequate muscle re-education of wrist dynamic stabilizers. When neuromuscular control of stability cannot be re-established, however, salvage procedures, such as partial fusions, proximal row carpectomy, or wrist implant arthroplasty may be necessary to regain painless function.

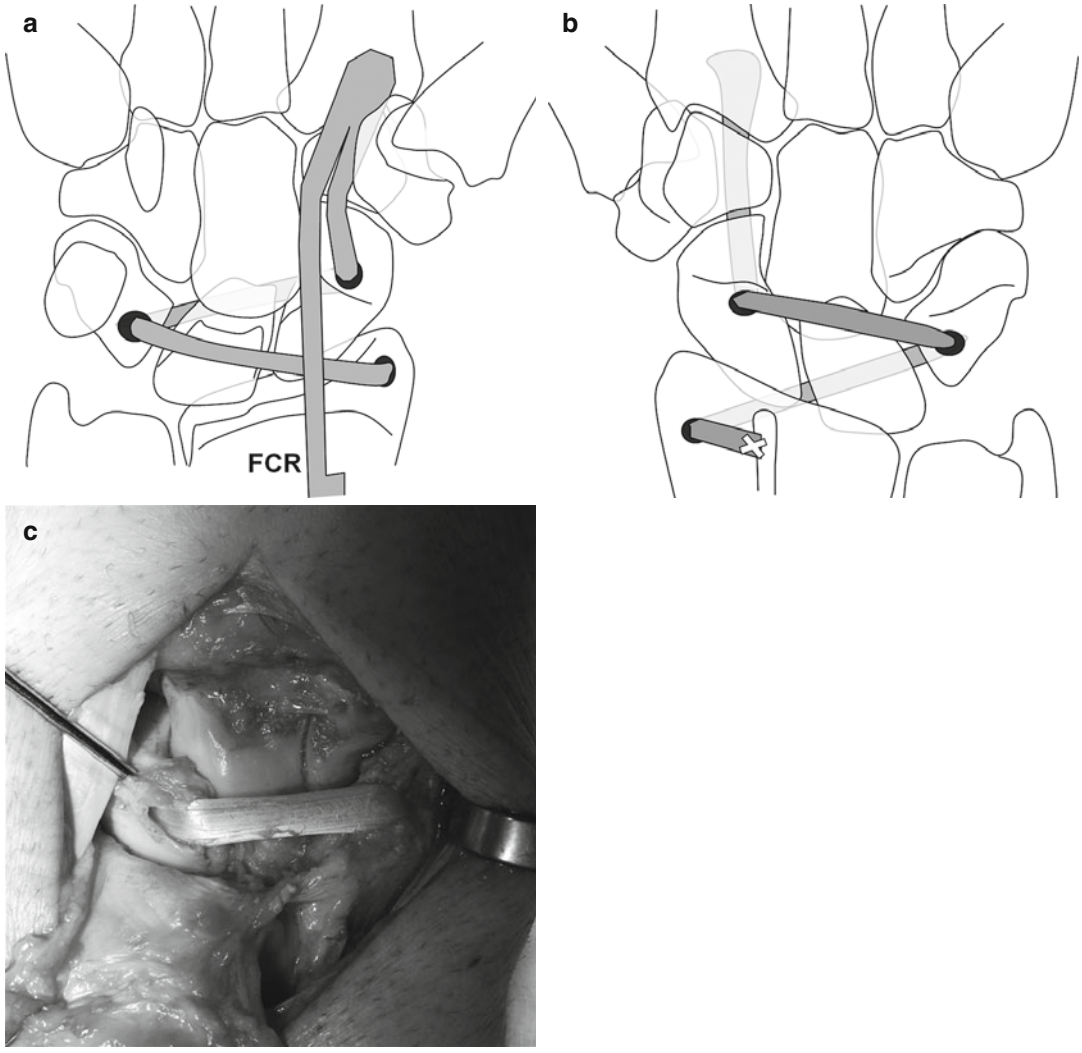

Fig. 10.7 (**a**–**c**) Spiral tendon reconstruction of a combined SL and LTq instability, a complex injury occasionally seen after self-reduced untreated perilunate dislocation. A strip of FCR tendon is passed from palmar (**a**) to dorsal (**b**) across the scaphoid, and back into the carpal tunnel across the triquetrum. Once in the volar aspect of the wrist, the tendon is brought under the contents of the carpal tunnel towards the radial styloid where it is tightly inserted into the radial styloid. (**c**) Surgical picture of the portion of tendon as it binds the dorsum of the scaphoid and triquetrum

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Chronic Distal Radio-ulna Instability

11

Marc Bransby-Zachary and Ian A. Trail

Keywords

DRUJ Laxity • DRUJ Anatomy • DRUJ Arthrology • DRUJ Stabilisers • DRUJ Kinematics • DRUJ Biomechanics • Classification of DRUJ Instability • Examining for DRUJ Instability • Investigating DRUJ Instability • Congenital DRUJ Instability • DRUJ instability in Rheumatoid arthritis • DRUJ instability following Fractures • DRUJ Instability following soft tissue injury • Lichtman/Collins Classification of DRUJ Injuries

Introduction

Instability may be defined as an abnormal pattern of joint kinematics that allows the Distal Radio-Ulna Joint (DRUJ) to sublux or dislocate. There must, however, be a loss of the control of this movement for the patient to experience symptoms. This is distinctive to joint laxity, which is asymptomatic.

Forearm rotation is permitted by movement of the proximal and distal radio-ulnar joints and by the interosseous membrane.

The distal radio-ulnar joint (DRUJ) is a diarthrodial trochoid articulation, that is a joint that allows free motion around a central axis. In this case the axis is around a line from the radial head to the ulna styloid. The ulna head should be considered as the fixed axis around which the distal radius moves. This can cause some confusion in the description of the relative movements between the radius and ulna. When describing the movements of the radio-ulna joint, surgeons tend to refer to the position of the ulna in relation to the radius, whereas it is the reverse that is anatomically correct. Descriptions of these movements in the literature vary in whether they use the correct fixed point of the ulna as the reference point on which the radius moves, or the apparent movement of the ulna on the radius. As a consequence, care needs to be taken over interpretation of movements at this joint. In this chapter, the movement of the radius in relation to a fixed ulna will be used, unless specifically indicated.

The stability of the DRUJ is dependent on the integrity of the soft tissue envelope which forms both static and dynamic restraints and the normal

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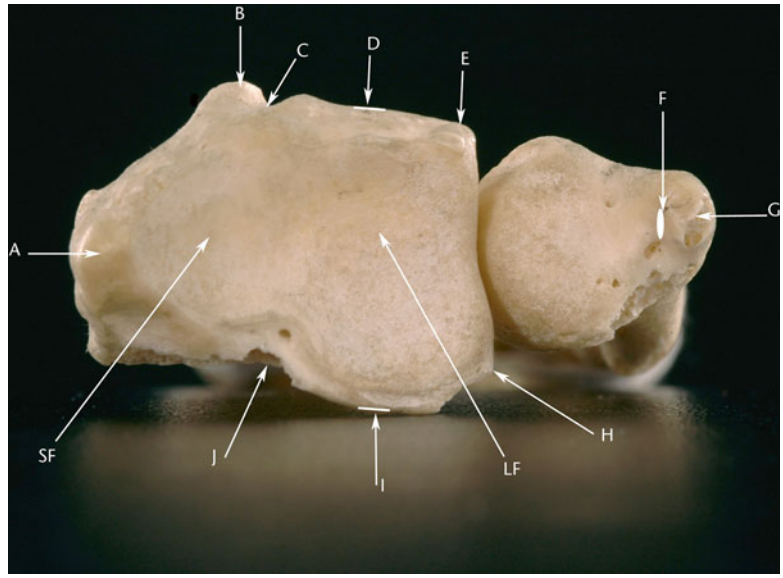

Fig. 11.1 Anatomy of distal radius and ulna (*A*) Radial styloid (*B*) Listers tubercule (*C*) Groove for extensor pollicis longus (*D*) Dorsal rim of radius (*E*) Attachment of Dorsal Radio-Ulna Ligament (DRUL) (*F*) Fovea of ulna. Insertion of PRUL & DRUL (*G*) Ulnar styloid (*H*) Attachment of Palmar Radio-Ulna Ligament (PRUL) (*I*) Palmar rim of radius (*J*) Footprint of ligament of Testut. *LF* lunate fossa, *SF* scaphoid fossa

bony architecture. To understand DRUJ instability, therefore, a detailed knowledge of both the anatomy and normal movement patterns is required.

Functional Anatomy

The DRUJ is formed by the articular surface of the head of the ulna, which forms 220° of the circumference of the ulna head and the sigmoid notch of the radius. The sigmoid notch is a shallow articulation which has a radius of curvature greater than that of the distal ulna. This difference is on average 4–7°, such that the curvature of the ulna head is about two thirds of that of the sigmoid notch. On average the radius of curvature of the ulnar articulation is 10 mm and the sigmoid notch is 15 mm. Thus, as well as rotation, antero-posterior translation of the ulna head in relation to the radius can occur. The apposing radial and ulna articular surfaces can be either parallel or oblique. The latter, either angulating away from the ulna proximally, or the reverse, angulating towards the ulna. The majority of the articular surfaces are parallel. In addition, in the transverse plane, the articular surfaces of the sigmoid notch can take various shapes, although predominantly is either flat or 'c' shaped. Finally, bony architecture probably contributes little to stability of the DRUJ, apart from the dorsal and palmar osteo-cartilaginous lips [1, 2] (Fig. 11.1).

The soft tissues that contribute to the stability include both static and dynamic stabilisers. These include:

1. Triangular fibro-cartilage complex and radio-ulnar ligaments
2. Capsular envelope
3. Ulno-carpal ligaments
4. Interosseous membrane
5. Extensor carpi ulnaris & sheath
6. Pronator quadratus

The triangular cartilage complex consists of two strong radio-ulnar ligaments that arise from the dorsal and ulna margins of the sigmoid notch, the dorsal radio-ulna ligament (DRUL) and the palmar radio-ulna ligament (PRUL) respectively and these insert into the fovea of the ulna head at the base of the ulnar styloid (Fig. 11.2). The floor of the extensor carpi ulnaris sheath has dense fibres that run to the fovea of the ulna merging with the DRUL. The ulna triquetral and ulna lunate ligaments arise from the palmar radio-ulnar ligament (PRUL).

The normal translation of the sigmoid notch over the ulna head from full pronation to full supination is approximately 4 mm. With the forearm in neutral rotation, the ulnar head feels as if it can glide when stressing it antero-posteriorly.

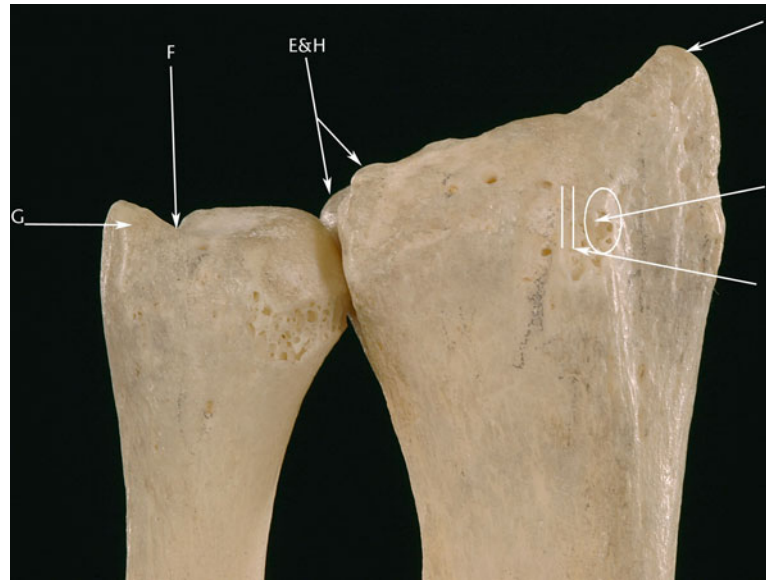

Fig. 11.2 Longitudinal section showing attachment of TFCC and radiolunar ligaments (*A*) Radial styloid (*B*) Listers tubercule (*C*) Groove for extensor pollicis longus (*E*) Attachment of Dorsal Radio-Ulna Ligament (DRUL) (*F*) Fovea of ulna. Insertion of PRUL & DRUL (*G*) Ulnar styloid (*H*) Attachment of Palmar Radio-Ulna Ligament (PRUL)

For the distal ulna to sublux or dislocate it must move beyond the edge of the sigmoid notch; for this to occur the ulna must move away from the radius. The interosseous membrane normally maintains a constant length throughout rotation, as it lies in an isometric position. Thus for separation of the radius and ulna to occur, the interosseous membrane must be torn in its distal portion. The pronator quadratus assists as a dynamic stabiliser compressing the DRUJ.

The extensor carpi ulnaris and its sheath also contribute to the stability. The deep portion of the sheath assists by virtue of merging with the DRUL. The ECU can have no effect with the wrist in pronation, since at that point it is lying over the dorsal part of the joint which is de-tensioned. On full supination, however, the ECU is lying medially and thus tension in this tendon will have the effect of compressing the DRUJ, providing dynamic stability.

In addition to antero-posterior movement of the DRUJ in forearm rotation there is also axial glide. With the forearm in full supination, the radius and ulna are parallel to one another, such that the length of the radius relative to the ulna is greatest in this position. As one pronates, the radius crosses obliquely over the ulna, thus making it relatively shorter than the ulna. This relative shortening is approximately 2–3 mm. This also occurs on gripping, since loading the radial head has the effect of shortening the radius.

Clinical Pearl

For true instability of the distal radioulnar joint, both the dorsal and palmar radio-ulnar ligaments, as well as the interosseous membrane, must be torn.

Kinematics and Biomechanics

Both the PRUL and DRUL are non-elastic. In neutral rotation both are tensioned equally. In a dry bone specimen, as the forearm is pronated the distance from the fovea of the ulna and the palmar margin of the sigmoid notch diminishes, thus de-tensioning the PRUL (Fig. 11.3). The distance from the fovea of the ulna to the dorsal margin of the sigmoid notch increases with this movement. As a consequence, to maintain a constant distance from the sigmoid notch to the dorsal lip of the sigmoid notch (since the DRUL cannot elongate) the radius must translate in a palmar direction (bringing the ulna head up to the dorsal lip of the sigmoid notch) [3, 4]. In supination, the reverse happens [5] (Fig. 11.4).

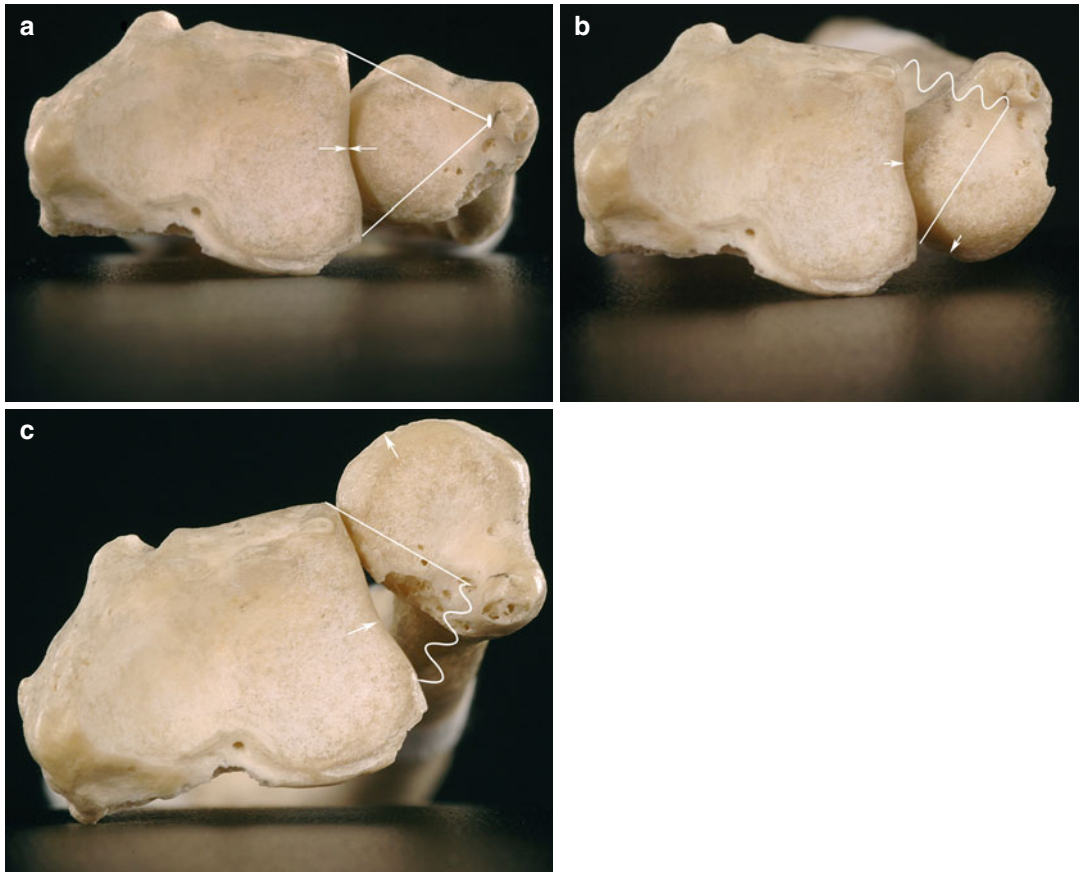

Fig. 11.3 Normal translation of the radius on the ulna head during pronation and supination demonstrating the tensioning and de-tensioning of the PRUL and DRUL during these movements. (**a**) Neutral rotation. Ulna head lies in the centre of sigmoid notch (*arrows*) and the PRUL and DRUL are equally tensioned (*Solid lines*). (**b**) Supination. Radius has translated dorsally on the ulna (*arrows*). This puts the PRUL under tension (*Solid line*) and de-tensions the DRUL (*curved line*). (**c**) Pronation. Radius has translated palmarly on the ulna (*arrows*). This puts the DRUL under tension (*Solid line*) and de-tensions the PRUL (*curved line*)

However, the above supposition is not universally accepted, in that the reverse of this tensioning has been proposed [6]. However, simple observation of a dry bone articulation will confirm the distance from the fovea of the ulna and the dorsal and palmar edges of the sigmoid notch. In addition, research has also shown that the superficial and deep parts of the PRUL and DRUL behave differently as discreet entities [7]. Other histological research, however, has failed to demonstrate these discrete areas and transducer studies, which measure the tension in these ligaments, confirm the expected translation and ligament tension on rotation. Specifically, that the dorsal capsule of the DRUJ is tight in pronation and the palmar capsule is tight in supination.

Much of the literature has concentrated on the key role of the TFCC for stability of the DRUJ. Clearly it must have an important role and, for instability to occur, it must be disrupted at least in part. It is self-evident, by observation of the anatomy, that when there is a dorsal dislocation of the ulna relative to the radius, then the distance between the palmar margin of the sigmoid notch and the fovea of the ulna is greatly increased. In this circumstance, the PRUL must be disrupted. The reverse is true in palmar dislocations. Thus, in either dorsal or ulna dislocations, the TFCC must be either at least in part or completely

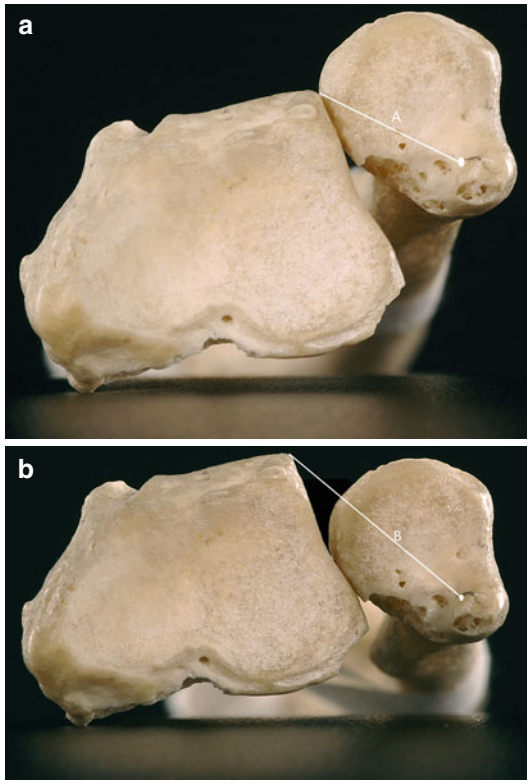

Fig. 11.4 Forearm rotation: The effect on DRUJ translation. (*A*) Full pronation. The radius translates in a palmar direction to allow the length of the DRUL to remain constant (**a**) (*B*) Full pronation. If no radial translation occurs the DRUL would have to lengthen (**b**) This is not possible since it is inelastic

Table 11.1 Lichtman/Collins combined classification of DRUJ injuries

Stable DRUJ:
(a) Acute TFCC tears: 1A 1D (central and radial)
(b) Degenerative TFCC tears (abutment)
Partially stable DRUJ
(a) Acute TFCC tears: 1B &1C (ulna and distal)
(b) ECU subluxation
(c) Degenerative TFCC tears
Unstable DRUJ
(a) Simple (reducible and stable)
(b) Complex: irreducible (interposed soft tissue)
Massive tear of TFCC
Displaced ulna styloid fracture
Distal radial fracture with comminuted sigmoid notch
Essex-Lopresti lesion
Galleazi fracture
Both bone forearm fracture

Nicolaidis et al. [19]

disrupted. This also includes avulsion of the TFCC from the sigmoid notch.

The ulno-carpal ligaments and capsule are also tensioned in pronation and supination. Biomechanical transducer studies have demonstrated the importance of the ulna-carpal ligaments throughout these movements.

Experimental analysis of the stability of the DRUJ, following sectioning of the soft tissue stabilisers, has been undertaken. Gofton et al. (2004) studied the effect of sequential sectioning of the dorsal and palmar radioulnar ligaments, the triangular cartilage, the ulnocarpal ligaments, the extensor carpi ulnaris and its sub-sheath, the pronator quadratus and finally the interosseous membrane [8]. They demonstrated that whilst the radioulnar ligaments and triangular cartilage could maintain stability in the absence of other soft tissue restraint, if the radioulnar ligaments and triangular cartilage were divided, the intact other soft tissue restraints were able to stabilise the DRUJ with normal kinematics. Thus, a tear of the TFCC alone will not create instability. For instability to occur, other major ligamentous disruption must take place.

Classification

Whilst Palmar (1989) has classified the injuries of the TFCC, this does not necessarily relate to instability, since other structures must be injured for this to occur [9]. Lichtman and Collins, however, have proposed a combined classification of DRUJ injuries. This includes all injuries of the DRUJ, rather than focusing on instability alone (Table 11.1) [19].

Instability can be considered as congenital or acquired. Causes of congenital instability include Madelungs deformity and other dysplasias (Fig. 11.5).

Acquired instability may be caused by a combination of bony and soft tissue injury, or soft tissue injury alone. When considering the bony

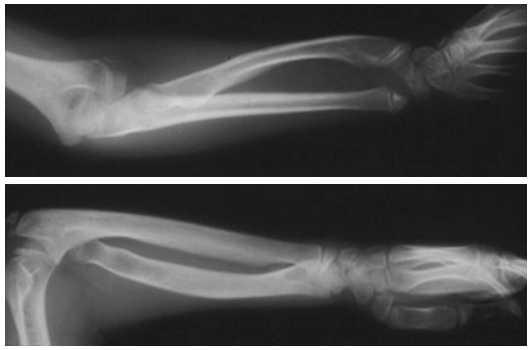

Fig. 11.5 Madelungs deformity

injuries, one should consider fractures that give rise to angular or axial deformity, or injuries to the sigmoid notch. Distal radial fractures with angulation, or angular malunion are the commonest cause of DRUJ dislocation and instability (Fig. 11.6). The more proximal the radial fracture, the smaller the angular deformity required to cause symptoms [10]. Axial instability occurs when there is radial shortening. The Essex-Lopresti injury is a complex lesion, including a radial head fracture, disruption of the interosseous membrane and axial instability of the DRUJ. More distal radial fractures, which have healed in a shortened position with disruption of the interosseous membrane distal to the injury, will lead to axial subluxation. However, in this injury the proximal interosseous membrane and radial head are still intact, ensuring that axial instability does not occur.

Sigmoid notch fractures may include dorsal or palmar marginal injuries or more complex patterns. In these, the ligamentous injury can be much less severe for dislocation and subluxation to occur, since the bony restraint for subluxation is diminished.

Soft tissue injuries that give rise to instability in the absence of a fracture are rare and represent a severe disruption. As stated previously, it is not enough for the TFCC and dorsal and palmar radioulnar ligaments to be disrupted in isolation for instability to occur. There must be enough ligamentous disruption for the radius and ulna to drift apart, such that the ulna can escape over the edge of the sigmoid notch. This will require the distal interosseous membrane to be disrupted, as well as various active restraints. Finally, fractures of the ulna styloid, particularly through the base, is in effect a complete tear of the TFCC. Both the dorsal and palmar radio-ulna ligament will be detached. This does not occur with fractures of the tip of the ulna styloid, as this is distal to the ligament attachment [11].

Instability can also present in inflammatory arthropathies, typically rheumatoid arthritis. This is due to persistent synovitis causing capsular and ligamentous laxity. Arthrosis of the DRUJ in the absence of an inflammatory arthropathy is usually post traumatic. Primary osteoarthritis is rare.

Clinical Evaluation

Patients with instability present with ulna sided wrist symptoms, typically pain and clicking. They will also describe a feeling of ‘giving way’, or sudden pain on small movements. These symptoms are intermittent. The congenital group will have a self-evident history of wrist abnormality, but may develop new symptoms due to DRUJ instability in their late teens or early twenties, usually related to new sports or activities taken up at that age. Whilst the DRUJ is often affected early in rheumatoid arthritis, it would be unusual for a patient to present with DRUJ instability in the absence of an obvious diagnosis or other clinical signs of a polyarthropathy. In all other cases there will be a history of injury, most commonly with an associated fracture, but if not, a history of significant trauma with persisting symptoms thereafter.

The differential diagnosis will include soft tissue problems, such as TFCC injury, injury to the luno-triquetral ligament or less commonly capito-hamate ligament and instability of the Extensor Carpi Ulnaris (ECU) tendon. Persistent symptoms from fractures which were not recognised, or remain un-united, may mimic DRUJ instability, such as the less common carpal fractures of the lunate, triquetrium or hamate, including the hook of the hamate. Capitate neck fractures can also give rise to ulna sided wrist pain. Intra-articular fractures in the radio-carpal joint that

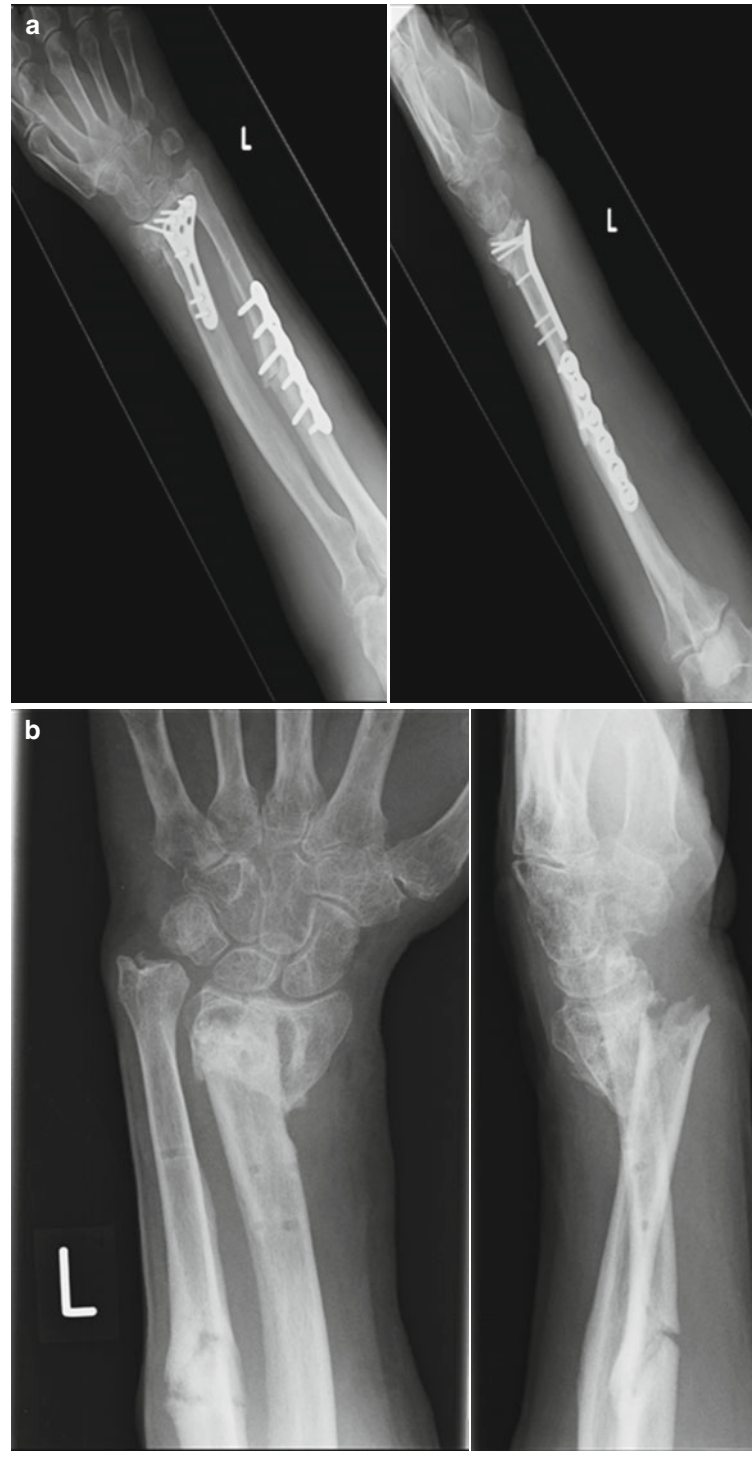

Fig. 11.6 Distal radial fracture and DRUJt instability (**a**) Fracture malunion post op. (**b**) Fracture malunion pre op

Fig. 11.7 Piano key signs

have healed with a step may give pain and clicking as can any fracture within the sigmoid notch. Keinböck's disease and pathology of the piso-triquetral joint must also be considered in the differential diagnosis.

In inflammatory arthropathies the difficulty is in the differentiation of the instability from the symptoms of arthrosis or synovitis.

Examination may reveal asymmetry of the DRUJ, if there is a subluxation or dislocation. Tenderness round the DRUJ may be elicited, as well as pain and clicking on forearm rotation. A piano key sign may be present (Fig. 11.7). This is a dorsally displaced ulna (more correctly the palmar displacement of the radius), which is reducible, but re-subluxes when the pressure on the distal ulna is removed. The translation test for the distal ulna should be diagnostic. The radius is held firmly between the thumb and fingers of the examiners hand. The ulna is then held in the other hand and moved in the antero-posterior plane. This should be done in neutral, full supination and full pronation. A difference in the range of translation, clicking during the movement or the reproduction of the patient's symptoms during this manoeuvre should confirm the diagnosis. However, this test is more difficult to evaluate correctly and identify instability than the description of the sign would suggest. This is due to the small difference between normal translation, which is in the order of 4–5 mm and subluxation, which may add only 1–2 mm of further movement. Different positions of forearm rotation will give different degrees of translation and thus more accurate assessment, comparing the translation to the normal side may be compromised. The key to the test is to demonstrate the escape of the ulna head over the margin of the sigmoid notch. This may be easier to feel as a relocation test. This is performed by compressing the ulna head into the radius, after maximum translation and then attempting to translate the ulna head back. In the normal wrist, this will still be a smooth movement, but in the subluxed ulna head, where the ulna head is either perched on or passed beyond the margin of the sigmoid notch, the ulna will relocate with a definite jump or click. A variation of this is known as the "press test" [12]. In this, the patient rises from a chair using his or her hands for assistance, pushing against a table top located to the front. Instability is shown by greater depression of the ulna head on the affected side and is often painful.

Reproduction of the patient's symptoms in this manoeuvre is also a strong positive indicator.

Other problems are that patients with generalised ligamentous laxity can be difficult to assess and laxity generally can be asymmetrical. In addition, clicking can be produced from a TFCC tear, or any instability of the ECU tendon. Pain can also arise from a luno-triquetral instability, which can be reproduced by ballotment. On grasping the lunate between the thumb and index finger of one hand and the triquetrium in a similar fashion with the other hand AP translation should be distinct and symptomatic. Finally, pain arising from the ECU tendon or tendon sheath may be exacerbated by gripping the distal ulna. As a consequence, awareness of the limitation of this test and taking care to examine the wrist for the differential diagnoses should lead to a more accurate diagnosis.

Evaluation

Plain radiographs are useful in dysplasia, arthropathy and fracture delineation. However, the DRUJ is difficult to interpret accurately on plain films. If the DRUJ is widely separated, diagnosis is clear. However, dislocation is hard to assess

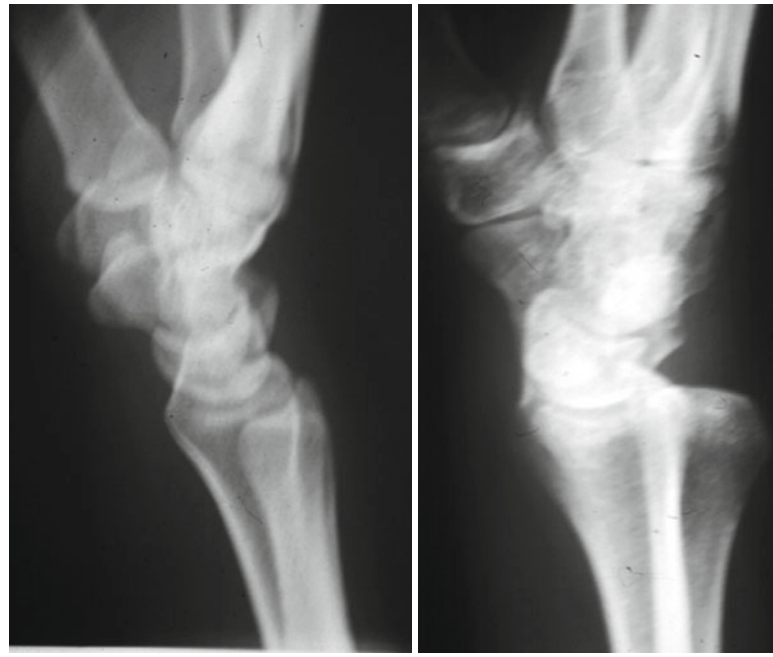

Fig. 11.8 Plain x-ray of simple DRUJt subluxation

and even if reported to be present this may prove not to be so on further imaging. Subluxation is also difficult to assess on plain radiographs (Fig.11.8). In addition, care needs also to be taken when assessing axial displacement, since the relative lengths of the radius and ulna change with forearm rotation. To assess ulna variance, the DRUJ antero-posterior film should be taken with the arm at 90° abduction at the shoulder and 90° flexion at the elbow, such that the wrist is sitting on the X-ray plate in an exact neutral position between full supination and mid pronation. This can then be compared to the normal side.

CT scanning provides the best assessment, giving clear views of the congruity of the joint. It also provides an excellent assessment of any peri or intra-articular fractures. However, little information regarding the soft tissues is revealed. The addition of arthrogaphy, to either plain films or CT, provides more information, particularly with regard to capsular and TFCC tears. False positive results can, however, arise, since central defects in the TFCC are common in the normal population, particularly after the age of 40. In addition, defects in the intercarpal ligaments can also occur, allowing contrast to pass from radiocarpal to midcarpal joints.

MRI scanning can identify the TFCC and the position of larger soft tissues, such as the ECU tendon. Generally, any TFCC tears visible on an MRI scan can be confirmed arthroscopically. There are, however, significant false negatives for TFCC and intercarpal ligaments injuries using a 1.5 T scanner, even with a wrist coil [13]. The gold standard for assessing the TFCC is still wrist arthroscopy. Obviously, the introduction of newer, more powerful MRI scanners into clinical practice, of the sort that are currently used for research, may change this. The evidence of bone bruising in relation to ligamentous attachments is a strong indicator of injury to these ligaments, as is the presence of increased signal within the capsule.

Ultrasound may be useful in the assessment of the ECU, particularly since dynamic assessment can be undertaken, which is something currently not possible with MRI or CT. Visualisation of the subluxing ECU and correlation to symptoms can be helpful. It may also be possible to assess the congruity of the DRUJ dynamically, although this assessment is very operator dependant. Finally, bone scanning with technetium 99 can be a useful screening tool for identifying occult fractures and it may also be positive in early

ligamentous disruption. Its use has, however, largely been superseded by MRI imaging.

Arthroscopy and EUA are more invasive, but can provide definitive answers. It is still the gold standard for assessment of the TFCC and the ulnocarpal ligaments, since these can be directly visualised. If there is a substantial tear of the TFCC, either from the radius or centrally, the DRUJ can be seen directly. Dynamic assessment of this joint can then be made during forearm rotation and wrist movement. In addition, EUA, either alone or in conjunction with wrist arthroscopy, can help, particularly with the translation test, which may have been proved too painful to perform in the clinic.

Treatment

Treatment of DRUJ instability is dependent on the underlying cause. It should be emphasised, however, that treatment that works well for one pathology may lead to disastrous results in others. For example, excision of the ulna head (Darrach's procedure) is a good operation that works well in older patients with advanced rheumatoid arthritis. The same procedure, if performed in a 40 year old with DRUJ pain and instability following a distal radial fracture, will usually lead to a very poor outcome. It is essential at the outset to recognise this and tailor ones treatment to the underlying pathology causing the instability and not treat instabilities of different causes in the same way, even if the instability itself appears similar.

Congenital Abnormalities

Madelungs deformity is the commonest congenital abnormality that gives rise to DRUJ instability (Fig. 11.5). In Madelungs deformity, the DRUJ is often widely separated and central and ulnar wrist pain can arise. It is important to recognise that the cause of this pain may not be the obvious dislocation of the DRUJ. The loaded wrist in Madelungs causes the carpus to sublux both in a palmar and ulnar direction. The ulna translocation is in itself a potent cause of instability symptoms. However, this is caused by carpal instability, which must be distinguished from the DRUJ instability. Increased loading with sporting activities can bring on the onset of symptoms in late teens and early twenties where none existed before. In early adulthood early arthrosis may also give symptoms that can be confused with DRUJ instability.

The more marked the Madelungs deformity, the less likely that the problem lies with the DRUJ, since the radius with marked deformity may not articulate with the distal ulna and the sigmoid notch will not have formed normally, or at all. Indeed, corrective osteotomy of such a deformity by Triplane osteotomy may create DRUJ symptoms for the first time, since such an osteotomy may bring the radius in contact with the distal ulna.

More minor dysplasias and minor Madelungs deformity may present with instability symptoms. These patients should be carefully assessed clinically and have CT imaging of the DRUJ to assess sigmoid notch development, ulna head sphericity and subluxation. If the bony development is normal and subluxation is present, a radial triplane osteotomy can be performed, correcting the palmar angulation, radial inclination and length of the radius (Fig. 11.9). The ulna may still appear to be too long and, as such, consideration should be given to undertaking an ulna shortening osteotomy at the same time. In the author's experience, however, any residual axial subluxation is well tolerated by the patient and rarely causes symptoms. As such, the author has avoided undertaking concurrent ulna shortening, preferring to perform this as an interval procedure, if the patient remains symptomatic.

Inflammatory Arthropathy

Patients with Rheumatoid Arthritis (RA) commonly develop DRUJ pain and instability. The ulna head is subluxed dorsally, giving the classic "piano-key" sign (Fig. 11.10). There is usually associated synovitis of the DRUJ and commonly of the extensor tendons as well. This dorsal subluxation puts the extensor digiti minimi pro-

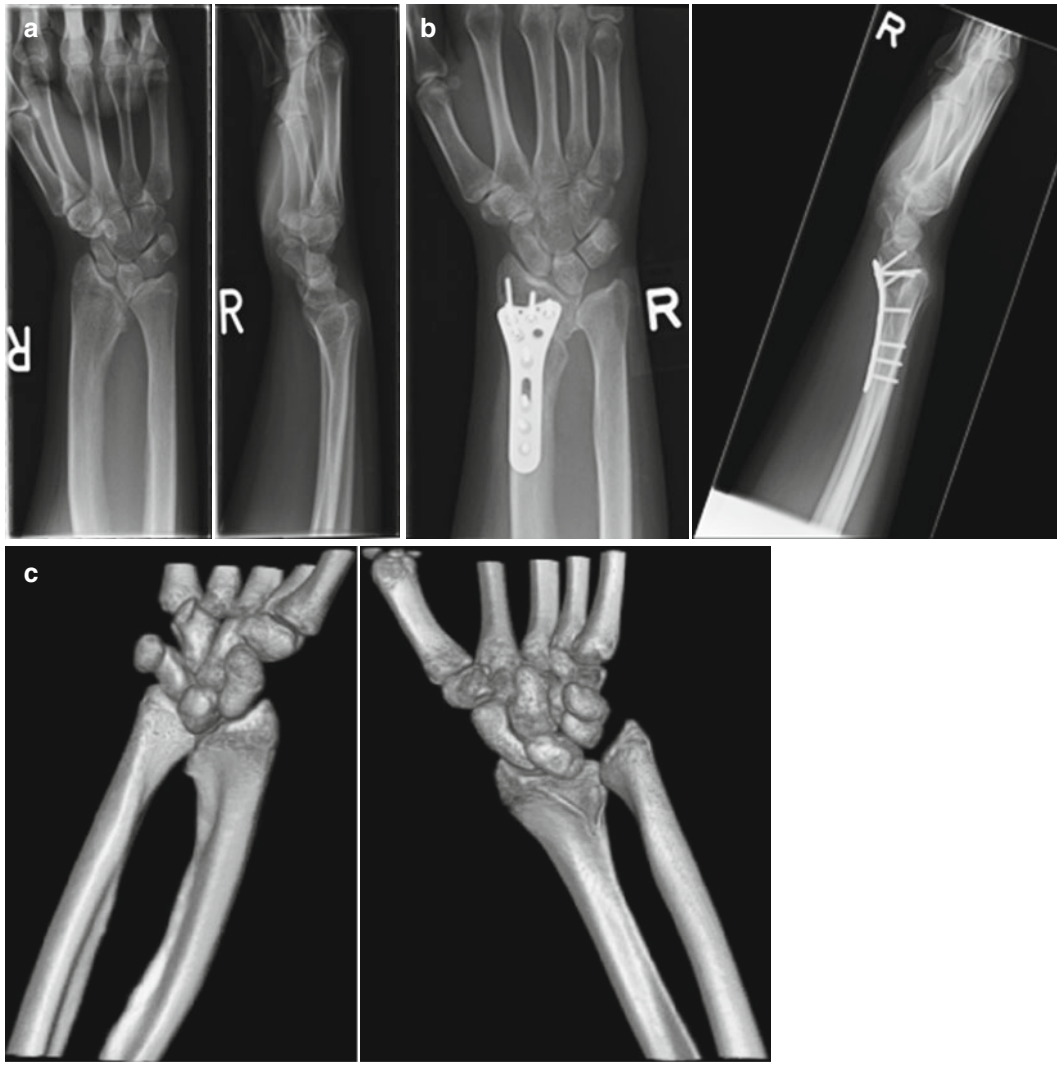

Fig. 11.9 Triplane osteotomy on the radius (**a**) Madelungs pre op. (**b**) Madelungs post operative. (**c**) Madelungs CT

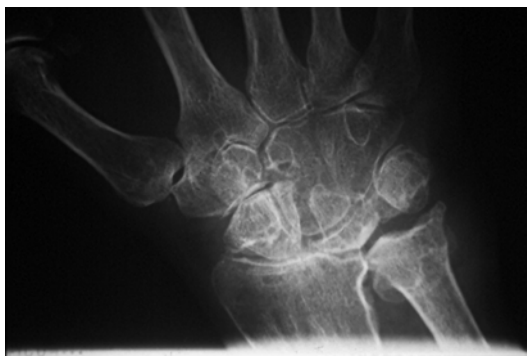

Fig. 11.10 Rheumatoid arthritis affecting the DRUJt

prius (EDMP) at risk first, since it lies directly over the radioulnar joint. In all such cases, this tendon must be carefully evaluated. If ruptured, the risk to the 4th compartment tendons is significant and these patients need urgent treatment.

The principle treatment of rheumatoid patients is medical. With the onset of the new disease modifying drugs and more aggressive and earlier medical intervention, the need for surgical intervention has decreased. These patients should be assessed jointly with a rheumatologist and a management plan formed before any surgical intervention is contemplated. If the patient has

well controlled disease but synovitis of the DRUJ persists, then injection of the DRUJ with a steroid preparation may be effective. Once the synovitis settles the pain often subsides, even though the subluxation persists. Injection in a patient with raised inflammatory markers gives benefit for a short time only and should only be considered in urgent cases, such as those in whom the EDMP has ruptured and who are having their medical treatment adjusted to control the disease.

In patients who have persistent symptoms of pain and instability surgical intervention is indicated. The choice of treatment is ulna head excision or ulna head replacement. Both these topics have been considered elsewhere in the chapter on inflammatory arthropathy. The important clinical points in surgery for these cases include the incision, the approach to the DRUJ, assessment and protection of the extensor mechanism, soft tissue stabilisation and which procedure to perform.

Currently, there are quite a range of surgical techniques in use, with little knowledge as to which procedure is optimal. The author's preferences, however, are as follows;

The incision should be midline. This easily allows access to the DRUJ and allows future extensor tendon and radiocarpal surgery (such as arthroplasty) to be performed through the same approach. An ulna incision, followed after a short time interval by the requirement for a midline incision, may lead to problems with wound healing, with particular concerns regarding the viability of the skin bridge between the two incisions. The wound should be deepened through the bed of the 5th compartment. This is to allow for any future DRUJ surgery, since this is the optimum approach for DRUJ arthroplasty (for which one must plan if instability following Darrach's procedure occurs).

If there is any concern with regard to extensor synovitis and the risk of extensor tendon rupture, the extensor retinaculum should be elevated from the 6th compartment to at least the 2nd compartment and passed beneath the extensors at the end of the procedure. If the ECU has subluxed over the ulna in a volar direction, as is often the case, then a sling using the free edge of the retinaculum can be fashioned bringing the ECU to lie over the ulna stump or ulna head. In planning this, the retinacular dissection needs to start well to the ulna side to give adequate length and the sling must be loose enough around the ECU not to cause attrition or synovitis in its own right. The relocation of the ECU tendon over the ulna head gives dynamic stability to the DRUJt.

Regarding the choice between an excision arthropasty or performing an ulna head replacement, the author would usually perform an excision arthroplasty in the rheumatoid patient, since this is simple and very well tolerated. The risk of symptomatic instability of the ulna stump if the resection is done at the correct level (just at the proximal margin of the articular surface of the ulna) is small. Subsequent revision to an arthroplasty following a failed excision arthroplasty is both a straightforward and usually successful procedure, particularly for restoring stability.

Fracture

A single bone forearm fracture which is displaced must inevitably disrupt the proximal or distal radio-ulna joint. In nearly all cases the force applied causing such a fracture is a distal indirect force. Thus, if the ulna fractures, the load will be transmitted along the intact radius to the proximal radio-ulna joint. If the ulna is either shortened or angulated, the radial head must dislocate; the standard Monteggia pattern of injury (Fig. 11.11). Likewise if the radius is fractured, the energy of the deforming force along the radius is dissipated and the distal radio-ulna joint is disrupted. As with the Monteggia injury, if the radius is shortened or angulated the ulnar head must dislocate, the Galleazzi pattern of injury (Fig. 11.12). For subluxation or dislocation to occur the soft tissues must also be disrupted. This will include the TFCC and radio-ulna ligaments as well as the interosseous membrane from the level of the radial fracture to the distal radio-ulna joint. If the fracture of the radius is proximal to the entire interosseous ligament, as would be the case in radial head and neck fractures and the fracture is shortened, the entire interosseous membrane must tear to allow the distal radio-ulna

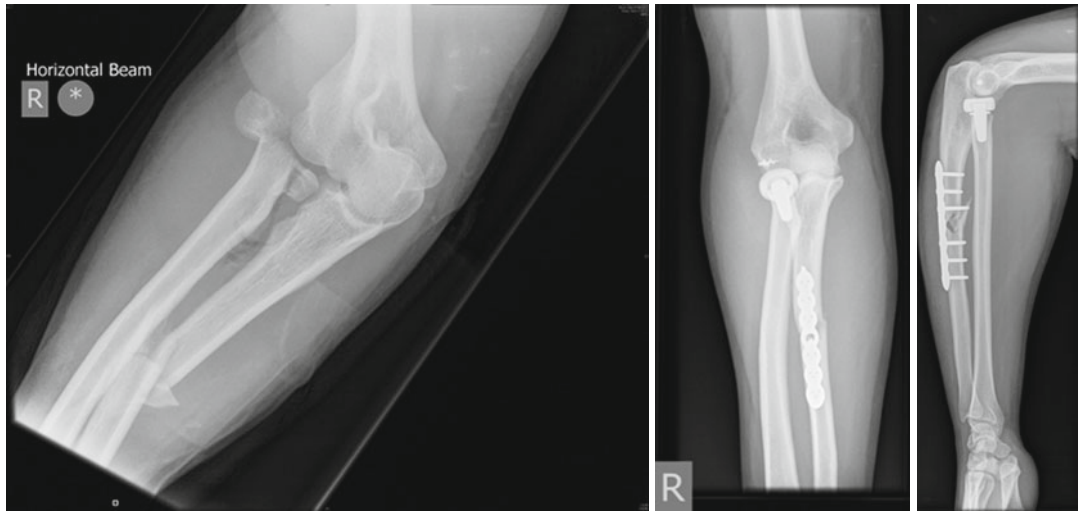

Fig. 11.11 Monteggia fracture

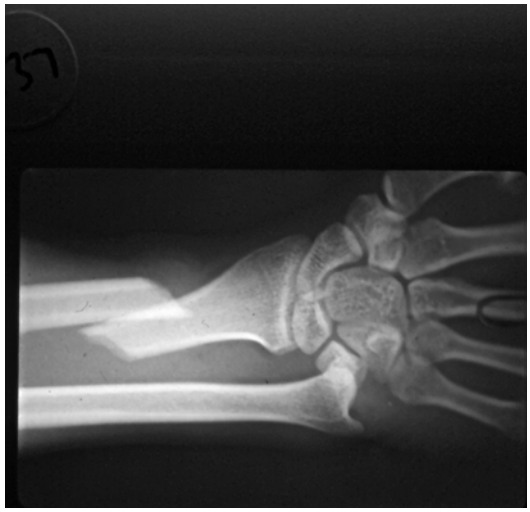

Fig. 11.12 Galleazzi fracture

joint to sublux axially, the Essex-Lopresti injury (Fig. 11.13). The ulno-carpal ligaments in this event, however, may remain intact.

When considering DRUJ subluxation or dislocation in radial fractures, one needs to consider the deformity. The angular and axial deformity should be considered in turn and treatment must be directed at treating each of these. Soft tissue procedures, undertaken in isolation, in the presence of persistent bony misalignment, either angular or axial, will usually fail. The principles of treatment are straightforward. In angular deformities, the radius must be brought back to its anatomical position by osteotomy, with or without the addition of a bone graft. With axial deformities alone, to restore the normal anatomical position it is easier to shorten the ulna to match the shortened radius than to lengthen the radius. Persistent symptoms of instability are unusual following correction of the bony abnormality. However, if this persists, additional soft tissue reconstruction can be considered.

Clinical Pearl

The commonest cause of apparent distal radioulnar joint instability is angulation or malunion of the radius. Correction of the latter will result in the resolution of instability in the majority of cases.

The more proximal the radial fracture the smaller the angulation required at the fracture site to cause disruption of the DRUJ. In proximal third fractures the degree of malunion may be so subtle as not to be clear on plain radiographs. CT scanning with 3D reconstruction can, however, be extremely helpful in delineating the angular malunion. Likewise, the accuracy of the corrective osteotomy needs to be greater and, as such,

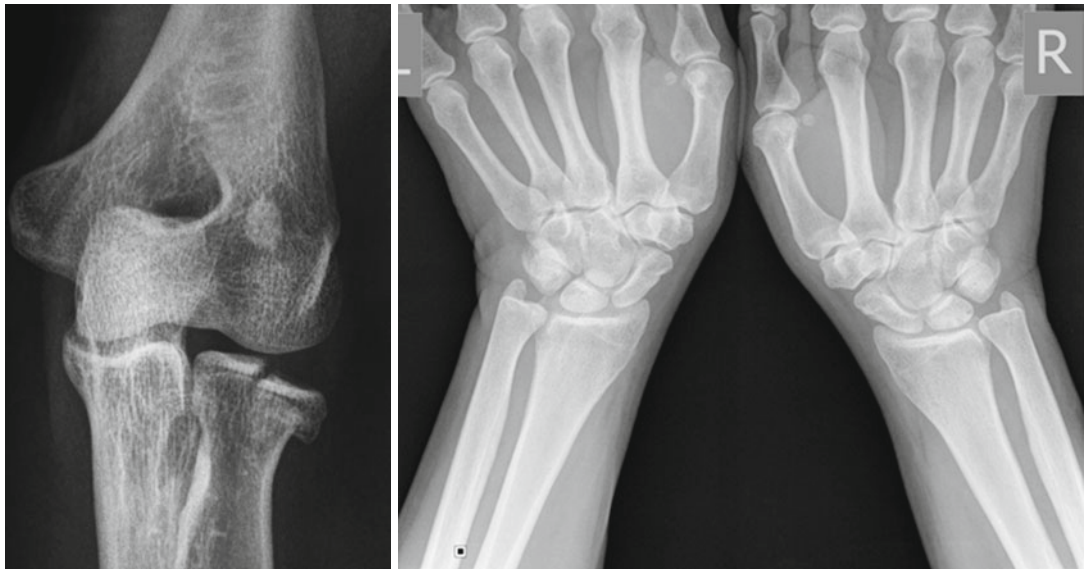

Fig. 11.13 Essex-Lopresti injury

appropriate pre-operative planning is required (Fig. 11.14). In the Essex Lopresti lesion the entire interosseous membrane is torn. Once torn, the interosseous membrane does not heal and these patients are then dependant on the healed radial head to prevent axial instability of the forearm. Unfortunately, the soft tissues around the DRUJ, even if repaired, are not sufficiently strong to prevent the deforming forces leading to further shortening. Thus, if a radial head excision has been performed, there is nothing to prevent further axial shortening. Such patients will therefore not respond to an ulna shortening, since the radius will continue to migrate proximally until either the ulna sided wrist pain returns due to further axial instability, or the radial neck 'bottoms-out' on the capitellum causing elbow pain.

The key to the management of these injuries is recognition of the severity of the initial injury at the time of the fracture. Radial head preservation by reconstruction should be the management of choice and only if this proves impossible should radial head replacement be considered. In patients who have persistent symptoms, salvage surgery by reconstruction of the interosseous membrane can be considered (Fig. 11.15). If this fails, then the formation of a single bone forearm is probably the only salvage option (Fig. 11.16).

Ulna styloid fractures and fractures of the dorsal or palmar edge of the sigmoid notch need to be considered separately. An ulna styloid fracture may be proximal or distal to the attachment of the TFCC. The majority of ulna styloid fractures are distal to the fovea of the ulna and these do not lead to instability. Fractures proximal to the TFCC attachment, however, completely detach the dorsal and palmar radio-ulna ligaments. This injury can potentially give rise to instability if associated with injury to the distal interosseous ligament. Dislocation or subluxation in this situation can be either dorsal or palmar. Any dorsal extension of this fracture will inevitably involve the ECU bed and, as a consequence, the deep ECU sheath will also be involved, as well as the dorsal capsule. Dorsal subluxation with the wrist in supination can therefore occur, since all the static restraints have thus been lost. This can be compounded by ECU subluxation. Palmar subluxation with the wrist in pronation may, in part, be stabilised by the function of the pronator quadratus.

Treatment of this injury is by reduction and internal fixation of the styloid fracture. Confirmation that the TFCC is attached to the styloid fragment is, however, obviously essential. An MRI scan may adequately define this. If, however, the MRI scan is equivocal, a diagnostic

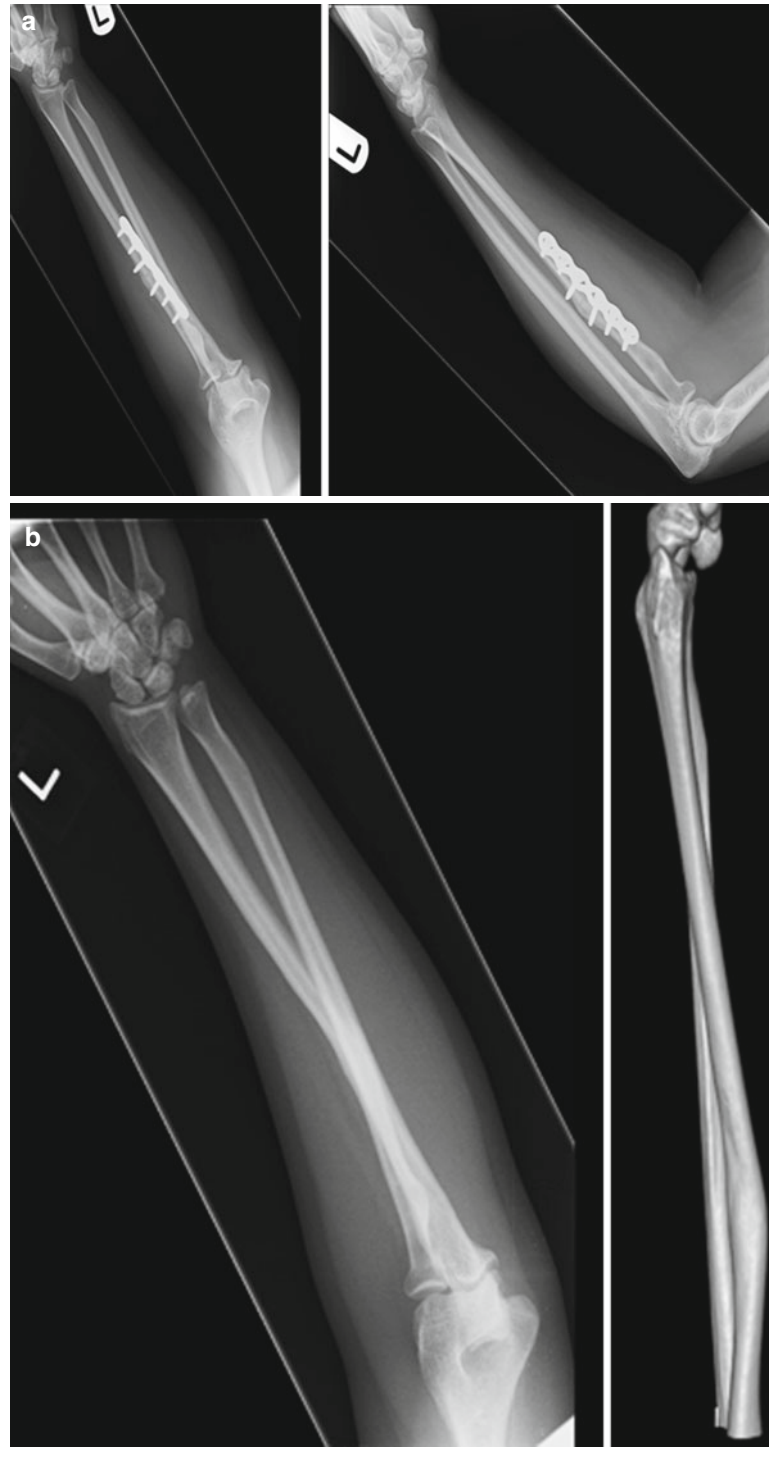

Fig. 11.14 Preop planning for radial osteotomy (**a**) Radial malunion post op. (**b**) Radius malunion XR & CT

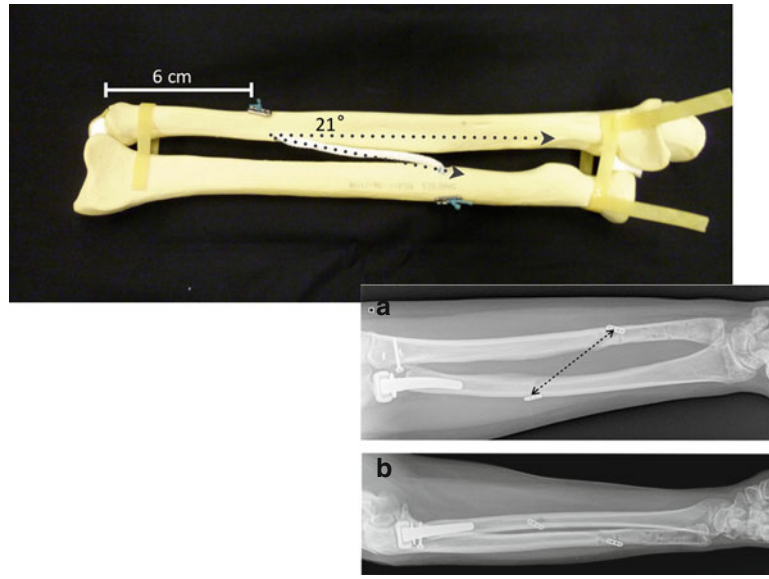

Fig. 11.15 Interosseous membrane reconstruction

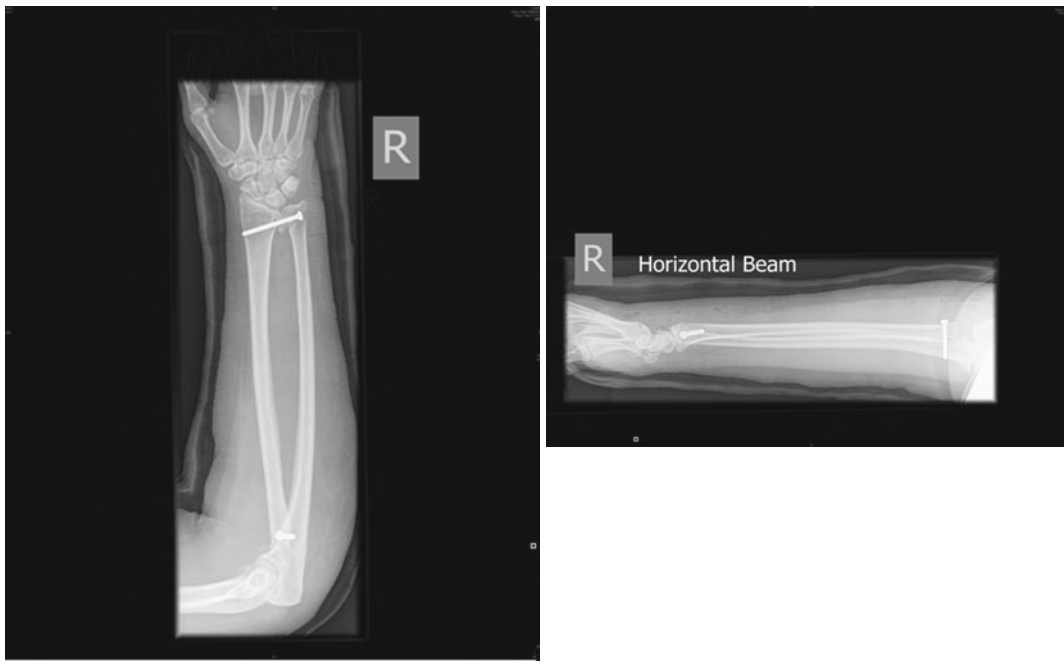

Fig. 11.16 Single bone forearm

EUA and wrist arthroscopy can be helpful. The bony fragment that includes the TFCC insertion is usually large enough to accommodate a small screw, such as a variable pitch compression screw. If the fragment is too small to accommodate such a screw the fragment may be held with a K-wire and wire suture. In established non-union, freshening of the bone ends is required to achieve

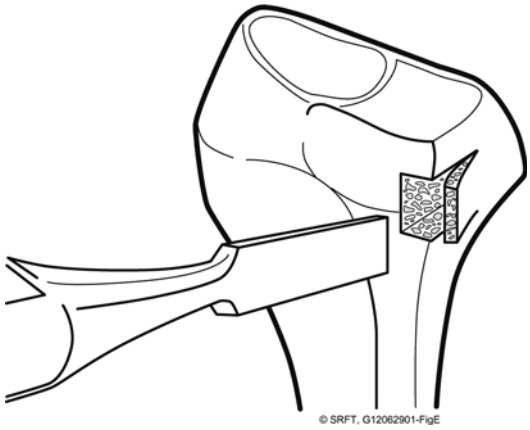

Fig. 11.17 Sigmoid notch osteotomy

union and care needs to be taken not to damage the TFCC attachment. Good cancellous to cancellous bone contact should negate the requirement for bone graft.

Fractures of the dorsal or palmar margins of the sigmoid notch cause instability, regardless of any soft tissue injury. The ulna may follow the fragment to lie in a subluxed position. This injury cannot be stabilised by soft tissue reconstruction alone and reduction and fixation of the fracture is generally required. The injury should be recognised at the time of fracture. Unfortunately, plain radiographs are not usually diagnostic, but will suggest the injury. Again, CT scanning is the best way to accurately demonstrate the bony lesion to facilitate surgical planning. Fixation must be rigid and may require buttress plating.

An alternative is to undertake an osteoplasty of the sigmoid notch. Specifically, either the anterior or posterior lip of the sigmoid notch is elevated and a bone graft inserted to fill the defect [14] (Fig. 11.17).

Soft Tissue Injury

For instability to occur at the DRUJ, following ligamentous injury only, significant trauma must have incurred. As stated previously, the triangular cartilage, dorsal or palmar radio-ulnar ligament and the distal interosseous membrane must be disrupted to allow a subluxation. In addition, the deep part of the ECU sheath and ulno-carpal ligaments will usually be involved. It should be stressed that a solitary TFCC tear, or even a complete detachment, in the absence of other ligamentous injury, will not cause instability. Thus, care must be taken to assess the full extent of the damage in patients with instability and not limit oneself to assessing the TFCC alone.

Assessment of these patients should include a careful history, paying attention to the mechanism of injury and its severity. It is unusual for the diagnosis to be made at the original presentation, in the absence of an initial dislocation. Symptoms are usually of intermittent pain and clicking and occasionally a feeling of giving way.

On clinical examination, an important sign to diagnose instability is to demonstrate excessive translation of the ulna in an antero-posterior direction, as described earlier. Reproduction of the patient's symptoms by this manoeuvre will occur if there is instability rather than laxity. Over-diagnosis can occur by not comparing the contralateral side, or appreciating that AP laxity of the patients DRUJs can be asymmetric. It is also essential that one excludes other causes of ulna sided wrist pain. ECU instability in particular may not be apparent and needs to be looked for specifically.

Plain radiographs are useful for the diagnosis of fractures, but give no help in the assessment of subluxation. Even complete dislocation can be difficult to appreciate from plain films. The congruity of the joint is best assessed by CT scanning. Whilst MRI scanning might be useful, any ligamentous injury may not be evident. Arthroscopy will demonstrate a TFCC disruption, but visualisation of the subluxation is only possible in large central or radial TFCC tears, since only in these injuries can the radioulnar articulation be fully assessed. Ulna avulsions of the TFCC are the usual pattern of injury in instability. The ability to dislocate and recolate the DRUJ during an examination under anaesthetic is diagnostic.

Overall, it is clear that diagnosis is not easy for either the generalist or specialist. As such, the clinician needs both careful consideration of the entire differential diagnosis, as well as having a high index of suspicion of the injury. It should further be stressed that DRUJ instability from ligamentous injury alone is rare. TFCC injuries and instability of the ECU are much more common diagnoses for intermittent ulna sided wrist pain and clicking.

Treatment for these patients is straightforward if the TFCC is reconstructable. A repaired TFCC which makes the dorsal and palmar radio-ulna ligaments competent will stabilise the joint, regardless of the extent of the other ligamentous injuries. The first stage in these patients would be an arthroscopy to assess the state of the TFCC. With an ulna avulsion, arthroscopic or open repair should be undertaken. Ideally, the repair should bring the ulna part of the TFCC back into the fovea of the ulna, at the base of the ulna styloid. This is best done by drilling paired drill-holes at the base of the ulna styloid and suturing the TFCC back into the fovea. Suturing the TFCC to the capsule adjacent to the base of the styloid may also give enough stability.

If the TFCC is not reconstructable, either due to a radial tear or complete disruption, a soft tissue reconstructive procedure to recreate the dorsal and palmar radio-ulna ligaments is required. This can be done with a strip of tendon or the use of a free palmaris longus graft. Several techniques have been described [15, 16]. Those with the greatest success aim to recreate the original anatomy of the DRUL and PRUL, achieve a sound bony healing of the tendon to the palmar and dorsal edges of the radius and the fovea of the ulna and to correctly judge the tension. The most commonly used technique is that described by Adams in 2000, using a strip of palmaris longus [17]. A tunnel is passed through the radius under fluoroscopic guidance. Using a similar technique, an obliquely directed tunnel is created in a distal ulna between the fovea and the ulna neck. A tendon is passed through the radial tunnel and then both ends through the ulna tunnel, thereafter, being fixed to the shaft of the ulna. The latter can be undertaken by various methods, including bony anchors (Fig. 11.18a, b). Initial reports have been promising [18].

Post operatively the forearm should be rested in neutral rotation until healing has occurred at 6 weeks. If the repair is sound then forearm rotation may be allowed. However, the patient needs to be compliant in restricting their activity. A reasonable compromise is to place the patient in a forearm cast, which allows some forearm rotation. This restriction limits the patient's activity enough so as not to put the repair at risk.

Clinical Pearl

In the rare instance of pure soft tissue abnormality, the optimum treatment would involve reconstruction of the anterior and posterior radioulnar ligament, by use of a tendon graft.

Summary

Instability of the DRUJ is most commonly associated with bony injury, inflammatory arthropathy or congenital abnormalities. In all cases, both the axial and antero-posterior components of instability should be considered. Clinical assessment is not straightforward and requires a clear understanding of the extensive differential diagnosis. Treatment must be directed at the cause. Successful operations for instability caused by one aetiology may not be successful in treating apparently similar instabilities of different aetiologies. Thus, soft tissue reconstruction in patients with malunion or congenital anomalies will not succeed, despite being successful in patients with ligamentous injuries. Likewise, excision of the ulna head is successful in patients with rheumatoid arthritis, but leads to poor outcomes if performed for trauma.

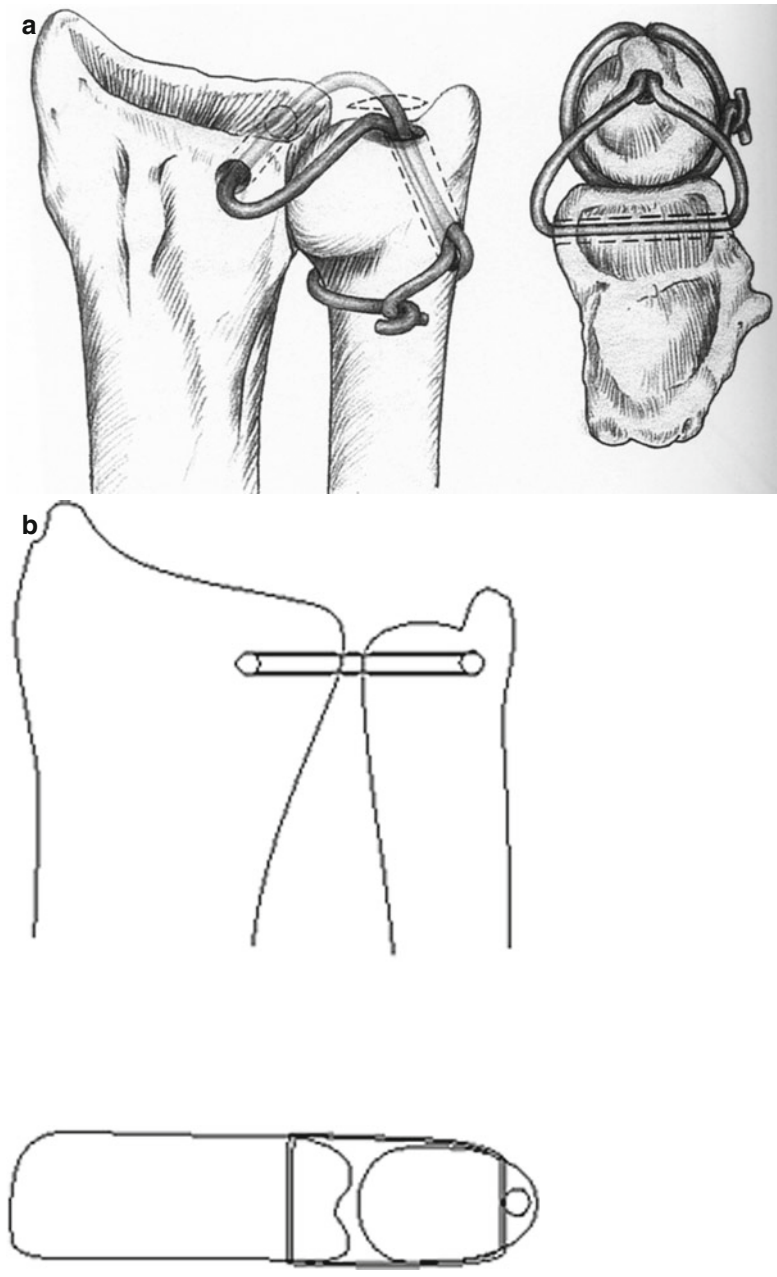

Fig. 11.18 (**a**) Adam's procedure. (**b**) Trail modification

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Part II

Nerve Compression

Carpal Tunnel Syndrome

12

Ian S.H. McNab and Sarah Tucker

Keywords
Aetiology • Acute • Chronic • Clinical diagnosis • Nerve conduction studies • Non-surgical treatment • Surgery • Dual portal release • Complications

Introduction

Carpal tunnel syndrome (CTS) is a collection of symptoms and signs arising from compression and/or traction on the median nerve at the level of the carpal tunnel. It is a common condition, with a prevalence of 3.8 % in a healthy population and is probably on the increase, related to an increasing mean Body Mass Index (BMI) [1, 2]. It is the commonest nerve compression syndrome and is therefore of great interest to health purchasers and providers alike. The response to the financial burden of treating carpal tunnel syndrome has been varied throughout the UK. In some areas carpal tunnel release (CTR) has been designated as an operation that requires exceptional funding, whilst in other areas it is available on a one stop clinic basis. The 2013 tariff for CTR is between £900 and £1,250 and it is likely to be one of the conditions that is very appealing to healthcare providers, in that it is usually simple to diagnose and straightforward to treat [3]. CTS is a clinical diagnosis, requiring assessment by a clinician with sufficient experience to filter out those patients that need investigation of an alternative cause of their symptoms. There is concern that the oversimplification of the treatment pathway, if there is no assessment by a hand surgeon, may lead to problems such that some patients may undergo unnecessary or unsuccessful CTR surgery before it is established that CTS is an incorrect diagnosis. Also the operation, though usually straightforward, is not without complications which can, on rare occasions, be catastrophic.

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Aetiology

Normal nerve function is dependent on the nerve being free to glide without constriction or localised pressure. Nerve compression symptoms of tingling and numbness occur when there is a

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mismatch between the space available for the nerve and the space needed by that nerve and/or tethering of the nerve.

Gelberman measured pressures within the carpal tunnel and showed that patients with CTS have a mean pressure of 32 mmHg, compared to a mean of 2.5 mmHg in controls [4]. In chronic compression there is a physical deformation effect, as well as an ischaemic effect. The deformation leads to detachment and retraction of myelin seen as a slowing of conduction. The ischaemic effect reduces the efficiency of the Na+ and K+ pumps and alters the fibroblast to schwann cell balance, which in time leads to fibrosis within the nerve. This is seen as loss of conduction and failure to transmit an impulse.

Ultrasound studies have shown that nerve gliding is reduced in patients with CTS, when compared to the normal population [5].

A mismatch in available space may be due to a narrow tunnel, a reduction of the space available within the tunnel, or an increase in the volume of the nerve itself. Obviously a combination of these factors is possible and may give rise to symptoms with very small changes in one of them. There are also a number of conditions in which the nerve shows an increased susceptibility to the effects of compression.

Narrow Tunnel

A case-control study of patients with carpal tunnel syndrome has shown that the mean wrist depth to wrist width ratio is lower in symptomatic patients, although this difference is small and may in fact be due to the difference in BMI [6]. Flexion and extension of the wrist further reduces the space available, accounting for the onset of symptoms in these positions, for example when driving.

Reduction in Space Available Within the Tunnel

This may be due to an inflammatory process, tumour or anomalous structures such as a persistent median artery or the muscle belly of a long flexor extending into the carpal tunnel. Space occupying lesions were found to be the cause of CTS in 3 % of cases, in a series of 779 cases, of which almost half were tophaceous gout, tenosynovitis, ganglia, lipomata and fibromata [7].

An Increase in the Volume of the Nerve

A common example of this is in diabetes, where changes in the phospholipid content within the nerve increases its volume.

Nerve Susceptibility to Compression

Diabetes

Diabetes also impairs axonal transport mechanisms within nerves, rendering them more susceptible to the adverse effects of compression. This may account for the incomplete recovery of the nerve seen in diabetics after decompression.

Acromegaly

Acromegaly gives rise to an increase in the endonuerial tissue, in which carpal tunnel syndrome may be superimposed on a peripheral neuropathy. CTS may be the first presenting symptom in acromegaly, so this diagnosis should be considered and those patients known to have acromegaly need to be aware of the possibility that they may develop CTS, so that they seek medical advice at the appropriate time.

Hypothyroidism

Hypothyroidism leads to axonal degeneration and altered conductivity and CTS may again be the first presentation. Administration of thyroxine usually reverses the changes relieving the symptoms.

Hereditary Liability to Pressure Paralysis (HLPP)

Hereditary liability to pressure paralysis (HLPP) These are rare disorders, occurring in a

small number of families, in which there is a reduced axonal diameter and poor development of the myelin layers. Patients may get recurrent lesions involving peripheral nerves that usually resolve spontaneously but may leave them with persistent paralysis. Some of these patients have been found to have electrophysiological changes at expected entrapment sites without the expected symptoms. These disorders are due to dysfunctional voltage gated ion channels which may also be referred to as channelopathies.

The Double Crush Phenomena

The double crush phenomena is a clinically observed situation in which a nerve that is compressed at one site appears to have an increased susceptibility to symptoms from compression at another site. This is probably explained by an effect on axonal transport.

Acute CTS can occur in association with wrist trauma, or secondary to sudden severe swelling in the carpal tunnel, such as after a bleed from a vascular malformation. This will present as rapidly evolving paraesthesia in the median nerve distribution, progressing to numbness if untreated. The threshold for this to occur will also be lower in any patient who already has any of the predisposing factors above.

The aetiology in this case is rapid onset of ischeamia, secondary to the sudden increase in pressure and is in effect a compartment syndrome of the carpal tunnel. As such, it needs to be released as a matter of urgency to prevent permanent nerve damage. An urgent carpal tunnel release should be performed, if necessary, using an extended approach to also address the underlying cause.

Presentation, Investigation and Treatment Options

If the patient's history and clinical findings are typical for CTS, the diagnosis can usually be made on assessment by an experienced clinician. However, the addition of nerve conduction studies (NCS) may be helpful when there are atypical findings and other confounding factors, such that the diagnosis is not as clear cut.

Patients with CTS typically present with a history of intermittent tingling in the hand, especially at night when the hand has assumed a flexed posture. The patient may be able to localise the tingling to the palm and radial digits of the hand on questioning. This often wakes the patient, who will demonstrate how they shake their hands until the tingling stops. Symptoms may also be associated with activities in which the hand is held in flexion, such as reading a book, driving, knitting and typing. With time, tingling will become more frequent and persistent, progressing to intermittent numbness and then to permanent numbness of the area innervated by the median nerve. The tendency to drop things may be noticed at quite an early stage as the sensory and proprioceptive fibres are affected. Weakness presents later, but becomes progressively worse in untreated CTS.

Hand pain is often a feature and may also be referred proximally into the forearm.

Symptoms that come and go without obvious provocation and are intermittent may indicate cervical involvement.

Examination (Table 12.1) follows the same routine as with any nerve compression syndrome;

Inspection, together with an examination of sensation to the digit. Motor power, particularly of the intrinsic, following by provocation tests of all potential compression sites along the nerve.

Provocation Tests

Tinel's Sign

The term 'Tinel's sign' in this context is actually a misnomer, as Tinel described paraesthesia

occurring in the distribution of a nerve when the nerve has been injured and percussion is used distal to the point of injury. This is specifically a sign indicating progression of recovery following a nerve injury, by eliciting symptoms from the immature advancing nerve ends. However, it is very commonly used in the context of carpal tunnel syndrome and other compression syndromes where it is taken to mean paraesthesia provoked by percussion at the site of the compression.

Percussive tapping is used to provoke the symptoms and should be started from the index or middle finger distally to proximal to the elbow following the line of the median nerve.

Table 12.1 Examination of typical findings in carpal tunnel syndrome

Tips on examination technique	Typical findings in early CTS	Typical findings in late CTS	Other findings that indicate a possible alternative diagnosis
Inspection			
Look for wasting of thenar eminence and trophic changes to the pulps and skin	Often no abnormal findings	Indentation of thenar eminence due to wasting of abductor pollicis brevis or flattening of whole thenar eminence	Interrosseus wasting may give rise to
		Trophic changes of the pulps and skin corresponding to the median nerve territory	Intermetacarpal guttering – ulnar nerve or T1 involvement
Sensation			
Changes in light touch are very sensitive. Test at the pulp of the index finger and the pulp of the little finger to see if the patient can detect a difference between the median and ulnar territory.	May not have any changes initially, or may describe index sensation as reduced compared with the little finger	Progresses to complete loss of sensation in median innervated area	Numbness including the little finger – consider ulnar involvement
Two point discrimination is an innervation density test rather than a threshold test, so will not show any detectable change until very late. If an objective measure is required then Semmstein Weiss monofilaments are a threshold test and therefore more sensitive.			Reduced sensation in a glove distribution – consider peripheral neuropathy as may be seen in diabetes Paesthesia or numbness in a dermatomal distribution, e.g. Thumb and index for C6, middle finger for C7 – consider radiculopathy
Motor power			
Test all muscle groups innervated by the median nerve; Distal to CT; Abductor pollicis brevis Proximal to CT; Flexor pollicis longus – e.g. use the 'O' sign (see Fig. 12.1) Flexor digitorum superficialis to all digits and flexor digitorum profundus to index and middle Flexor carpi-radialis	Often full strength maintained	Progressive weakness of muscles innervated distal to CT	Weakness of muscles innervated proximal to CT may indicate other pathology or compression more proximally, such as deep to the heads of PT or at the neck

Table 12.1 (continued)

Tips on examination technique	Typical findings in early CTS	Typical findings in late CTS	Other findings that indicate a possible alternative diagnosis
Provocation tests (see text for description)			
'Tinel's' sign	Often positive at carpal tunnel	May become negative in very late stages	May be positive in proximal forearm on compression deep to PT
Phalen's test	Usually positive, but may take 40 s or more to elicit paresthesia	Positive within a few seconds, may go numb rather than experience paraesthesia. In very late cases where numbness is dense and permanent no change in symptoms can be elicited and the test is negative	
Pronator provocation	Negative	Negative	May be positive if compression is occurring deep to heads of PT

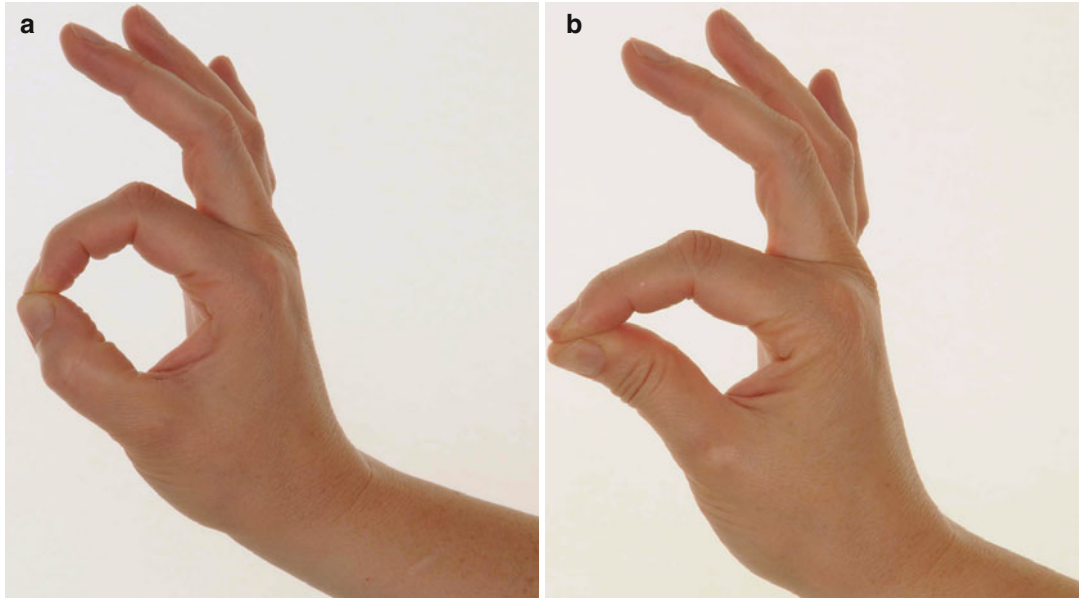

Fig. 12.1 (**a**) The O sign for testing FPL Shows a competent O and the strength of FPL can be tested by the examiner linking a finger through the O and attempting to break it against resistance from the patient. (**b**) When FPL is very weak the patient is unable to make an O as seen here

The patient should be asked before the percussion is started to indicate if at any point it causes unpleasant tingling. Symptoms elicited at the CT indicate likely compression, although percussion should continue proximally to check whether they are also elicited at the pronator teres (PT) site.

Phalen's Test

In 1966 Phalen described his wrist-flexion test in which 'the patient is asked to hold the forearms vertically and to allow both hands to drop into complete flexion at the wrist for approximately 1 min' [8]. Figure 12.2 shows the posture the hands should be placed in to perform Phalen's

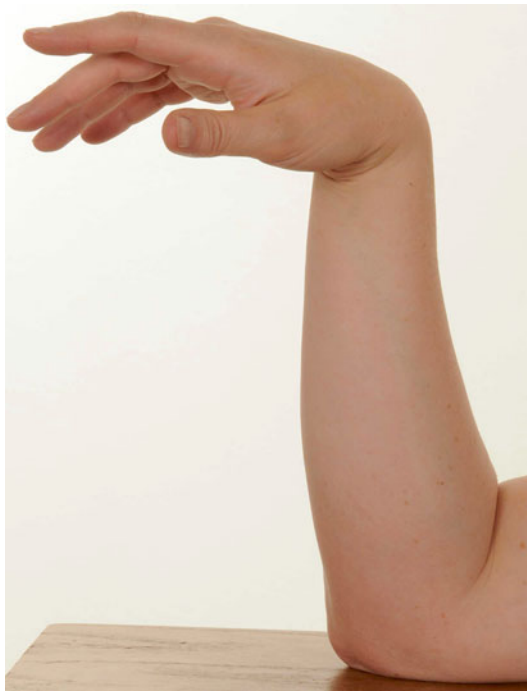

Fig. 12.2 The position of the hands for eliciting Phalen's sign

test. The test is positive if it produces CTS symptoms in less than 1 min.

Many variations on this test have been described, some of which are thought to be more sensitive, although this may be at the expense of a higher false positive rate. Also, most of them involve an amount of pressure applied either by the examining doctor or by the patient pressing their hands together. Both of which introduce an element of variability that may make the sign less reliable and comparison between case series' more difficult.

Pronator Provocation

The space available for the nerve deep to PT is reduced by resisted contraction of the muscle with the elbow extended. If the gap between the two heads of pronator is palpated and light pressure applied, the patient will experience discomfort if compression is present. Rarely, however, actual paraesthesia is elicited.

It can be seen from the table that in the early stages there is little to find on examination, other than possible positive provocation signs. However, if there is a clear history of typical symptoms and one or more positive provocation signs, this is usually enough to make a diagnosis of CTS. In later stages, provocation tests may become negative, but other signs become apparent. If the history or findings are NOT typical then nerve conduction studies (NCS) should be considered in order to confirm or exclude the diagnosis.

Clinical Pearl

If there are any findings indicating possible compression by pronator teres (PT) then nerve conduction studies (NCS) should be used to assess involvement of the nerve at this level.

However, when a referral for NCS is made to confirm the diagnosis of CTS many centres would only carry out a focused examination at the carpal tunnel level. Therefore, if there is suspicion about possible compression at PT level, then these clinical findings must be included in the NCS referral to help guide the appropriate tests – i.e. for the NCS to assess the median nerve at the CT and more proximally in the forearm. Similarly, if symptoms extend into the ulnar nerve territory this should be highlighted.

Treatment Options

Treatment options that have been found to be of benefit include splinting, steroid injection and surgery.

Ultrasound treatment may be beneficial, but the quality of the evidence is poor and the benefit is probably no greater than that seen with other conservative treatments [9]. Lastly, there is no evidence of any benefit from the use of diuretics [10].

Splinting

A recent Cochrane review concluded that many of the trials for splinting were poorly conducted

and provided only weak evidence of beneficial effect. However, where patient's symptoms are mainly nocturnal, a night splint can be very effective at relieving their symptoms in the short term, with benefit being experienced up to 1 year from the initiation of treatment [10].

Clinical Pearl

The splint needs to be positioned, such that it holds the wrist in neutral, which maximises the cross sectional area within the carpal tunnel. However, many commercially available wrist splints are designed to support a painful wrist in a functional position and they therefore hold the wrist in 20–30° of extension. This is not appropriate for carpal tunnel syndrome, as it increases the traction on the nerve and reduces the space within the tunnel. As a consequence, patients who are using off the shelf splints may experience little benefit, or even be made worse.

Steroids

Treatment of carpal tunnel syndrome with injection of steroids into the carpal tunnel has also been the subject of a recent Cochrane review.

The conclusion was that:

Steroid injection provided clinical improvement 1 month after injection, compared with a placebo,

Compared with oral steroid, an injection of steroids at the carpal tunnel provided greater benefit for up to 3 months,

The improvement seen with steroid injection is not significantly greater than that seen with anti-inflammatory medication and splintage,

Improvement is no greater with 2 injections than 1

Long term benefit has not been demonstrated [11].

Relief of symptoms with steroid injection is both diagnostic and predictive of a beneficial outcome from surgical release. It may, therefore, be useful in those cases where there is doubt over the diagnosis and symptoms are severe enough to warrant an invasive diagnostic procedure that may also give temporary relief. However, the injection requires expertise, so injection into the carpal tunnel by an untrained practitioner should not be used as a substitute for expert clinical review. Long term effects of steroid injection into the carpal tunnel are not known and, in the author's experience of referrals following injection in the primary care setting, complications of injection can include complex regional pain syndrome and tendon rupture. In conclusion, steroid injection has a limited place, but can be offered to patients with mild symptoms of recent onset, in whom splintage has not sufficiently relieved their symptoms. Follow up, or clear instructions to return if symptoms are not relieved within 4 weeks, should be given so that there is no undue delay to further investigation or treatment. Also, the likelihood of the temporary nature of the relief should be explained to the patient, along with the advice that if symptoms return surgical treatment should be considered.

Surgery

Surgery has been shown to be more effective than steroids or splinting and many patients who are managed with these non-operative measures will at some stage require surgical release [12].

Surgical Techniques and Rehabilitation

Surgical Techniques

Surgical decompression should be considered where symptoms are of greater than 6 months duration and progressive, or severe. Surgical approaches can be divided into

Open

Endoscopic

- Single portal
- Dual portal

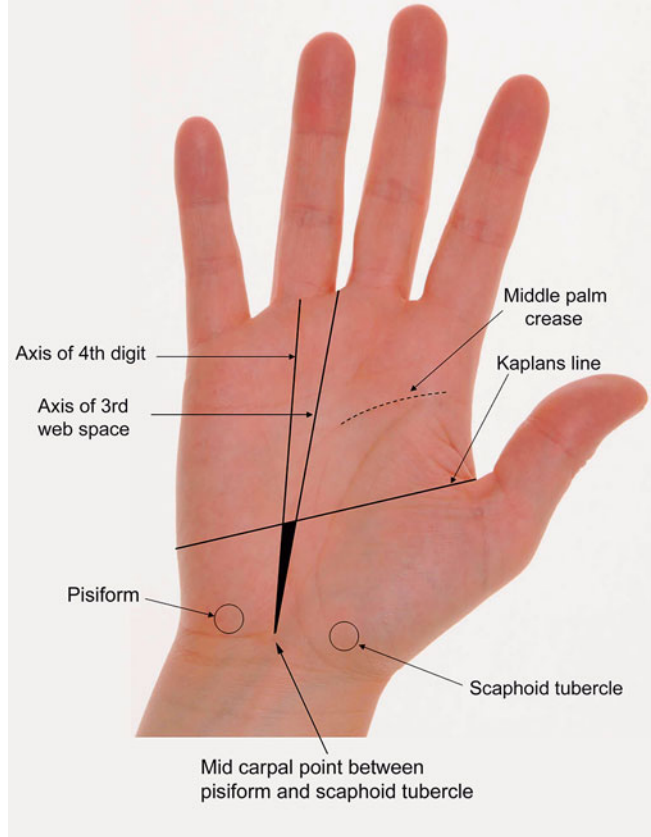

Fig. 12.3 The safe zone for incisions in carpal tunnel release

Open Carpal Tunnel Release (CTR)

Open CTR can usually be carried out under local anaesthetic. A tourniquet is not necessary if bupivacaine with adrenaline is used and infiltrated with sufficient time to allow maximal effect from the adrenaline [13].

The incision needs to be within the safe zone to avoid injury to the palmar cutaneous branch of the median nerve and to approach the median nerve from its ulnar side to minimise risk of injury to the recurrent motor branch. Common approaches involve a longitudinal incision from a point midway between the pisiform and the scaphoid tubercle, either along the midline of the 4th ray or towards the 3rd web space. The distal extent of this incision is usually limited to Kaplan's cardinal line. This was originally described by Kaplan in 1953 as a line from the apex of the first web space parallel to the middle palmar crease [14]. This marks the distal extent of the transverse carpal ligament (TCL) and the superficial palmar arch is always distal to this. The space between these limits can therefore be considered as the safe zone (Fig. 12.3). However, if a good view of the distal part of the dissection cannot be obtained the skin incision can be extended judicially in order to provide safe exposure, as long as the surgeon is aware that they are approaching the area where critical deep structures are likely to be present. Proximally, the incision can be extended if exposure is needed proximal to the distal palmar crease, with either a short transverse limb along the distal palmar crease in an ulnar direction, or a zig zag across it.

After incising the skin, the incision continues down through the subcutaneous fat and the palmar fascia before dividing the TCL. It is essential that the surgeon is cognisant, not only of the normal anatomy, but also of the anatomical variations as have been described, in order to minimise the risk of nerve damage [15]. The recurrent motor branch is particularly at risk when it traverses the

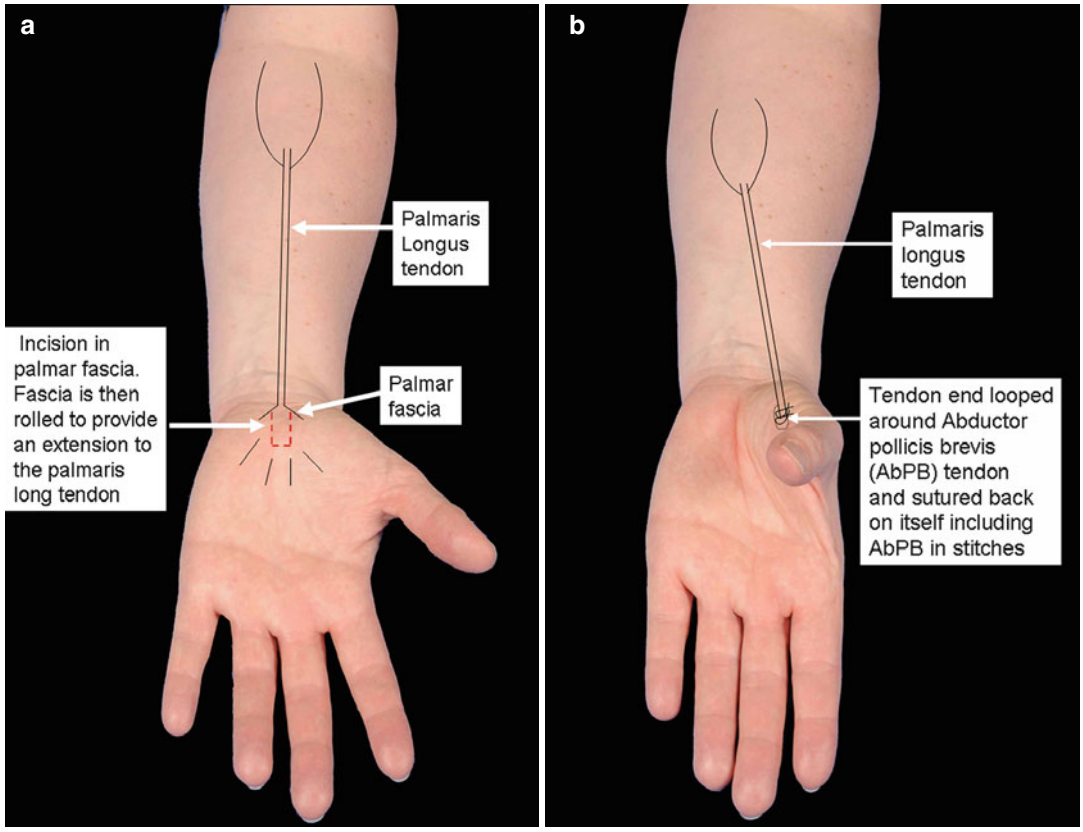

Fig. 12.4 The Camitz Procedure; Transfer of Palmaris Longus to the base of the thumb to provide abduction. (**a**) The tendon to palmaris longs is released distally with an extension from the palmar fascia (**b**) this is attached to the Abductor Pollicis Brevis tendon to abduct the thumb. This transfer therefore provides abduction but also places the thumb in a position from which FPL can provide some opposition

TCL, or emerges from the ulnar side of the median nerve. Incision of the TCL should be carried out with great care and ideally loupe magnification, proceeding both proximally and distally until there is a complete release. Distally this is indicated by the exposure of the palmar fat pad, with appropriate care being taken to protect the superficial palmar arch. Proximally, the release should include any thickened or tight distal forearm fascia. The completeness of the release can be tested by carefully inserting a Macdonald's dissector along the line of the nerve – there should be plenty of room for it to move freely.

The result of a review of randomised control studies of internal neurolysis concludes that there is no evidence of any benefit, indeed the increased scarring on a nerve would seem to indicate that it may actually be harmful [16].

In cases with severe muscle weakness and loss of opposition a Camitz procedure (Fig. 12.4) can be performed at the same time. This provides mainly abduction rather than opposition and is weaker than the alternative opposition transfers. However, it places the thumb such that the action of flexor pollicis longus can be used to provide some opposition.

Skin closure should be with interrupted sutures, with either a rapidly absorbed suture such as Vicryl Rapide ©, or a nonabsorbable monofilament.

Post-operative Regime for Open CTR

Traditionally patients were splinted post-operatively and told to refrain from heavy use of the hand for up to 6 weeks. However, a well designed randomised control trial showed that patients who are splinted

post-operatively have more scar pain and take longer to return to work than those who have a padded dressing removed on the first day after the operation and were then allowed full active motion of the fingers [17]. In this trial, the mean number of days for unsplinted patients to return to activities of daily living was 6, light duty work 15 and full work 17 days. These findings are confirmed by a review of subsequent randomised control trials, showing that there is no benefit in splinting CTR patients post-operatively and that patients instructed to return to all normal activities at 2 weeks showed no adverse effects from doing so [18].

Authors preferred regime:

Small dressing to incision, followed by padded wool and crepe

Instructions to rest and elevate the hand for 24–48 h

Reduce the dressing and start to use the hand after this and carry out a home physiotherapy regime

Wound check at 7 days

Return to full normal use at 2 weeks

Expect to have some discomfort on weight bearing to the base of the hand for 3–6 months, but not to be concerned by this as it will usually settle

Endoscopic Carpal Tunnel Release

Indications for endoscopic release include pressure to return to work, particularly in bilateral cases and avoidance of a palmar scar where patients need to do a significant amount of weight bearing – for example in paraplegic patients who self propel in a wheelchair. Cost implications for the equipment prevent the universal usage of endoscopic techniques in the absence of a significant benefit for most patients as the return to full function is little different than that following an open technique and pillar pain can still be a problem [19]. What is relevant, however, is that surgeons who do offer endoscopic release do need to ensure that they undertake a sufficient number of cases to maintain their skills.

Dual Portal Technique

A 1 cm transverse incision is made midway between the proximal and distal wrist creases ulnar to palmaris longus. A Macdonald's blunt dissector is introduced through this incision into the carpal tunnel and used to clean the dorsal surface of the TCL – this action provides tactile feedback on the texture of this surface confirming that the correct space has been entered. The trocar and slotted sheath is then introduced into this same space and the second portal site is defined by palpating the trocar tip and incising over it as close to the distal edge of the transverse carpal ligament as possible. The trocar and slotted sheath are then advanced out through this second portal to exit the skin and the central trochar can be withdrawn. In this way the slotted sheath is present within the carpal tunnel, with the slot facing volarly. The camera is introduced into the slotted sheath from distal to proximal and a hook knife is inserted through the proximal portal, so that the TCL can be divided under direct vision. The metal casing is visible outside of the hand, meaning the correct alignment of the slot can be maintained and confirmed at all times.

Single Portal Technique

In this technique only a proximal portal is required and a camera and blade are inserted from proximal to distal. The CTR is then divided in a single distal to proximal movement, taking care not to alter the alignment of the blade, as confirmation of the correct alignment is more difficult once division of the TCL has been started.

Post-op Regime for Endoscopic CTR

Minimal dressings are applied and the patient is allowed to return to normal use of the hand as soon as it is comfortable to do so, elevating the hand when at rest.

Outcomes

There is very little known about the natural history of carpal tunnel syndrome. The functional impairment in severe cases can be extreme and therefore treatment is indicated in order to relieve symptoms and to prevent permanent nerve damage and functional impairment. However, there is some evidence that this expected progression may not always occur, as shown in a review of the outcome at 6 years of those patients who were listed for CTR but cancelled their surgery. In this paper, improvement was seen in both those who cancelled their surgery and their matched controls who did have surgery, although the improvement was greater in the latter. Also, the rate of recovery is not known in the untreated group [20].

CTR is considered to be a very effective, satisfying and reliable operation, as 87 % of patients rate their outcome as good to excellent. However, in one study a return of symptoms in 57 % was observed at around 2 years later, although these were rarely of sufficient severity to cause the patient to seek further medical assistance [21]. In this same report it was noted that the number of patients involved in heavy occupational use of their hands dropped from 27 % of patients pre-operatively to 6 % post-operatively.

Outcomes are similar between open CTR and endoscopic CTR as shown by a randomised control trial of the two techniques, with the exception of return to work. This was on average 8 days earlier in the endoscopic CTR group, with the additional cost per patient totalling £98 [19] However, the patients undergoing open CTR were not instructed to return to full normal activity at 2 weeks, although this is now known to be an acceptable time frame for recovery (see above).

Complications of Treatment

Complications

Complications include haematoma, bleeding, infection, nerve damage, pillar pain and complex regional pain syndrome. A review of 186 patients who had undergone CTR by expert hand surgeons found 34 complications in 22 patients. These were incomplete division of the TCL in 12 cases (6 %), damage to the palmar cutaneous branch of the median nerve in 11 cases (6 %), complex regional pain syndrome in 4 cases (2 %), hypertrophic scarring, palmar haematoma and bowstringing of the tendons each in 2 cases (1 %) and adhesions of the flexor tendons in 1 case (0.5 %) [22]. These are acceptable figures, with no major transection of the median nerve. However, in a postal questionnaire of 708 hand surgeons in the USA, concerning complications they had treated (though not necessarily caused) from CTR in a 5 year period, there were 495 cases of laceration of the median or ulnar nerve or one of their branches and 88 tendon lacerations [23]. This finding shows that there are a significant number of severe complications from CTR in the wider setting, where not all the primary surgeons would have been specialist hand surgeons.

Recalcitrant and Recurrent Cases (Fig. 12.5)

If a patient is found to still have symptoms at review, the history, examination and investigations must be revisited very carefully in order to determine the management pathway.

In a review of 200 cases requiring further surgical interventions the findings were of incomplete release in 54 %, tethering of the nerve in scar tissue in 23 %, circumferential fibrosis around and within the nerve in 8.5 %, nerve laceration in 6 %, tumour in 0.5 % and no specific cause found in the remainder [24].

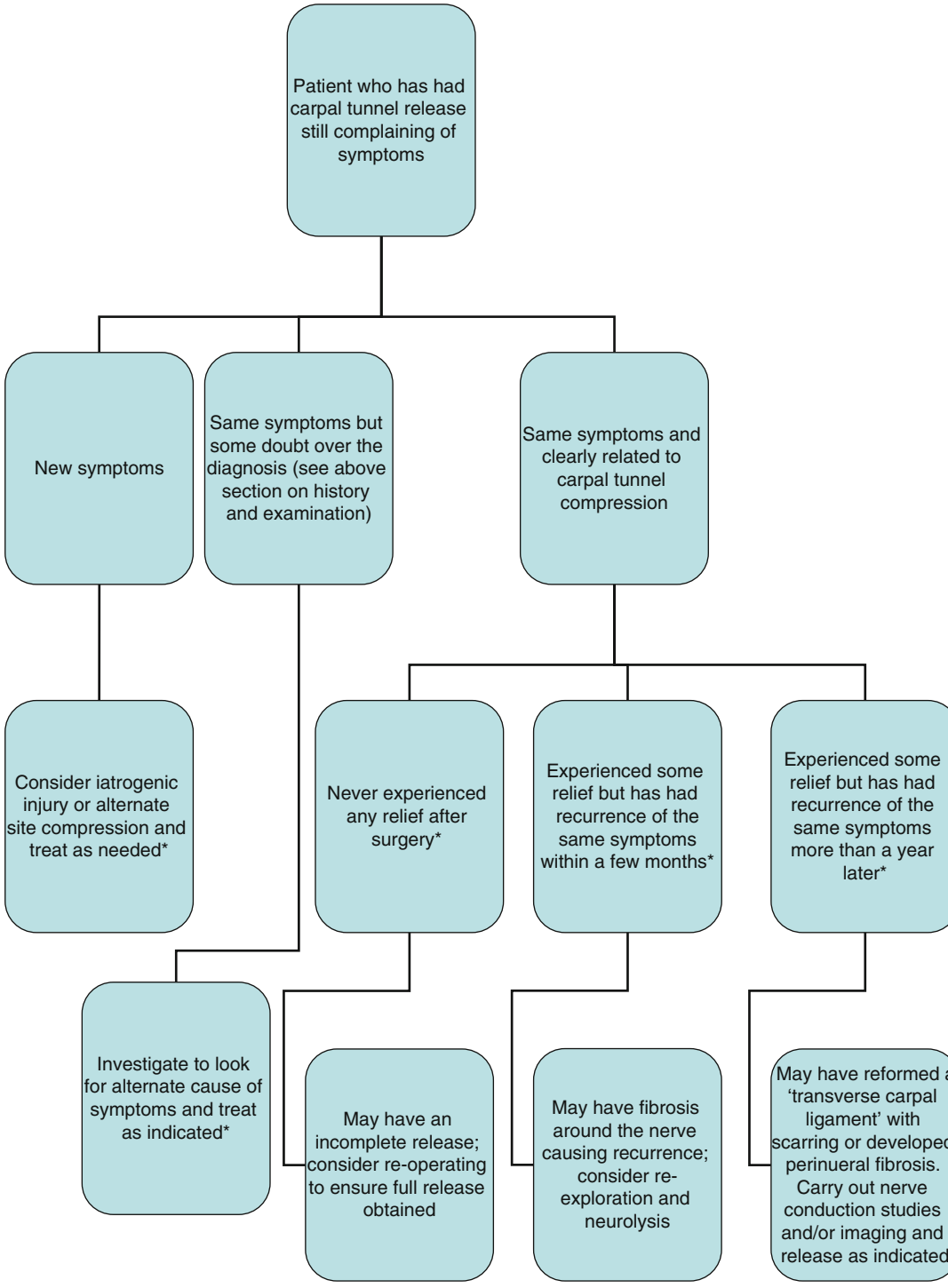

Fig. 12.5 Flow chart illustrating a suitable care pathway for failed CTR. *MRI may be helpful in differentiating these groups diagnostically [25]

Conclusions

Carpal tunnel syndrome is a common condition. In the vast majority of cases it is a straightforward clinical diagnosis, but expertise is required to exclude other pathologies in some cases.

A splint and/or steroid injection can be considered in mild cases of short duration.

Surgical release of the transverse carpal ligament is the only treatment with proven long term benefit. This can be done either endoscopically or open, depending on the patients requirements for return of function and the surgeon's expertise.

Significant complications of carpal tunnel release are rare but can be severe.

Recurrent or recalcitrant cases need careful evaluation and treatment by a hand specialist.

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Nerve Compression – Median Nerve Entrapment in the Forearm

13

B.J.R. Sluijter and M.J.P.F. Ritt

Keywords
Nerve Compression • Median Nerve Entrapment Forearm • Pronator Syndrome • Anterior Interosseous Nerve Syndrome

History

In 1848 Struthers described entrapment of the median nerve by the ligament bearing his name. But it was Seyffarth in 1951 who defined pronator syndrome as entrapment of the median nerve by the two heads of the pronator teres muscle or the fibrous arch of the flexor digitorum superficialis [1]. A few years later, in 1958, Kopell and Thompson proposed a surgical solution for this syndrome [2]. Since Seyffarth's description more anatomic structures causing median neuropathy have been described. Because, despite their etiologically differences, they produce the same pattern of sensory alterations and pain, all causes of median nerve compression in the forearm are termed pronator syndrome (PS). Although there is still controversy whether compression of the nerve plays a role and it thus can be regarded as a compression syndrome, isolated neuritis of the anterior interosseous nerve was described by Kiloh and Nevin in 1952 and is sometimes referred to as Kiloh-Nevin syndrome [3].

Few studies describe the incidence and prevalence of pronator and anterior interosseous syndrome in the general population. One study reported that in 238 patients presenting with median nerve neuropathy 84 % involved the carpal tunnel, less than 10 % had pronator syndrome and less than 2 % was diagnosed with anterior interosseous nerve syndrome (AINS) [4]. Another series of 902 patients operated on in a 5 year period for compression syndromes of the upper extremity described only ten cases of proximal median nerve entrapment, or approximately 1 % [5]. PS seems to present most commonly in the fifth decade. Women are more likely to be affected than men, especially when performing physically demanding jobs. No such observations have been made for AINS.

Anatomy

Plexus Brachialis

Innervation of the upper extremity takes place through the ventral rami of the spinal nerves C5 trough T1 which form the brachial plexus. The

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ventral rami become the superior, medius and inferior truncus. These in turn rearrange into the lateral (C5–7), medial (C8-T1) and posterior (C5-T1) cords. The cords become the distal nerves of the upper extremity. The median nerve (C6-T1) stems from the lateral and medial cords, with the lateral cord contributing mainly sensory fibres and the medial cord contributing mainly motor fibres.

Median Nerve

The median nerve lies anterior to the axillary artery. In the upper arm it crosses to the medial side of the brachial artery. It courses between the brachial muscle and the medial intermuscular septum towards the elbow, where it passes through the antecubital fossa and under the bicipital aponeurosis or lacertus fibrosus. The nerve then continues between the superficial and deep head of the pronator teres (PT) muscle. Proximal to the pronator teres muscle motor divisions to the palmaris longus, flexor carpi radialis (FCR), flexor digitorum superficialis (FDS) and variably to the flexor digitorum profundus (FDP) muscles branch off in an ulnar direction. As the median nerve passes through the arm the fibres of the anterior interosseus nerve (AIN) and the median nerve remain separate within the median nerve. When the median nerve passes deep to the PT, the AIN exits in a radial direction. Both the AIN and the median nerve pass deep to the fibrous arch of the flexor digitorum superficialis. After branching of, the AIN runs on the anterior surface of the interosseous membrane between the flexor pollicis longus (FPL) and FDP muscles, alongside the anterior interosseous artery. It passes through the pronator quadratus muscle and ends just distally. The AIN consists of motor and sensory fibres. It is responsible for sensation in the radiocarpal, intercarpal, carpometacarpal and radioulnar joints. It provides motor innervation to the FDP of the index and middle finger, the FPL and the pronator quadratus muscles. Five centimetres proximal to the wrist, the palmar cutaneous nerve branches from the main trunk of the median nerve. It runs ulnar to the FCR tendon in the direction of the thenar eminence, where it innervates the skin of the palm of the hand and the proximal thenar area. The median nerve continues through the carpal tunnel toward the palm of the hand. On the last part of its route it innervates the opponens pollicis, abductor pollicis brevis, flexor pollicis brevis and the first and second lumbrical muscles of the hand. It also gives off digital cutaneous branches that supply the radial three and a half digits on the palmar side and the index, middle and ring finger on the dorsum of the hand.

Examination

Determining whether a patient has PS or AINS starts with a detailed medical history. General health should be determined, including neurological status, past operations and recent trauma, and so on, to rule out other possible causes. Also, the onset and nature of the problem should be assessed.

A thorough physical examination should follow. The neck and shoulder are inspected for pain, muscle weakness and sensory deficits. The physician looks for signs of muscle atrophy. The range of motion is assessed for all joint is the upper extremity. The arm is palpated, to rule out pathological masses and to see if there is pain when applying pressure over muscles. If there is paresthesia in the median nerve distribution after 30 s or less of pressure on the pronator teres muscle, this is considered to be a positive pronator compression test. The extent and pattern of sensory symptoms in the arm and hand are noted. The Phalen, Tinel and Durkan test [6] are performed at the wrist level to rule out carpal tunnel syndrome (CTS). Muscle weakness of the FPL and the FDP of the index and long finger are examined. Ask the patient to make an ‘OK-sign’ by pinching the thumb and index finger together. Patients with complete AINS will exhibit hyperextension of the index finger distal to the interphalangeal joint and thumb interphalangeal joint due to weakness of the FPL and FDP 2.

Table 13.1 Characteristics of pronator syndrome and anterior interosseous syndrome

	Paresthesia	Muscle weakness	Direct compression	Tinel 's sign	EDX	MRI
PS	Thenar, palm, Lateral 3 digits	Not generally, may involve APB, OP, FPB, FPL, FDP 2/3, PQ, FCR	May be positive at PTM	May be positive at PTM	May help differentiate	Questionable
AINS	None	FPL, FDP 2/3, PQ	Negative	Negative	May help differentiate	May show muscle deterioration

PS pronator syndrome, *AINS* anterior interosseous syndrome, *APB* abductor pollicis brevis, *OP* oppones pollicis, *FPB* flexor pollicis brevis, *FPL* flexor pollicis longus, *FDP 2/3* flexor digitorum profundus index and middle finger, *PQ* pronator quadratus, *FCR* flexor carpi radialis, *PTM* pronator teres muscle, *EDX* electrodiagnostic examination

There are three specific tests to determine the site of the nerve entrapment in PS. The first is resisted pronation with the forearm in a neutral position. If this elicits pain the pronator teres muscle is likely to be the origin of the nerve entrapment. Next, the forearm is placed in maximal supination. The elbow is put in resisted flexion at 120–130°. If this is symptomatic the lacertus fibrosus is implicated. Lastly, if resisted flexion of the proximal interphalangeal joint of the long finger is painful, the FDS is most likely the source of the problem. Table 13.1 gives an overview of clinical and diagnostic options.

Pronator Syndrome

Presentation

Patients presenting with pronator syndrome will often complain of pain in the volar aspect of the forearm. This is most marked in people performing repetitive upper extremity movements. The pain or discomfort can usually be provoked by performing a specific movement. Sensory symptoms are present in the median nerve and palmar cutaneous branch distribution of the hand. These include numbness and paresthesias in the thumb, index finger, middle finger, radial side of the ring finger, thenar eminence and palm of the hand. Incomplete PS may be due to a reversed Martin-Gruber anastomosis, also called Marinacci communication [7]. Motor symptoms are not usually present in pronator syndrome, although there may be weakness of the thenar musculature. If the symptoms are present for a few months there may be a positive Tinel's sign over the compression site.

Electrodiagnostic conduction studies may not be conclusive in PS, and can therefore not be used to predict the outcome of therapy [8, 9]. Needle examination can reveal denervation in the pronator teres and more distal muscles. Decrease in motor and sensory conduction across the pronator teres muscle may also be found. It is advisable to conduct both studies. Although these tests may not be conclusive in PS, they can be used to rule out CTS and more proximal causes of muscle denervation, i.e. cervical radiculopathy or brachial plexopathy. Extensive literature exists on the use of ultrasound studies in CTS, but hardly any papers have addressed the use of ultrasound in PS specifically. Ultrasonography has proved to be a good addition to electrodiagnostic studies in CTS [10]. It can therefore be used to rule out CTS but not to confirm PS. It is questionable whether MRI can aid in diagnosing PS. If the neuropathy has been present for several months an increased signal may be seen in the muscles innervated by the median nerve on STIR or T2-weigted fat-suppressed images. Normal anatomical structures causing compression of the nerve will be difficult to appreciate on MRI. Therefore, in absence of an anomaly, i.e. pathological mass, MRI is not helpful in locating the origin of entrapment [11].

Differential Diagnosis

The differential diagnosis of pronator syndrome include, amongst others, carpal tunnel syndrome, Parsonage-Turner syndrome and thoracic outlet syndrome.

Although there is considerable overlap between PS and CTS, there are a few differences that help distinguish between the two syndromes. In PS both the median nerve and the palmar cutaneous branch of the median nerve are affected. This will result in dysaesthesia, not only in the thumb, index and middle finger, as in CTS, but also in the thenar eminence and palm of the hand. Careful assessment of the extent of sensory changes in the hand will help distinguish between the two. Furthermore, there should be no positive Phalen's test in patients with PS. There is, however, some indication that a percentage of patients with PS may have a positive Tinel's sign at the wrist [12]. CTS often presents with nocturnal symptoms, which are seldom present in PS. While usually not conclusive in PS, electrodiagnostic conduction tests may be used to confirm CTS. In a selection of patients PS and CTS will coincide. In these patients the physical examination of the arm and hand should raise suspicion. Electrodiagnostic studies, response to corticosteroid injections at the proximal compression site and at the carpal tunnel, and response to conservative treatment may help confirm the simultaneous existence of PS and CTS. If the diagnosis is confirmed, both sites of compression may be released in one operation [13].

Clinical Pearls – Differentiate PS from CTS

In PS there is:
Dysaesthesia over proximal palm and thenar area.
Negative Phalen's test.
Absence of nocturnal symptoms.
Positive provocative tests (see above).

Parsonage-Turner syndrome, also known as neuralgic amyotrophy or brachial plexus neuralgia is a peripheral nervous system disorder affecting the upper limb [14]. There are two forms of Parsonage-Turner syndrome; hereditary autosomal dominant and idiopathic. Both forms seem to result from a vulnerability to brachial plexus injury followed by an immune-mediated response to the injured plexus. It is characterised by a sudden onset of severe pain in the shoulder or arm. This is followed by muscle weakness and atrophy. The syndrome can vary greatly in presentation and nerve involvement. In severe cases it results in complete loss of function of the muscle. A thorough medical history with specific questions concerning the onset of the symptoms and a physical examination of the shoulder and arm should aid differentiation between PS and Parsonage-Turner syndrome [14].

Neurogenic thoracic outlet syndrome occurs when the brachial plexus is compressed at the thoracic outlet. The thoracic outlet is formed by the clavicle, scapula, first rib and the anterior and middle scalene muscles. Changes in any of these structures can lead to compression of the underlying plexus. Common causes include presence of a cervical rib, clavicular callus after a fracture, cervical whiplash, congenital fibromuscular bands or muscle hypertrophy from repetitive overhead activities. Although some symptoms may mimic PS, the cause of thoracic outlet syndrome lies more proximal and will thus result in associated proximal signs. The physical exam will often include neck and shoulder pain and reveal a pattern of paresthesia of the arm not related to the innervation pattern of the median nerve [15].

Cervical radiculopathy is a descriptive term used to define a disorder in which one or more cervical nerves cause pain, weakness, numbness or clumsiness due to problems at the root of the nerve near the spinal cord, the cervical nerve itself or both. The most common cause of cervical radiculopathy is compression of the root which can result from, amongst others, degenerate changes and decreased disc height. Less frequent causes include nucleus pulposus herniation, tumors and infections. Depending on the affected nerve or nerve root, cervical radiculopathy can mimic a number of distal nerve entrapments. The history and examination of the neck, shoulder and arm will help differentiate between cervical radiculopathy and PS or

AINS. Provocation test of the neck, i.e. moving the head to the affected side, the absence of reflexes and normal nerve-conduction studies all point to cervical radiculopathy [16].

Diabetes and alcohol induced neuropathy may in some cases imitate the sensory changes associated with CTS, but seldom AINS or PS. The medical history and concomitant neurologic changes in the legs and feet will usually be sufficient to tell them apart.

Anterior Interosseous Syndrome

Presentation

Unlike pronator syndrome, AINS seldom presents with sensory loss or paresthesia. Symptoms are related to the muscles innervated by the AIN, namely the flexor digitorum profundus of the index and middle finger, the flexor pollicis longus and the pronator quadratus muscles. Patients will generally complain about problems with fine motor skills such as writing, typing or pinching. Depending on whether the patients have a complete or incomplete AINS, function loss of one or more of these muscles will be present. In incomplete AINS there is usually an isolated weakness or function loss of the FPL or FDP. Variable innervation of the FDP to the middle finger by the median and ulnar nerve may cause incomplete AINS. Presence of distal interphalangeal joint flexion in the middle finger does therefore not necessarily exclude AINS.

Although conduction studies will not be useful in diagnosing AINS, needle examination can be used. The flexor pollicis longus, flexor digitorum profundus and pronator quadratus muscles may show varying degrees of denervation. Moreover, electrodiagnostic studies can help differentiate between AINS and CTS or flexor tendon ruptures [9, 17]. Ultrasound is usually not conclusive in AINS when there is no detectable cause, i.e. a tumor. This is partly due to the fact that the nerve itself is small and lies deep within the arm. In rare cases enlarged fascicles or loss of muscle tissue in established disease may be seen [18, 19]. When AINS is evaluated using MRI imaging, changes can be seen in the signal intensity of FPL, FDP and PQ muscles on STIR or axial T2-weigted fat-suppressed images. Also, if a space occupying lesion is causing AINS this will be seen on MRI. Normal anatomical structures leading to AINS are more difficult to perceive. However, a MRI made at the time of diagnosis can be used to monitor therapy related changes through additional imaging. Recovery should result in normalisation of the signal intensity whereas the development of fatty muscle atrophy signals deterioration of the neuropathy [11].

Differential Diagnosis

Brachial plexus neuritis or Parsonage-Turner syndrome can also mimic AINS. As with PS, a complete medical history and physical examination aids in differentiation between the two.

Incomplete anterior interosseous syndrome may be mimicked by rupture of the FPL, FDP 2 or both. The absence of sensory symptoms in particular can make differentiation problematic. Awareness of the differential diagnosis and scepticism of the diagnosis of tendon rupture in absence of an explanatory mechanism, i.e. rheumatoid arthritis or trauma, can avoid an unnecessary exploration of the FPL or FDP 2. When there is uncertainty about the aetiology, ultrasound or electrodiagnostic examination should be considered. Other disorders that can mimic AINS include flexor tendon adhesion, stenosing tenosynovitis and congenital absence of the FPL tendon.

Clinical Pearls – Differential Diagnosis in AINS

Parsonage-Turner syndrome: this is a neuritis and not a mechanical compression; these patients usually present with a history of severe pain for several weeks.

Rupture of FPL and/or FDP 2 e.g. in rheumatoid arthritis.

Stenosing tenosynovitis.

Congenital absence of the FPL.

Aetiology

In General

PS and AINS originate from pressure on the median and anterior interosseous nerve, respectively. The duration and severity of the pressure will determine the degree of damage to the nerve. If the pressure is present long enough or increases over time a number of changes occur in the nerve tissue. The first change to occur is the blood-nerve barrier becoming permeable. This leads to an increased influx of fluid. The fluid causes the connective tissues to distend, leading to increased pressure on the blood flow to the nerve, causing ischemia. If the pressure exists long enough it results in demyelination of the nerve and finally in axonal degeneration. The outer nerve fascicles are more susceptible to the pressure due to their location. This may explain differences in clinical presentation.

The Double Crush Theory

First described by Upton and McComas [20], the double crush theory purports that proximal nerve compression will render the nerve more susceptible to damage distally. This is said to be the result of axial flow restriction in the nerve fibres. Several animal studies exist aimed at providing proof for the double crush syndrome (DCS) theory. However, they primarily showed that two nerve lesions cause a more profound neurologic deficiency than one lesion [21, 22]. No animal study to date has proved that proximal nerve compression leaves the distal nerve prone to pathological changes or has made clear what causes the DCS. Also, clinical studies show a wide variety in the percentage of a second nerve palsy in patients with CTS [23, 24]. Those authors opposed to the diagnosis DCS point out that the percentage of patients with cervical radiculopathy who also have carpal tunnel syndrome seems to be similar to the incidence of CTS in the work force, and not elevated as suggested [25]. Furthermore, they stipulate that most of the cervical roots involved in the radiculopathy do not contribute to the median nerve [26]. These lesions are then shown to be two non-related neuropathies in one individual. This may suggest that shared aetiologic factors, i.e. degeneration, repetitive labour, etc., contribute to the high frequency of co-existence of cervical radiculopathy and distal neuropathy. It is advisable to be aware of the possibility of two nerve entrapment syndromes coexisting in patients, especially manual labourers.

Specific Aetiology of PS and AINS

There are a number of causes that can lead to median nerve compression in the forearm, apart from the anatomical restriction sites which are discussed later. These include space-occupying lesions, direct trauma (i.e. bone fracture, muscle trauma), indirect muscle trauma (i.e. repetitive movements causing myotendinous strain or muscle tears), Volkmann contractures and external pressure. Masses in the upper extremity causing neuropathy include lipomas, primary tumors (e.g. sarcomas, schwannomas) and metastatic disease [27]. Although rare, greenstick fractures, supracondylar fractures of the humerus [28] and elbow dislocations [29] mainly in children may lead to damage to, or pressure on the median nerve. Iatrogenic damage to the nerve, for example after minimally invasive elbow surgery [30], is fortunately rare. Direct or indirect trauma to muscle or bone may lead to scar tissue an callus that may entrap the median nerve [31]. Volkmann contractures develop as a result of ischaemic injury to the forearm muscles due to either brachial artery injury as with fractures of the humerus or external pressure on the artery or compartment syndrome of the upper limb. Fractures of the humerus, especially supracondylar fractures result in direct pressure on the artery. External pressure applied by a tourniquet or an ill-fitting cast may also lead to raised pressure. Compartment syndrome can have a number of causes, i.e. infection, hematoma, crush injuries or burn wounds. The first consequence of elevated pressure is decreased arterial blood flow leading to tissue ischemia. This is followed by edema of the soft tissue distal to the point of compression

Table 13.2 Possible anatomic locations of median nerve compression

	Pronator syndrome	Anterior interosseous nerve
Anatomical site of compression	Pronator teres muscle, deep and superficial head	Pronator teres muscle, deep head
	Flexor digitorum superficialis arch	Flexor digitorum superficialis
	Ligament of Struthers	Gantzer's muscle (accessory head of the flexor pollicis longus)
	Lacertus fibrosus	Tendinous origin of the palmaris profundus
	Accessory bicipital aponeurosis	Accessory lacertus fibrosus
	Snapping brachialis tendon	Blood vessels Aberrant radial artery branch, Inconsistent median artery, Anterior interosseous arteries crossing the nerve

Lee and LaStayo [32]

resulting in compromised venous and lymphatic drainage. If the source of the compression cannot be removed or the compartment is not released this vicious circle leads to muscle and nerve ischemia that becomes irreversible and will ultimately result in tissue breakdown.

Sites where the nerve passes restricted anatomical structures are vulnerable to increased pressure due to the limited space. Table 13.2 lists anatomic sites where the nerve can be compressed in both PS and AINS. A frequent cause of median nerve compression is the existence of fibrous bands in the arm. At the elbow, in patients with a residual supracondylar process, the nerve can be compressed under the ligament of Struthers. This is a fibrous band that runs between the supracondylar process and the medial epicondyle. In the general population 0.7–2.7 % has a vestigial supracondylar process. Other causes of nerve compression at the elbow include a snapping brachialis tendon or an accessory bicipital aponeurosis. More distally, fibrous bands between the deep and superficial heads of the pronator teres muscle, a tendinous deep head of the pronator teres muscle and the proximal arch of the flexor digitorum superficialis muscle can restrict the nerve. The normal bicipital aponeurosis or lacertus fibrosus is another possible point of restriction. In AINS an accessory head of the flexor pollicis longus also known as Gantzer's muscle or an aberrant vessel, such as an infrequent branch of the radial artery, may be the cause of nerve compression.

Treatment

No randomised controlled trials have assessed the therapeutic options in PS and AINS. Therapy is ideally tailored to the patients symptoms after discussing the options with the patient. Treatment includes conservative and operative options.

Non-operative Management

In the acute phase of PS and AINS non-operative management is advocated. This includes resting the affected limb, immobilisation and avoidance of aggravating activities. If the symptoms are job related the patient is advised to temporarily perform alternative work or avoid actions that elicit pain. If resting is difficult or the symptoms are severe a splint or sometimes even a cast can be considered. A posterior elbow splint in 90° flexion and the forearm in mid rotation is advised for approximately 2 weeks in PS [32]. For an additional 2–4 weeks aggravating activities are avoided but normal use of the affected upper extremity is slowly regained.

There are several non-operative modalities described in the treatment of PS and AINS. These include ultrasound, electrical stimulation, iontophoresis and nerve gliding/mobilisation exercises [32]. However, there is insufficient evidence for the routine use of any of these modalities.

If rest and immobilisation do not alleviate the symptoms non-steroidal anti-inflammatory drugs and local steroid injections [12] should be considered. Although the physician should be aware that evidence of the effect of steroids is based mainly on case reports. If symptoms persist despite conservative therapy for several weeks to months, surgery should be discussed with the patient.

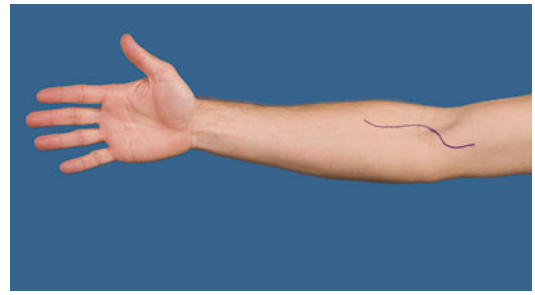

Fig. 13.1 Modified proximal Henry's approach

Operative Technique

The operation should be tailored to match preoperative clinical findings. If physical examination reveals pain with stress on a particular tendon or muscle unit or with direct palpation, this compression point should be explored. There are a number of possible incisions described for this operation. These include extensive exposure (modified Henry's approach), lazy S-incision in the proximal volar forearm and one or two longitudinal, oblique or transverse incisions. We advocate the use of a wide exposure in cases of PS and AINS (Fig. 13.1). The incision starts at the antecubital fossa and extends for approximately 10 cm distally (either straight longitudinal or lazy-S). Keeping in mind the preoperative findings the surgeon should select an incision that allows examination of all possible compression sites. The use of a tourniquet is standard practise. After incision the cutaneous sensory branches are identified and preserved. For PS patients the lacertus fibrosus or bicipital aponeurosis is identified first and incised. The exploration is continued distally, in the direction of the pronator teres muscle. The tendon of the superficial head of the muscle is lengthened using a step-lengthening tenotomy. This allows for a better exposure of the median nerve as it passes between the two heads of the muscle. In AINS there may be fibrous bands from the pronator teres muscle that cause the compression. These should be released. The median nerve is then followed in the direction of the arch of the flexor digitorum superficialis muscle. The tendinous part of the arch is divided to release the median nerve. If there is reason to believe that the ligament of Struthers is the cause of the neuropathy, the incision can be extended proximally. Careful haemostasis is imperative in order to prevent postoperative pressure on the nerve due to a haematoma. Bupivacaine may be left in the operation site for postoperative pain management. Two days after surgery the dressing may be removed. At this stage the patient is instructed in careful exercise of the limb. Normal activities and work can be started 6–8 weeks post-operatively.

Clinical Pearls – Tricks, Tips and Caveats in Decompression of the Median Nerve (PS and AINS)

Use a wide incision starting just above the elbow crease. After the release of the bicipital aponeurosis, run a finger along the median nerve and if a ligament of Struthers is suspected, the incision is extended more proximally (Fig. 13.1).

Be careful not to damage the medial antebrachial cutaneous nerve, which can cause painful neuromas postoperatively.

The tendon of the superficial head of the pronator teres is located just lateral to the radial vessels in the distal part of the exposure at the forearm.

Beware of inadequate decompression of the median nerve: release the superficial head of the pronator teres (step cut incision), the deep head of the PT, as well as the superficial tendinous arch of the FDS.

Release the tourniquet before wound closure and perform meticulous haemostasis.

Outcome

Pronator Syndrome

Correct treatment of PS begins with an accurate diagnosis. As stipulated before, this may not always be easy. There are even those who question the existence of PS as an identifiable entity [33].

Reports on treatment of PS are few and mostly consist of uncontrolled retrospective case studies (level IV evidence). The original report by Seyffarth described 17 patients treated with novocaine injections once or twice a week and massage of the affected limb. He reported that nine patients improved greatly, six were subjectively free from symptoms and two improved somewhat [1]. In another study of seven patients with PS, all patients were treated with corticosteroid injections and advice concerning the use of the affected limb at work. They reported adequate relief of symptoms in five out of seven patients. It was recommend that patients with persistent or recurrence of symptoms should be considered for operation [12]. The largest series of patients describes 39 patients with PS [8]. Thirty-six arms were operated on in 32 patients, with good to excellent results in 28 arms (78 %), fair results in five (14 %) and no alleviation in three (8 %) patients. Only two of the patients in the conservative group improved, four were unchanged and one deteriorated. It must be said that the symptoms of the patients were present for an average of 23 months (range 1–120 months).

It is not clear how long conservative treatment or corticosteroid injections should be tried before resorting to surgery [5]. The suggestions made in the literature do not seem to be supported by clinical studies. Based on the severity and duration of symptoms and the patients characteristics, the physician and patient should decide on the course of action after discussing the therapeutic options. It seems prudent to start with conservative treatment, with or without corticosteroid injections if the symptoms have not been present for longer than 1 or 2 years. If this period has passed, the symptoms worsen or alleviation is not achieved after a reasonable period, surgery should be considered.

Anterior Interosseous Syndrome

There is currently no comprehensive data on the aetiology and natural history of AINS. Although there is still controversy, AINS is regarded by many as a transient neuritis in most cases, although compression of the nerve can play a role. The physician treating patients with AINS should be aware of the fact that most cases of AINS probably never present clinically at all. Patients that are seen in the medical office represent the more problematic cases [34]. Often, in literature, palsy of the anterior interosseous nerve with different aetiological backgrounds are examined together and this affects the outcome of trials researching therapeutic options for AINS [35, 36]. Moreover, the level of evidence in AINS is mostly level IV, since most reports consist of uncontrolled retrospective case studies.

When reviewing the literature on therapeutic options for AINS a few observations can be made. The first is that at least part of the patient population recovers spontaneously [37, 38]. One report on 21 AINS patients treated with vitamin B12 and electrical stimulation, reported substantial recovery for all patients. They did point out that patients under 40 years of age showed signs of recovery within 12 months, whereas older patients started to recover after an average of 15 months [39]. This leads to the second observation that it is advisable to wait at least several months before attempting surgical intervention. It is still up for debate how long that period exactly should be. Suggestions range from 6 weeks [40] to 1 year [41]. On the other hand, there is some evidence suggesting that AIN palsy evident for more than 2 years is unlikely to recover [42]. Several authors concluded that it is sensible to wait 8–12 months before considering surgery [34, 41, 43]. During this time the patient is seen at regular intervals for clinical and, if necessary, additional examination. This approach would, however, require that causes treatable by operation, i.e. tumors, traumatic lesions and so on, or a more proximal origin of the symptoms should be ruled out before the observational period is commenced. Also, if progressive worsening of the symptoms is found during conservative treatment, surgery should be considered more readily.

When considering surgery, it should be kept in mind that surgical intervention does not necessarily shorten the time to recovery [38, 44], nor does it always lead to a full recovery. Moreover, during surgery it is not always possible to find an identifiable area of compression [37, 45]. This could be due to an incorrect diagnosis and possibly account for prolonged recovery time postoperatively. Other considerations include patient age, gender, whether the symptoms are work related and whether there is a complete or incomplete AINS.

All these considerations should be discussed with the patient prior to any surgical intervention. Preferably, nerve decompression should be reserved for those cases where there is a defined area of compression.

Complications

Complications of surgical nerve decompression include persistent symptoms due to misdiagnosis, cutaneous nerve branch injury causing dysesthesia and injury to the motor branches of the median or interosseous nerve resulting in muscle weakness or failure. The operation may cause extensive scarring to the muscles or nerves of the forearm, causing muscle problems or neuropathy.

Summary

In the elbow region and forearm, the median nerve can be compressed at the bicipital aponeurosis, the ligament of Struthers (connects medial epicondyle with a supracondylar process of the humerus), fibrous bands between the deep and superficial origin of the pronator teres, the arched origin of the FDS.

Symptoms and signs of PS or AINS include: tingling or dysaesthesia in the median nerve distribution (no nocturnal preference), aching of the forearm especially with rotation or grip, weakness of FPL and FDP 2 (failure to make the OK-sign), positive Tinel's sign at the site of proximal compression of the median nerve (negative over the carpal tunnel), resisted contraction of FDS, PT or biceps may evoke symptoms.

Investigations: MRI may show a space occupying lesion or muscle oedema, nerve conduction studies may be inconclusive in PS and AINS but can help to rule out CTS, insertional EMG can however be of use revealing denervation of the pronator teres and more distal muscles, ultrasound cannot confirm PS or AINS, but can rule out CTS.

Differential diagnosis of PS and AINS include: CTS, Parsonage-Turner syndrome (neuritis), rupture of FPL and/or FDP 2 e.g. in rheumatoid arthritis, stenosing tenosynovitis, congenital absence of the FPL, thoracic outlet syndrome, cervical radiculopathy.

Treatment: in general non-operative, observation for several months, avoid provocative manoeuvres. Exploration of the antecubital fossa and decompression of the median nerve is warranted after failed conservative treatment of several months or when a space-occupying lesion or other mechanical causes for compression are suspected.

Surgical treatment: Beware of inadequate decompression of the median nerve and release the superficial head of the pronator teres (step cut incision), the deep head of the PT, as well as the superficial tendinous arch of the FDS. If a ligament of Struthers is suspected, extend the incision more proximally.

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Nerve Compression: Ulnar Nerve of the Elbow

14

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Keywords

Ulnar nerve • Cubital tunnel • Compression neuropathy at elbow • Weakness in hand • Clumsiness in hand • Numbness or tingling in ring and small fingers • Elbow anatomy • Elbow examination • Treatment • Complications • Revision surgery

Introduction

Ulnar nerve compression neuropathy was first described by Panas in 1878 [1]. Feindel and Stratford initially described the significance of the 'cubital tunnel' and coined the term in 1958 [2]. Entrapment of the ulnar nerve is regarded as one of the most common compression neuropathies of the upper extremity, second only to carpal tunnel syndrome [2–4]. Although the nerve can be compressed at any location along its course, the most common location is at the elbow; and the most common site is at the cubital tunnel [5]. Diagnosis is made from a combination of history, examination findings, and provocative tests. Some authors still advocate the use of electrodiagnostic studies to confirm findings and localize the compression. Still, no standard exists for the surgical treatment of cubital tunnel that is refractory to conservative measures [6]. The available evidence at this point is insufficient to identify the best treatment technique [7]. Understanding the anatomy and pathology are critical steps towards successfully diagnosing and managing ulnar nerve compression regardless of the technique used.

Anatomy

The ulnar nerve is the terminal branch of the medial cord of the brachial plexus . It contains fibers derived from the ventral rami of C8 and T1 with occasional contribution from C7 (5–10 % of patients have a flexor carpi ulnaris motor branch from C7). The ulnar nerve enters the arm medial to the axillary artery and courses along the medial head of triceps and brachialis muscle eventually lying posteromedial to the brachial artery. It traverses the medial intermuscular septum posteriorly and passes through the Arcade of Struthers, a thickened fascia between the medial head of

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triceps and intermuscular septum located approximately 8 cm proximal to the medial epicondyle. The nerve enters the ulnar sulcus 3.5 cm proximal to the medial epicondyle.

At the elbow, the ulnar nerve continues posterior to the medial epicondyle and enters the cubital tunnel proper. The retrocondylar groove is the most common site of compression according to intraoperative electrical studies [8]. The roof of the cubital tunnel consists of a fibrous aponeurosis that thickens to form the cubital tunnel retinaculum also known as the 'arcuate ligament' or 'Osborne's Ligament [9].' Khoo et al. described the retinaculum as 4 mm wide and extending from the medial epicondyle to the olecranon [10]. The fibers are oriented in a transverse fashion and tension is dependent on elbow position (fibers are tightest in flexion). The retinaculum serves as the proximal-most roof of the tunnel and prevents anterior subluxation of the nerve with elbow flexion. The deep layer of the aponeurosis of the two heads of the flexor carpi ulnaris (FCU) muscle, also termed 'Osborne's fascia,' forms the distal roof of the tunnel. The floor of the cubital tunnel consists of the elbow capsule and the posterior and transverse components of the medial collateral ligament. The walls of the tunnel are the medial epicondyle and olecranon respectively.

Just before entering the cubital tunnel, the ulnar nerve gives off its first branch, which is thought by some to provide articular proprioception [11]. However, anatomic studies by Jabaley et al. and Watchmaker et al. have contested that articular branches, if present, are rarely found [12, 13]. Within the cubital tunnel, the ulnar nerve gives off multiple motor branches to the FCU and the ulnar half of the flexor digitorum profundus (FDP). An average of 3.4 motor branches to the FCU has been documented, with the majority branching on the ulnar side of the nerve [14].

After exiting the cubital tunnel, the ulnar nerve passes between the humeral and ulnar heads of the FCU. The ulnar nerve pierces the flexor pronator aponeurosis 3 cm distal to the cubital tunnel and travels deep to the FCU on the surface of the FDP. The flexor pronator aponeurosis may compress the nerve up to 5 cm distal to the medial epicondyle [15]. The branches of the ulnar nerve continue into the hand through Guyon's canal. Terminal branches of the nerve provide motor innervation to the hypothenar muscles, all the interossei, third and fourth lumbricals, adductor pollicis, medial head of the flexor pollicis brevis, and articular branches to the adjacent carpal joints. Terminal branches provide sensory innervation to the ulnar aspect of the palm, dorsal ulnar hand, small finger, and half of the ring finger.

Ulnar Nerve Blood Supply

The blood supply to the ulnar nerve was initially described in work by Sunderland [16]. Three main extrinsic arteries have been identified: the superior ulnar collateral artery, inferior ulnar collateral artery and posterior ulnar recurrent artery. Yamaguchi et al. demonstrated a consistent but segmental extraneural and intraneural vascular supply from these vessels [17]. Some authors, however, have questioned the clinical significance of the extrinsic blood supply to the ulnar nerve [18, 19]. Accurate characterization has implications for safe transposition. Lundborg and Sunderland showed that the integrity of the intrinsic microcirculation of peripheral nerves is critical for oxygen supply to individual nerve fascicles [20, 21]. Preservation of the proximal and distal intrinsic blood supply may allow its safe transposition but the clinical evidence awaits future study [22].

Medial Antebrachial Cutaneous Nerve Anatomy

Clear understanding of the position and anatomy of the medial antebrachial cutaneous nerve (MABCN) is crucial to avoid injury during surgery . Leffert was the first to demonstrate the importance of avoiding injury to MABCN during ulnar nerve decompression [23]. The MABCN is a direct branch of the medial cord (C8-T1) and descends in the arm anterior and medial to the brachial artery; it emerges from under the brachial

fascia adjacent to the medial epicondyle and olecranon to innervate the skin over the anterior and medial surface of the forearm [24]. Dellon and MacKinnon reported that a common cause of medial elbow pain following cubital tunnel surgery was injury to posterior branch of the MABCN. In their study, 23 of 25 patients with persistent symptoms following surgery for ulnar nerve decompression had evidence of injury to the cutaneous nerve [25]. Sarris et al. found similar findings in their cohort of 20 patients with recurrent symptoms following surgery [26]. Lowe evaluated 97 patients undergoing primary surgery for cubital tunnel to identify the proximity of MABCN branches. The authors found that branches lie on average 1.8 cm proximal to the medial epicondyle and 3.1 cm distal to the medial epicondyle [27]. They emphasized that knowledge of the anatomy can prevent iatrogenic injury to the MABCN and neuroma formation.

Avoiding injury to the MABCN can definitely improve the chance for clinical success in treatment regardless of surgical technique chosen [27, 28]. However, if injury is identified, persistent neuromas should be resected, electrocauterized and transposed deep into muscle [29–31]. An alternative treatment is to suture the nerve stump, end to side, into a functioning nerve. This may permit the nerve sprouts to incorporate into an intact nerve as opposed to forming a recurrent neuroma [32].

Clinical Pearl

Always look for, and preserve, branches of the Medial Antebrachial Cutaneous Nerve following skin incision

Sites of Potential Compression

The areas of compression in cubital tunnel syndrome have been well described in the literature. Sites of compression lie 10 cm proximal to and 5 cm distal to the elbow so it is imperative that the clinician understands the anatomy in this region. There are five potential sites of compression, including the Arcade of Struthers, the intermuscular septum, the area of the medial epicondyle, Osborne's ligament and Osborne's fascia.

The Arcade of Struthers and the intermuscular septum only appear to cause nerve compression following anterior transposition or with a nerve that subluxates anteriorly over the medial epicondyle during elbow flexion [33]. The septum and arcade must be released in these circumstances. The ulnar nerve can be compressed along the length of the Arcade which averages 5.7 cm. Hypertrophy of the medial head of the triceps and snapping of the triceps head are two more well-reported causes of compression in this region [33, 34].

The area of the medial epicondyle and epicondylar groove is thought by some authors to be the most common site of compression [8]. The nerve may be compressed by bone spurs in an individual with osteoarthritis [35]. Compression from a post-traumatic valgus deformity at the elbow can be another cause of cubital tunnel syndrome (Fig. 14.1).

Space occupying lesions such as ganglia, soft tissue tumors and hypertrophic synovium have the potential of impinging the nerve in this location [36, 37] (Fig. 14.2). The anconeus epitrochlearis is an anomalous muscle found outside the groove that is present in 3–28 % of cadavers [31, 38]. When present, the muscle is divided in conjunction with ulnar nerve decompression [39] (Fig. 14.3). Habitual subluxation or dislocation of the nerve from the groove makes the nerve more susceptible to injury as the nerve can become inflamed from repetitive friction over the medial epicondyle. Childress found a 16 % incidence of subluxation in his cohort of asymptomatic individuals. Patients with nerves that subluxed to the tip of the medial epicondyle were termed 'Type A,' while those with a nerve that subluxed beyond the medial epicondyle were 'Type B' [40]. The authors surmised that although asymptomatic, the position and hypermobility make the nerve vulnerable to harm.

The cubital tunnel proper was initially described by Osborne [9]. Its limited dimensions and volume make it a common cause of compression. The dynamic changes that occur

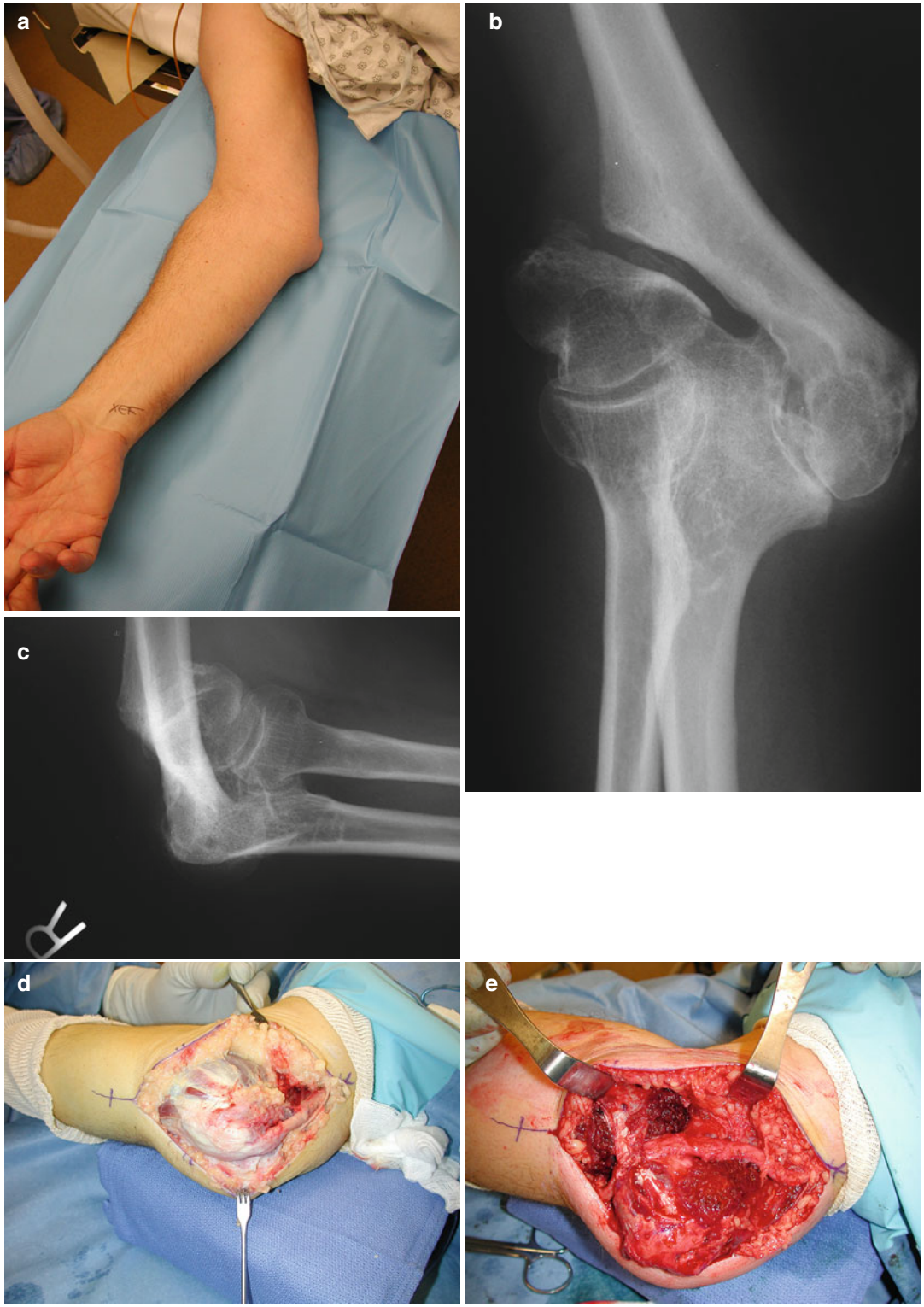


Fig. 14.1 Picture of a patient with a cubitus valgus deformity (**a**). AP (**b**) and lateral (**c**) radiographic images show a persistent traumatic valgus non-union. Intraoperative images before (**d**) and after ulnar nerve decompression and anterior transposition (**e**)

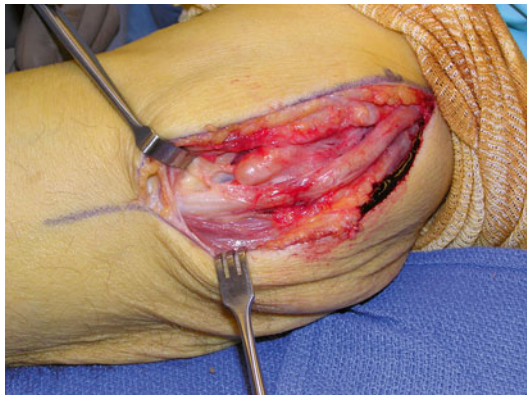


Fig. 14.2 Cyst located within the cubital tunnel is one example of a space occupying lesions (ganglia, osteochondritis, soft tissue tumors, hypertrophic synovium) that has the potential of impinging the ulnar nerve within the cubital tunnel

during motion can mechanically stretch the nerve and alter pressures within the tunnel, leading to ischemia and irritation of the ulnar nerve. Appelberg et al. described the dynamic anatomy of 15 cadaveric elbows in flexion and extension [4]. Flexion pushed the ulnar nerve anterior and medial secondary to the bulge of the medial head of the triceps during this motion. The nerve immediately adjacent to the epicondyle was found to stretch and elongate 4.7 mm during flexion. The roof of the tunnel plays a vital role in entrapment in the cubital tunnel proper. Thickening of Osborne's ligament and Osborne's fascia can cause direct nerve entrapment and are another critical structure to be released during surgical decompression.

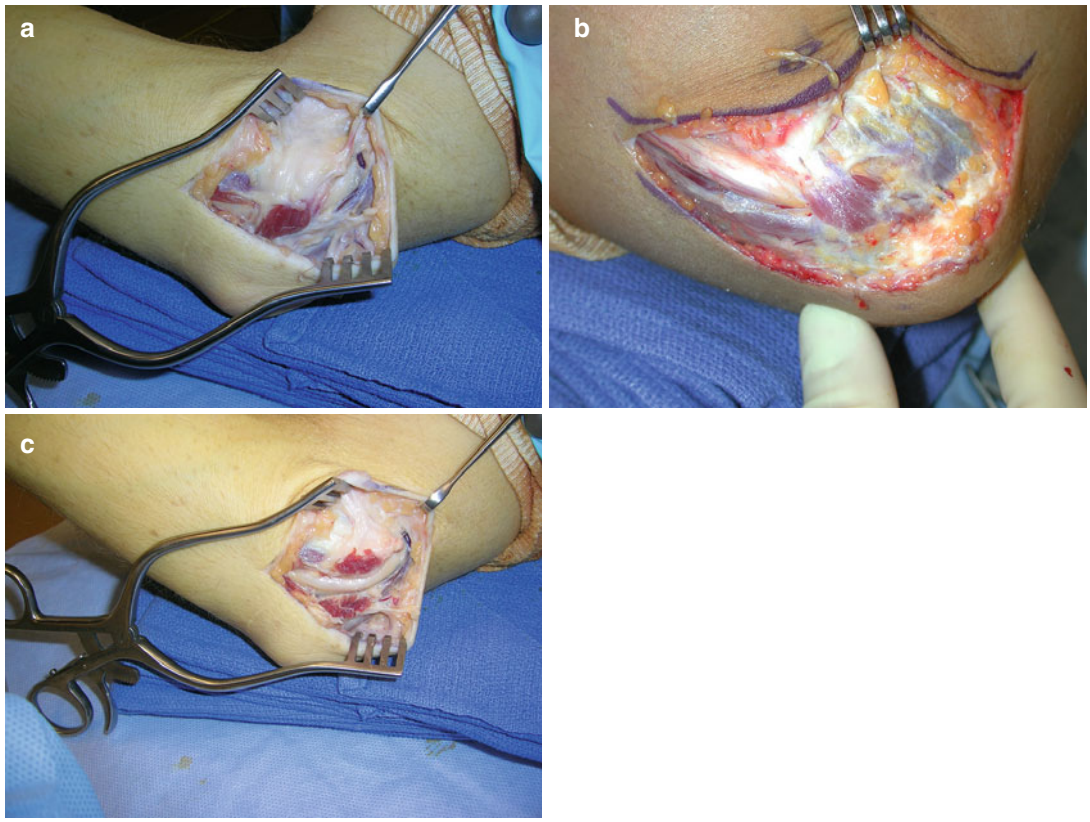


Fig. 14.3 The anconeus epitrochlearis is an anomalous muscle that is present in 3–28 % of cadavers. The clinician must be familiar with its appearance and aware of its compressive potential. (**a**, **b**) The anconeus epitrochlearis arises from the medial border of the olecranon and triceps and inserts onto the medial epicondyle. (**c**) Ulnar nerve following complete release of the anomalous muscle

After exiting the cubital tunnel, the ulnar nerve passes between the humeral and ulnar heads of the FCU. The flexor pronator aponeurosis may compress the nerve up to 5 cm distal to the medial epicondyle as mentioned above [15]. This area must be incised at the time of ulnar nerve decompression to ensure that the nerve is free at its most distal extent in cubital tunnel syndrome.

Karatsa et al. noted that the regional anatomic structures of the elbow show variability in the number and location of fibrous bands that have the potential to compress the ulnar nerve [41]. It is essential that during surgical release the nerve be released at all sites of compression from its proximal to distal end.

Clinical Pearl: Potential Sites of Nerve Compression

Arcade of Struthers between medial intermuscular septum and medial head of triceps

Cubital tunnel itself – Osborne's ligament

Osborne's fascia between heads of FCU

Pathology

The ulnar nerve can be subjected to traction and compression within the confines of the cubital tunnel with dynamic motion. Mechanical irritation and ischemia are thought to be factors associated with this disease process [4, 36, 42].

With elbow motion, associated changes occur to the shape and space of the tunnel. Bulging of the medial collateral ligament, tightening of the arcuate ligament or Osborne's ligament and firing of the FCU and medial head of the triceps are just a few of the changes observed. MRI studies showed that the tunnel is circular in shape and most spacious in extension. With flexion, the tunnel adopts a wider and flatter configuration. The tunnel becomes triangular or ellipsoid in flexion with a measurable height decrease of 2.5 mm. The cubital tunnel has been shown to narrow by 39–55 % with elbow flexion which places the nerve at increased risk for ischemia [4, 43].

Pechan and Julius found that the ulnar nerve is on maximal stretch with the shoulder abducted, elbow flexed and wrist extended [42]. In this setting, the ulnar nerve was observed to elongate 4.7 mm [4]. The authors found that the intraneural pressure of the cubital tunnel increases 600 % in this position. Wierich and Gelberman used MR and ultrasonographic imaging to measure the intraneural and extraneural pressure of the cubital tunnel in twenty cadaveric arms and found that intraneural pressure was significantly higher than extraneural pressure with the elbow flexed greater than 90° [43]. The tunnel pressure is on average 9 mmHg in extension. In flexion, the pressure increases to 63 mmHg; representing a seven-fold increase in pressure on the ulnar nerve.

Clinical Pearl

Ulnar nerve intraneuaral pressure increases sevenfold in elbow flexion

Perfusion studies in animals have shown that blood flow and axonal transport are affected by compression and stretch. Nerve damage is related to strain; lengthening of only 8 % has been shown to decrease neural blood flow in the sciatic nerve of rats and rabbits [44, 45].

There has been no scientific data to support particular vocational or avocational activities as causal risk factors for developing cubital tunnel syndrome. There are specific occupations that have been speculated to be associated with the diagnosis. Painters, carpenters, musicians, basketball players and tennis players all perform activities that involve repetitive elbow flexion. Other suggested risk factors include prior trauma, fracture, habitual subluxation and systemic disease [46].

Diagnosis

Diagnosis is typically based on history and examination. Sunderland studied the intraneural topography of the ulnar nerve as it courses within

the elbow and forearm [21]. He observed that the motor fibers that supply the intrinsics have a more superficial course compared with those that supply the FCU and FDP. He believed that this explained why intrinsic weakness of the hand is a common finding on presentation. Similarly, the sensory fibers of the ulnar nerve have a superficial location as the nerve traverses the elbow potentially explaining why paresthesias in the small and ring finger are another common finding.

The earliest sign of cubital tunnel syndrome is typically numbness and tingling of the ring and small finger. Some patients have difficulty localizing their symptoms. Patients may complain of medial elbow pain with radiation into the forearm [47]. Subjective motor loss, usually described as grip weakness, is a frequent complaint; this is especially true when torque is applied to a tool. Additionally, pinch weakness and difficulty with grasp can be involved. These complaints are usually related to intrinsic weakness. Early on, the same patients may exhibit clumsiness and difficulty with fine motor coordination. Sensory loss over the ulnar dorsal portion of hand helps differentiate between cubital tunnel syndrome versus compression of the ulnar nerve at Guyon's canal. The onset of symptoms and whether they are intermittent or constant can provide information about the chronicity and severity of disease. It can be helpful to determine if there is symptomatic worsening with the elbow in a flexed position, if the patient experiences night pain, and what relieves their symptoms.

Clinical Pearl

Sensory loss over the ulnar dorsal portion of hand helps differentiate between cubital tunnel syndrome versus compression of the ulnar nerve at Guyon's canal

Forced elbow flexion may increase symptoms with cubital tunnel compression

The presentation of cubital tunnel is sometimes indistinguishable from other disease processes. A careful history of comorbidities should be performed for thyroid disease, diabetes mellitus, haemophilia, acromegaly (Fig. 14.4), and peripheral neuropathy. One must have a high index of suspicion to rule out cervical radiculopathy, thoracic outlet, and compression at guyon's canal which can each be easily confused with cubital tunnel syndrome. Cervical root compression can present with neck pain, worsened with neck extension and ipsilateral rotation and improved with shoulder abduction. Patients with thoracic outlet syndrome complain of a vague shoulder ache with numbness along the medial forearm. Symptoms tend to worsen with overhead activities. Ulnar tunnel syndrome presents with wrist pain and, when associated with ulnar artery thrombosis, digital ischemia. Patients with ulnar artery thrombosis and ulnar nerve compression at the wrist have a history of repetitive wrist trauma and a positive Allen's test. A key distinguishing feature in patients with ulnar nerve compression at the wrist is the lack of dorsal hand numbness.

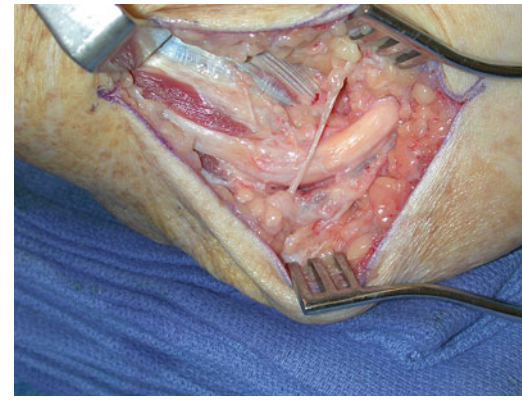


Fig. 14.4 Intra-operative image of an acromegalic ulnar nerve. Note the markedly enlarged ulnar nerve. Peripheral nerve enlargement seems to be an intrinisic part of the disease

The Double Crush phenomenon was initially described by Upton and McComas in 1973 [48]. It was hypothesized that impairment of axoplasmic flow at more than one site along a nerve can cause neuropathy. Moreover, compression at one site renders other sites more sensitive to compression. It is certainly possible that multiple sites within the cubital tunnel can be involved or

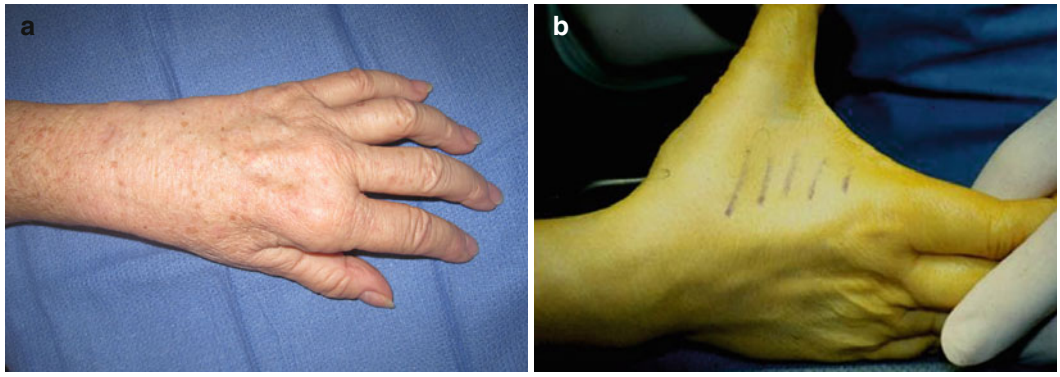


Fig. 14.5 Atrophy in patients with long-standing cubital tunnel is most evident at the dorsal first web space (**a**, **b**). This 'sunken in' region represents muscle loss of the first interosseous muscle

that local compression can be superimposed on proximal disease (cervical compression, thoracic outlet, etc.). A high index of suspicion is necessary to rule out other causes of neuropathy and physical examination should evaluate all potential sites of compression.

Sensory hypoesthesia within the ulnar nerve distribution can be objectively evaluated by Semmes-Weinstein or two-point discrimination tests. Motor symptoms present as either intrinisic or extrinsic weakness. Atrophy and clinical weakness is usually not seen for months to years following onset and subsequently can be an indicator of chronicity of symptoms. Atrophy is most evident at the dorsal first web space, representing loss of muscle volume in the 1st interossei muscle (Fig. 14.5). Mallette et al., however, noted that atrophy on presentation was four times more common in cubital tunnel than carpal tunnel [49]. As compression advances, loss of thumb adduction can present as Froment's sign, compensatory flexion at the IP joint of the thumb to aid in pinch against the index finger. Froment's sign confirms weakness of the adductor pollicis. Loss of adduction of the index and middle finger can be elicited with the 'crossed fingers test' representing weakness of the first volar and second dorsal interosseous muscles (Fig. 14.6). Wartenberg's sign reveals an inability to adduct the small finger, presenting as an ulnar deviated digit (Fig. 14.7). Wartenberg's confirms weakness of the third palmar interossei. A weak grip may be the result of denervation of the ulnar nerve innervated FDP to the small finger and ring finger. Advanced signs

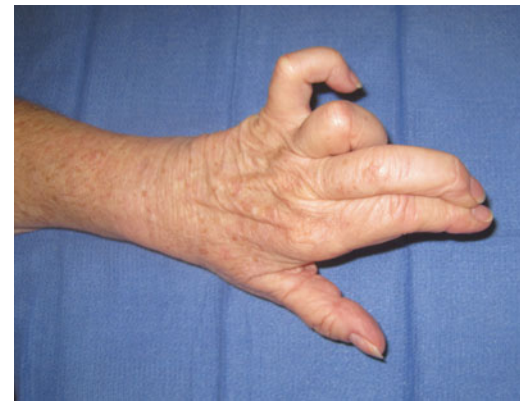


Fig. 14.6 Demonstration of loss of adduction of the index and middle finger secondary to ulnar nerve neuropathy. A positive 'crossed fingers test' represents weakness of the first volar and second dorsal interosseous muscles. There is also apparent 'clawing' of the ring and small finger

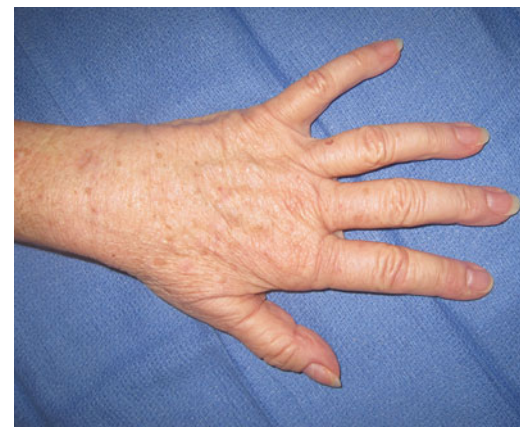


Fig. 14.7 Wartenberg's sign reveals an inability to adduct the small finger, presenting as an ulnar deviated digit. Wartenberg's sign confirms weakness of the third palmar interossei

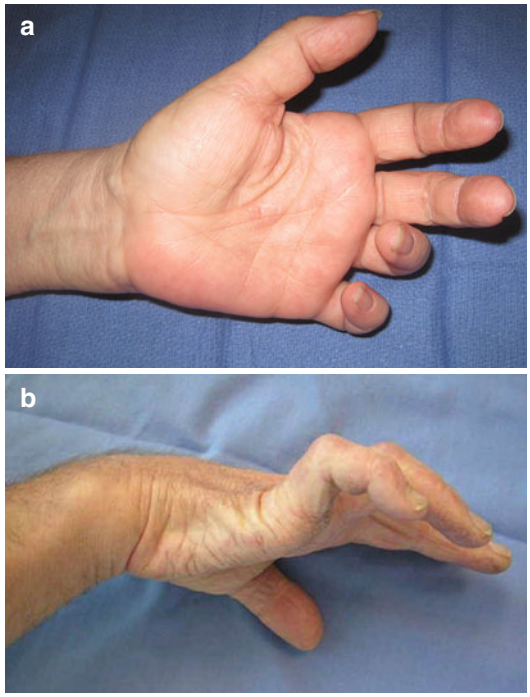


Fig. 14.8 Intrinsic minus posture, or ulnar clawing, of the ring and small finger secondary to weakness of the intrinsics (third and fourth lumbricals and interossei) (**a**, **b**)

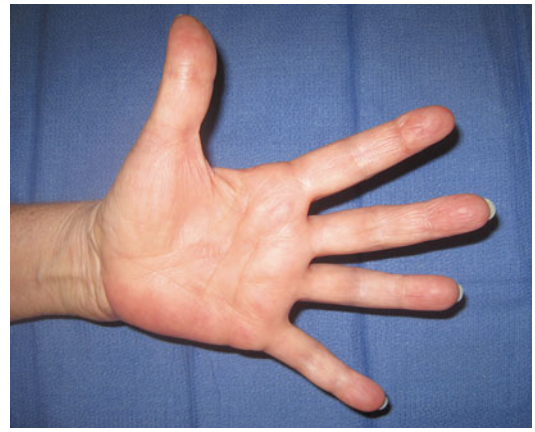


Fig. 14.9 Masse's sign is visible flattening of the hand secondary to loss of the dorsal transverse metacarpal arch combined with hypothenar muscle atrophy

of ulnar nerve compression include clawing (hyperextension of the metacarpophalangeal joint and flexion of the interphalangeal joint) of the ring and small finger secondary to weakness of the third and fourth lumbricals (Fig. 14.8). The claw hand is typically seen in conjunction with a low ulnar nerve lesion where the intrinsics are denervated and unable to flex the MP joints. With intact radial nerve innervated extensors the MP joints hyperextend. Since the FDP to the ring and small finger retain their innervation from the ulnar nerve in the forearm there is flexion of the PIP and DIP joints. Masse's sign is flattening of the hand secondary to loss of the dorsal transverse metacarpal arch and hypothenar atrophy (Fig. 14.9).

On examination the nerve may be palpable and tender at the retrocondylar groove of the elbow [50] Tinel's test involves gentle percussion of the nerve along its course in the retrocondylar groove. Percussion causes paresthesia along the ulnar nerve distribution. The test, however, produces a high number of false positives. Rayan et al. found a positive result in 23.5 % of normal volunteers [51]. Novak et al. described the sensitivities and specificities of a series of tests to diagnose cubital tunnel. A positive percussion test (Tinel's test) had 70 % sensitivity, 98 % specificity, 94 % PPV, and 87 % NPV [52]. The elbow flexion test is performed by flexing the elbow maximally with the forearm supinated and the wrist extended for 1–3 min. This maneuver is analogous to Phalen's test for carpal tunnel syndrome. A positive test reproduces symptoms in the ulnar nerve distribution. After 1 min of compression, the test was found to have 32 % sensitivity, 99 % specificity, 93 % PPV, and 74 % NPV. However, after 3 min of compression, the test was found to have a 75 % sensitivity, 99 % specificity, 97 % PPV, 89 % NPV [52]. Combining the elbow flexion test and pressure at the retrocondylar groove revealed a 98 % sensitivity, 95 % specificity, 91 % PPV, 99 % NPV after 1 min of compression. The scratch collapse test is more recently described by Cheng et al. [53]. The elbow is flexed at the patient's side and the examiner resists external rotation of the shoulder while the area overlying the ulnar nerve is scratched. A temporary loss of resistance is a positive test. Cheng et al. reported 69 % sensitivity, 99 % specificity, 99 % PPV, 86 % NPV.

Imaging is not a frequent part of the work-up for ulnar nerve compression neuropathy, but in some instances it can be useful. Plain radiographs

can identify post-traumatic malunion, heterotopic bone and arthritis. St John and Palmaz found that radiographs displayed abnormalities in 20–29 % of patients with cubital tunnel (versus 6 % in the control group) [54]. Imaging studies should be ordered on an individual basis for patients that display risk factors identified by history and physical examination.

Electrodiagnostic studies can be performed to confirm diagnosis, localize compression, and rule out other disease processes (cervical compression, upper motor neuron disease, thoracic outlet, peripheral neuropathy, etc.). EMG studies evaluate the function of larger myelinated nerve fibers that are vulnerable to compression. Fibrillations and sharp waves reveal whether axonal degeneration has occurred. The first dorsal interosseous muscle is the most commonly affected. The abductor pollicis brevis (T1) should be examined to exclude a C8-T1 nerve root or inferior brachial plexus lesion. Concerns about electrodiagnostic studies exist because just a few normally functioning nerve fibers can lead to an artificially normal result. Greenwald et al. and others believe electrodiagnostic testing is unnecessary in predicting surgical outcomes [55–58].

Absolute slowing of nerve motor conduction velocity at the elbow of <50 m/s supports the diagnosis of cubital tunnel syndrome [59]. Decreased conduction velocity of more than 10 m/s from regions above and below the elbow, decreased amplitude of more than 20 % (Green's), and absence of sensory responses or evidence of muscle atrophy are highly suggestive of cubital tunnel disease [60]. Double crush phenomenon may be detected with F-wave indicating cervical compression or thoracic outlet syndrome [61].

It is postulated that compression induces endoneurial oedema, demyelination and remyelination, inflammation, fibrosis, distal axonal degeneration, growth of new axons, and thickening of the perineurium and epineurium [62]. Ultrasound is a relatively new study that has been used to identify these changes. Wiesler evaluated 15 elbows with cubital tunnel confirmed by clinical exam and NCS and compared them with a control group of 60 elbows from normal volunteers [63]. There was a strong correlation with cubital tunnel and an increase in the cross-sectional area of the nerve. The average cross sectional area was 0.065 cm^2 in controls vs 0.19 cm^2 in the ulnar cubital tunnel group. Pearson coefficient between motor nerve conduction velocity of the ulnar nerve and cross sectional area was 0.80. A cut-off point of cross sectional area of 0.10 cm^2 or higher yielded a sensitivity of 93 % (14/15 elbows) and a specificity of 98 % (59/60 elbows) with a PPV 93 %, and NPV 98 %. The authors caution that greater standardisation is required [63]. Ultrasound can also be useful to detect compression due to an anconeus epitrochlearis [64] ganglion [65], or nerve subluxation [66].

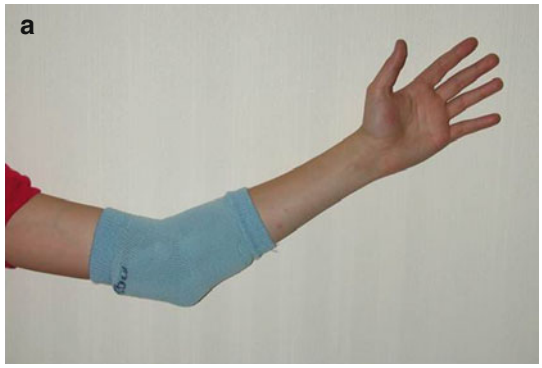
Staging

In 1950, McGowan developed a classification system for ulnar nerve compression based on the severity of motor deficit [67]. Lesions classified as 'grade I' displayed no muscle weakness where 'grade II' lesions exhibited partial weakness and 'grade III' lesions exhibited severe weakness and atrophy. Sensory findings were later introduced into the staging classification in 1988.

Dellon and Amadio created a sophisticated rating system to assess nerve function using a scale from 0 to 10, with 10 representing severe disease with evidence of muscle atrophy [68]. Kleinman and Bishop formulated a 12-point grading scale incorporating objective data and patient reported outcomes [69]. Unfortunately, the complexity of these classification systems limits their clinical utility. Most clinicians utilise a system of involved motor and sensory manifestations to determine severity of nerve compression.

Non-operative Treatment

Non-operative treatment is appropriate for mild to moderate symptoms and includes activity modification and patient education. The patient is taught to avoid activities that result in sustained increased stretch and pressure on the



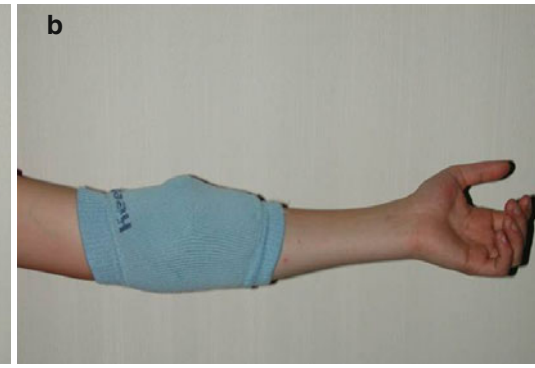


Fig. 14.10 Soft elbow pads (**a**, **b**) are a staple part of non-operative treatment for cubital tunnel and are worn during the day and night to limit flexion to 45–70°. Activity modification, patient education and stretching of the flexor carpi ulnaris muscle are other commonly applied techniques of conservative care

nerve. The patient should be counseled to consider avoiding actions that require the elbow to be fully bent for long periods of time and to avoid resting the elbow on hard surfaces. Soft elbow pads are worn during the day and night splints are provided to limit flexion to 45–70° (Fig. 14.10). Patients are instructed in exercises that stretch the FCU and to avoid sleeping with a flexed elbow. Dimond and Lister reported an 86 % improvement of symptom severity in 73 patients who underwent splinting during an average 8.7 months [70].

Svernlov et al. followed patients with mild to moderate cubital tunnel syndrome who were treated non-operatively [71]. Patients were divided into three groups based on the method of treatment offered (night splints, nerve gliding exercises, or education and activity modification). The authors observed that 89.5 % of patients improved regardless of group. Dellon et al. prospectively studied 121 patients treated non-operatively for a minimum of 3–6 months [72]. The authors evaluated the patients that went on to need surgical treatment. Only 21 % of patients with mild symptoms went on to require surgery within 6 years. Thirty-three percent of the patients with moderate symptoms required surgery within 3 years, while 62 % with severe symptoms required surgery over the same time frame. The authors found that symptom severity was highly correlated with surgical intervention.

Operative Treatment

Operative treatment is chosen for those who failed non-operative treatment and present with weakness, atrophy, and significant denervation on electrodiagnostic studies. Tomaino et al. suggested that subjective symptoms of cubital tunnel syndrome alone warrant operative intervention [56]. The authors argued that electrodiagnostic studies and objective symptoms (atrophy, weakness, loss of two-point discrimination, etc.) reflect a more advanced state of ulnar neuropathy. Advanced disease is associated with potentially irreversible intraneural changes raising the risk for incomplete recovery. In their prospective study, surgical treatment (in situ release and medial epicondylectomy) was offered to patients with McGowan I electrodiagnostic-negative cubital tunnel syndrome who did not benefit from initial implementation of non-operative treatment. Sixteen patients (18 elbows) were enrolled in the study. All patients had complete relief of symptoms following in situ release and medial epicondylectomy and 17 of 18 elbows had return of normal range of motion and grip strength. The authors found that surgery has favorable outcomes and low morbidity in this patient population.

Principles of surgical decompression include release of all sites of potential entrapment, protection of the MABCN, creating a straight path for the nerve to follow if transposed, haemostasis,

and early elbow motion. Treatment options involve simple decompression, decompression with anterior transposition (subcutaneous, intermuscular, submuscular), partial and complete medial epicondylectomy, and endoscopic release. There is controversy in the current literature over which procedure is optimal. The choice depends on surgeon preference as meta-analyses and review of the literature has consistently shown little difference between procedures [6, 73–75]. Absence of consensus on nerve-specific outcome measures limits the ability to design convincing randomised trials for comparison [76].

Macadam et al. performed a recent meta-analysis of ten studies [75]. Analysis was limited to randomised controlled trials and comparative observational studies. The authors compared outcomes of in situ decompression versus decompression and anterior transposition. Over 449 simple decompressions were compared with 457 transpositions for cubital tunnel syndrome. Odds of improvement with simple decompression versus anterior transposition were 0.751, 95 % confidence interval (0.542, 1.040). No difference in clinical outcomes was found between the techniques. Subanalyses on the basis of transposition technique (submuscular versus subcutaneous) showed no statistical differences.

Surgical Technique

In Situ Decompression

In situ decompression was first described by Osborne in 1957 [40]. The technique is performed with a 3–4 cm curvilinear incision centered over the course of the ulnar nerve between the medial epicondyle and the olecranon (Fig. 14.11). Supple skin allows easy subcutaneous dissection. The ulnar nerve is identified proximal to the ligament of Osborne. Passage is created above the nerve proximally and distally. With a deep retractor, the nerve is released approximately 6 cm proximal (Fig. 14.12) and

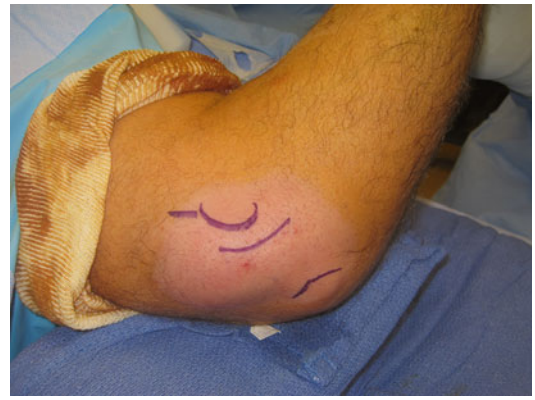
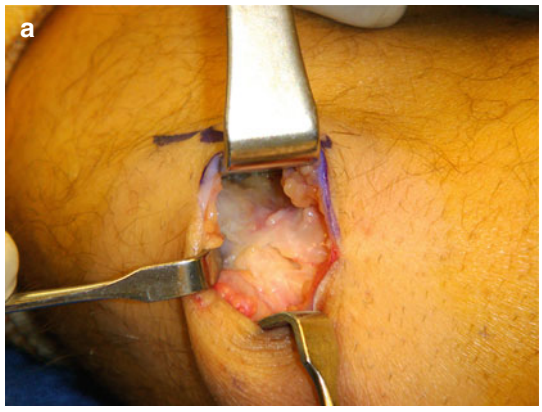


Fig. 14.11 In situ decompression utilizes a 3–4 cm curvilinear incision centered over the course of the ulnar nerve between the medial epicondyle and the olecranon. Supple skin allows easy subcutaneous dissection proximal and distal



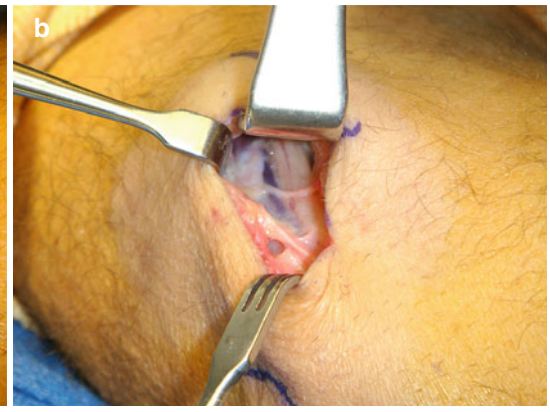


Fig. 14.12 In-situ ulnar nerve decompression preserves the bed of the nerve proximally (shown) and distally. Dissection through the fascia proximal to the medial epicondyle (**a**) is performed to protect branches of the Medial Antebrachial Cutaneous Nerve (MABCN). Complete release of the ulnar nerve proximally with preservation of branch of the MABCN (**b**). Circumferential dissection is not performed as part of this technique

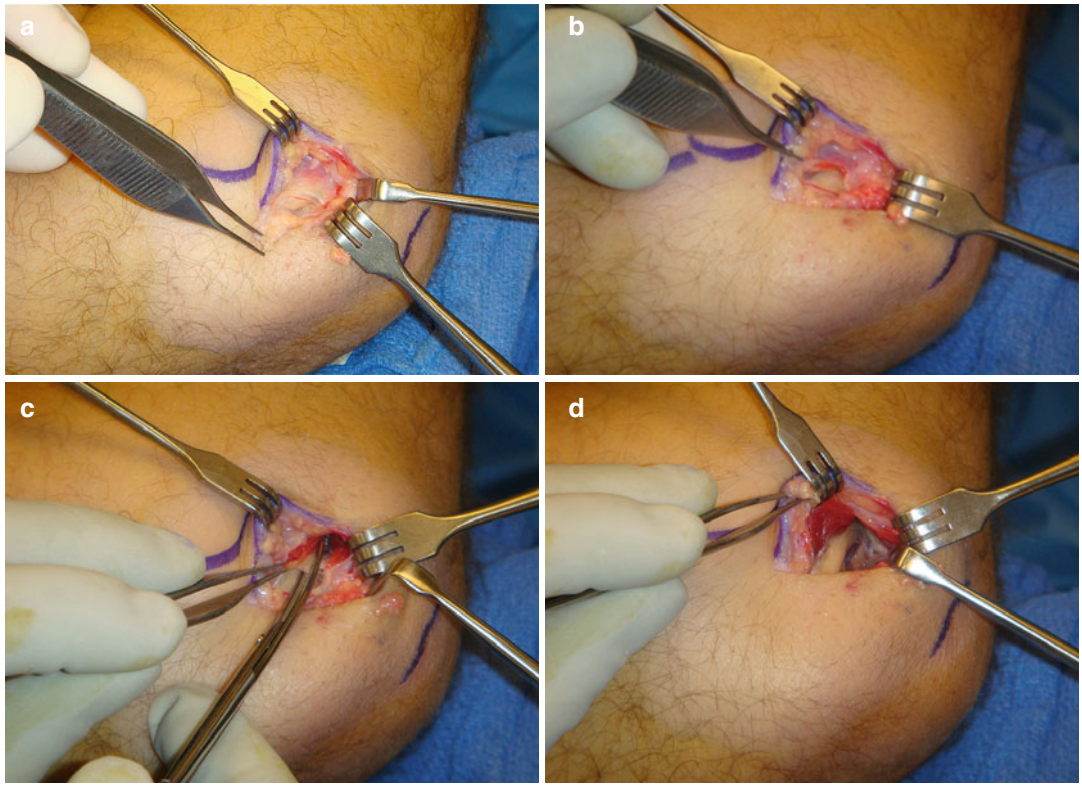


Fig. 14.13 The ulnar nerve distally is identified entering the cubital tunnel beneath the ligament of Osborne (**a**). The ligament is released and dissection is carried up to the two heads of the flexor carpi ulnaris (FCU) (**b**). The deep layer of the aponeurosis between the ulnar and humeral heads of the FCU, termed 'Osborne's fascia', is incised next (**c**). Complete distal release of the ulnar nerve following in situ decompression is shown (**d**)

distal (Fig. 14.13) to the epicondyle until the nerve is surrounded by healthy appearing fat. The approach, like all the methods to be described, takes care to protect branches of the MABCN. Circumferential dissection is not performed as part of this technique [77]. After decompression, the ulnar nerve should be observed through a full range of motion. The nerve should be lax or even redundant in full extension and should remain within the cubital tunnel during elbow flexion (Fig. 14.14). If the nerve subluxates when the elbow is flexed, it should be transposed anteriorly.

Relative contraindications for simple decompression include severe cubitus valgus [6, 78, 79], a subluxing ulnar nerve, advanced compression and recurrence following a previous surgery [6, 68, 80]. There is, however, some controversy over the best treatment for advanced compression. Some have suggested that simple decompression for patients with severe symptoms can yield good to excellent results [7, 81, 82].

The post-operative management involves little or no immobilization. Sling or bulky dressing is provided for the first few days to allow the wound to settle. Immediate return to activities of daily living is allowed as tolerated by the patient, however, heavy lifting is restricted until 4–6 weeks postoperatively.

In situ decompression appears to equal results obtained after anterior transposition [83–85]. Proponents of simple decompression favour its relative simplicity. Another advantage is that it does not influence blood supply [85, 86]. The reported success rate is between 80 and 92 % [8, 87–89].

Bartels prospectively compared simple decompression with anterior subcutaneous

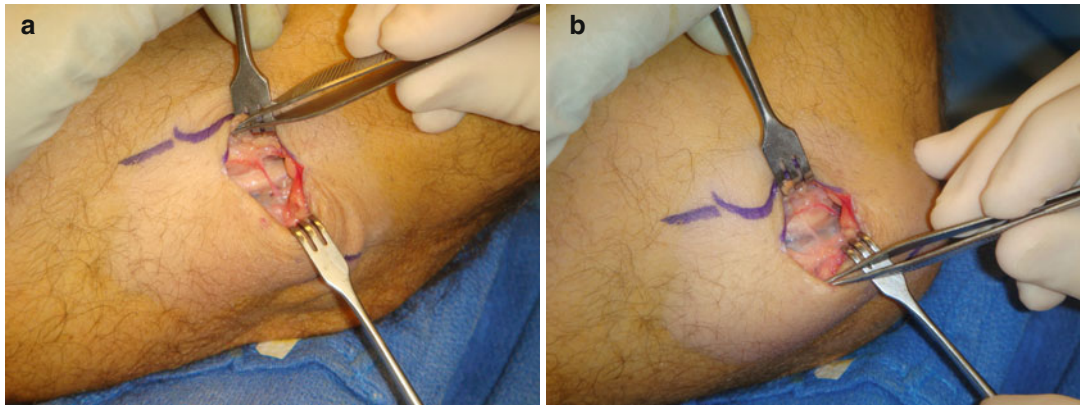


Fig. 14.14 After decompression, the ulnar nerve should be observed through a full range of motion. The nerve should be lax or even redundant in full extension and should remain within the cubital tunnel during elbow flexion. The intraoperative pictures show the position of the ulnar nerve with the elbow in extension (**a**) and flexion (**b**). There is no anterior subluxation with the elbow in a flexed position following decompression

transposition [90]. 100 and 52 patients were followed for an average of 1 year. Outcomes were equivalent. A higher complication rate was observed within the transposition group. A total of 30 complications were observed. Twenty-three complications were found in the anterior transposition group and only seven were found in the simple decompression group. The most common complication was loss of sensation around the surgical scar. Cost analysis found simple decompression to be less expensive. Nabhan followed the results of 66 patients who were randomised to treatment by simple decompression or subcutaneous anterior transposition [85]. No statistical significant difference was found in outcomes (pain, motor, sensory, NCV). The authors recommended simple decompression, describing it as a less invasive procedure.

Anterior Subcutaneous Transposition

The first ulnar nerve transposition was reported by Curtis in 1898 [91]. The goal of transposition is to move the nerve anterior to the elbow axis of flexion and thus create a straight path for the nerve, which decreases tension and presumably enhances neural blood flow. Anterior transposition removes the nerve from the volume limited cubital tunnel and away from sites of mechanical irritation. The basic principles of decompression are the same regardless of technique.

Transposition of the ulnar nerve requires a longer skin incision than simple decompression. The medial intermuscular septum is identified, dissected free and resected. The plexus of veins associated with the posterior surface of the septum must be coagulated. Approximately 4 cm of the septum is excised, beginning at the medial condylar ridge and extending proximally. Particular attention is paid to the proximal and distal ends of the nerve after transposition and care is taken to avoid sharp bends (especially at the FCU). A fascial flap based in proximity to the medial epicondyle can be created. The ulnar nerve is lifted and transposed anterior to the condyle and is held by the fascial sling. Motor branches to the FCU and FDP are preserved. The senior author prefers to create a trough in the distal 25 % of the flexor pronator mass to ease the bend. This eliminates the need for a fascial flap. Additionally, incision of the posterior septum of the humeral head of the FCU allows the nerve to follow a more direct path (Fig. 14.15).

Rehabilitation is similar to in situ decompression. The arm is protected in a bulky dressing and sling for 2 days followed by return to activities of daily living. Weirich studied 36 patients with cubital tunnel syndrome that underwent anterior subcutaneous transposition [42]. Twenty patients

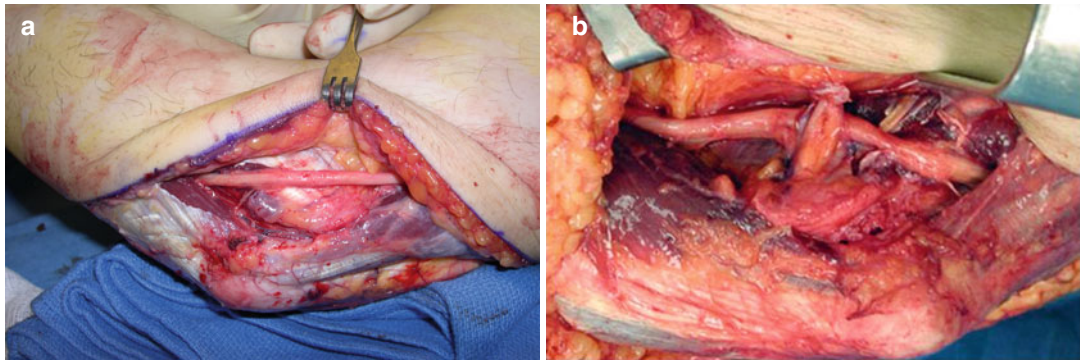


Fig. 14.15 Anterior subcutaneous transposition creates a straight path for the nerve (**a**), which decreases tension and enhances neural blood flow. Particular attention is paid to the proximal and distal ends of the nerve after transposition and care is taken to avoid sharp bends. The ulnar nerve is lifted and transposed anterior to the condyle and is held by the fascial sling (**b**)

underwent immediate post-operative mobilisation and began active range of motion exercises on the day of surgery. Sixteen patients were immobilised (well-padded plaster splints with elbows flexed at 90°) for an average of 14.4 days before beginning active and active-assisted range of motion exercises. Quantitative outcomes were not significantly different between groups (grip strength, pinch, two-point discrimination) at final follow-up. Both groups had improvement in first dorsal interosseous and adductor pollicis muscle strength. However, the immobilised group returned to work later than the mobilised group (2.75 months versus 1 month).

Those that are critical of transposition have concerns about its new superficial location and the extensive dissection required. Circumferential dissection has the potential to decrease blood supply to the nerve [92]. Placing the nerve in a subcutaneous location potentially exposes it to trauma and injury. Because of these concerns, we try to avoid anterior subcutaneous transposition in slender patients with little subcutaneous fat about the elbow.

Richmond followed 18 patients after subcutaneous transposition and observed good to excellent results in 83 % of their patients at mean follow-up of 23 months [93]. Rettig reported good results in athletes that underwent anterior subcutaneous transposition [94]. In his retrospective review of 20 athletes, all returned to full activity and reported minimal to no symptoms following surgery. Similar results were found in other studies [95–97].

Intramuscular Transposition

Intramuscular transposition was initially described by Adson in 1918 [98]. The technique for decompression of the nerve is the same as with other forms of transposition with special attention to the proximal and distal ends of the transfer. Once the nerve is mobilised over the top of the flexor-pronator, a 5–10 mm trough is made in the flexor pronator mass along the course of the nerve in its anterior location [99]. The nerve is placed in the trough. The fascia is repaired with the elbow flexed and the forearm fully pronated. Care is taken to not allow the repaired muscle to compress the nerve. Alternatively the fascia can be left unrepaired. Anecdotally, we have seen no adverse consequences of placing the nerve in a shallow trough in the flexor pronator mass and not repairing the flexor pronator fascia. Following transposition, the nerve should glide freely within the new intramuscular tunnel.

The advantage of intramuscular transposition is that it requires less dissection than submuscular transposition and allows the nerve to follow a straight path. It potentially removes the nerve from a vulnerable subcutaneous position as with subcutaneous transposition. Kleinman and Bishop found this procedure to be simple and

reliable with 87 % good or excellent results in their cohort of 47 patients followed for an average of 28 months [69]. Concerns exist regarding formation of cicatricial scar, although less so than potential scarring following submuscular transposition [23, 100]. Some authors feel the nerve may be vulnerable to traction forces when the flexor pronator muscles contract and have expressed concern that muscle division can lead to post-operative haematoma [32].

Kleinman recommended immobilizing the elbow in a bulky long arm dressing for 3 weeks in mid-pronation and 90° of flexion [101]. Dellon argued that earlier mobilisation can avoid the complication of fibrosis and scar [102].

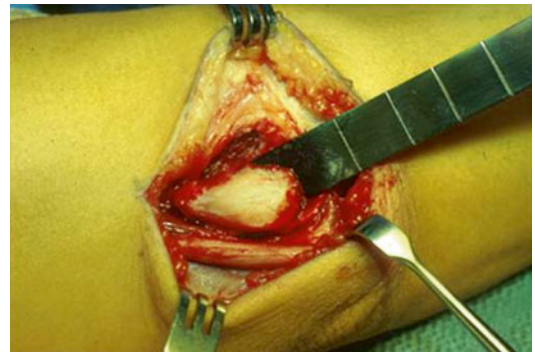


Fig. 14.16 Medial epicondylectomy is performed along the posterior third to half, leaving behind a ridge of bone to protect the attachment of the ulnar collateral ligament. After the osteotomy is made, the flexor pronator mass is repaired

Submuscular Transposition

Submuscular transposition was initially described by Learmonth in 1942 [103]. After the ulnar nerve is decompressed, the entire flexor-pronator mass is detached. A cuff of tissue is left behind for repair to its original position. Dellon offered a modification of the original technique in his description of a Z-lengthening of the flexor pronator fascia [104]. This technique eliminates pressure from submuscular placement of the large diameter nerve. Once again, complete excision of the intramuscular septum proximally and dissection distally to free the ulnar nerve is imperative to prevent potential sites of "kinking" or compression. The ulnar nerve is placed completely beneath the flexor pronator mass and repair of muscle is performed over the transposed nerve [77].

Concerns about submuscular transposition involve the extensive dissection and potential of creating a new site of compression. The need for immobilisation following this procedure has been questioned by some because of the potential for scaring and fibrosis. A study by Weirich compared postoperative immobilisation with early motion [42]. No impairment in outcomes was found between the two groups. However, early mobilisation may allow an earlier return to work as mentioned previously [42, 105, 106].

Biggs et al. prospectively reviewed 44 patients in a randomised series comparing in situ release versus submuscular transposition [83]. In their series, 61 % of patients improved with in situ release and 67 % improved with submuscular transposition. There was a high rate of infection in the submuscular transposition group compared with simple decompression (14 % deep infection rate for the submuscular transposition group versus no infections in the in situ group). Recent studies have confirmed that there are no differences in outcomes between in situ decompression and submuscular transposition [107]. Two recent meta-analyses of the literature determined no difference between simple decompression and anterior transposition of any kind [74, 75].

Medial Epicondylectomy

Medial epicondylectomy for ulnar nerve palsy was first described by King in 1950 [108]. Since its initial description, modifications have been made to the technique. The flexor pronator mass is incised longitudinally and the medial epicondyle is exposed subperiosteally. Partial medial epicondylectomy is performed along the posterior third to half, leaving behind a ridge of bone to protect the attachment of the ulnar collateral ligament. After the osteotomy is made, periosteum over the bone is repaired. A near-complete epicondylectomy can be performed just to the site of insertion of the medial collateral ligament (Fig. 14.16).

Rehabilitation following medial epicondylectomy is the same as all other forms of ulnar nerve

decompression. The elbow is protected in a sling and bulky dressing for 2 days followed by return to activities of daily living. The criticisms associated with medial epicondylectomy include instability from injury to the ulnar collateral ligament, medial elbow pain, and weakness from muscle detachment. When a small portion of epicondyle is preserved, instability is uncommon [109, 110]. Heithoff reported a 10 % loss of grip strength and 5 % loss of pinch strength at average follow up of 2.3 years following epicondylectomy [58]. Medial elbow pain was also noted in 10 % of the patients in his cohort. Despite these results, a recent retrospective review comparing medial epicondylectomy to anterior subcutaneous transposition revealed no differences in outcomes between the two techniques [111].

Endoscopic Release

Endoscopic cubital tunnel release was first described by Tsai in 1995 [112]. The technique offers a minimally invasive alternative to open surgical decompression. It has been suggested that the limited soft tissue dissection will result in shorter recovery time and less scarring [113]. Many variations exist, but most involve a small incision at the condylar groove [112, 114–117]. Space is made between the nerve and fascial covering and overlying subcutaneous adipose tissue. The endoscope and tenotomy scissors are used to release constricting fascial bands over the nerve.

Ahcan et al. published the findings of 36 patients with cubital tunnel that underwent endoscopic release [114]. According to the authors, decompression of 20 cm of nerve was performed through a 3.5 cm incision. Postoperatively, all patients showed improvement by electrodiagnostic testing. Excellent and good results were obtained in 33 out of 36 patients. One complication of a postoperative haematoma that subsequently resolved with no residual symptoms was reported. Hoffmann described similar findings in a cohort of 75 patients (76 endoscopic decompressions) [115]. They found that symptomatic improvement occurred in 96 % of patients and that NCV/NCS improved in all patients. Four patients developed postoperative haematomas that resolved and nine developed numbness in the MABCN distribution (8 out of the 9 improved within 3 months). Watts noted that the endoscopic technique caused less pain and provided greater satisfaction [118].

Cobb et al. examined recurrence of symptoms following 134 consecutive cases of endoscopic cubital tunnel release [113]. The authors observed a recurrence rate between 0.02 and 5.24 %. They noted that endoscopic cubital tunnel release has a recurrence rate that is not higher than open cubital tunnel release literature controls (0.02–5.24 % versus 12 % following open decompression).

Jiang et al. modified the endoscopic technique by using carbon dioxide insufflation to accomplish a subcutaneous dissection anterior to the flexor-pronator mass. In doing so they were able to perform a endoscopic-assisted subcutaneous transposition of the ulnar nerve. All 12 patients had improvement of their symptoms and 10 of 12 were rated as having an "excellent" outcome [119].

While the evidence is anecdotal, it is our opinion that the endoscopic technique carries a higher risk of injury to the ulnar nerve as it is the only technique for ulnar nerve decompression where we have seen or heard reports of ulnar nerve transection. As with any new technique there is a steep learning curve.

Clinical Pearl: Author's Recommended Surgical Approach

Simple in-situ release
Ensure branches of MABCN seen and preserved
Force flexion of elbow to ensure no subluxation
Bulky bandage
Sling for 48 h
Unrestricted use after 2–3 weeks

Failed Operative Treatment

Failure following cubital tunnel release can be defined as the inability to relieve preoperative symptoms or worsening of symptoms postoperatively

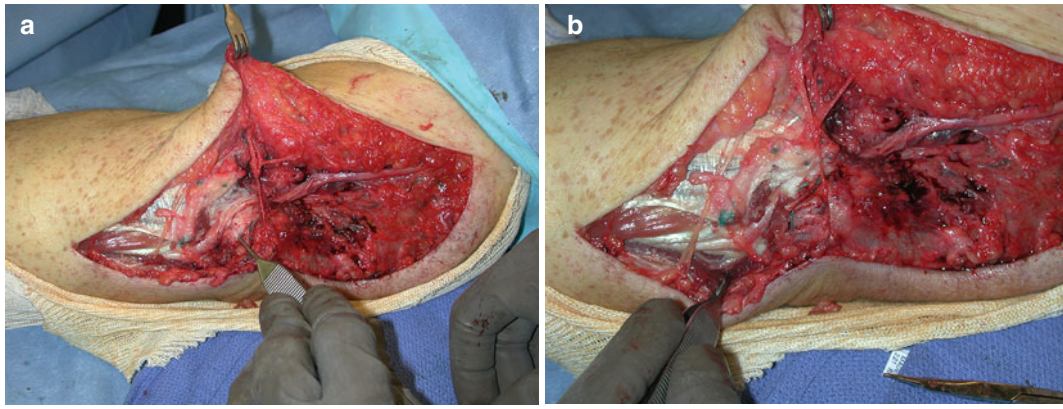


Fig. 14.17 Failed submuscular transposition. Dense cicatricial scar has developed following surgery and is serving as a new site of compression (**a**, **b**)

[28, 120]. Recurrence has been defined as return of symptoms after a 3 month period of resolution following surgery [120, 121]. Reasons for failure and recurrence are multifactorial and are best managed by determining if they have occurred as a result of preoperative, intraoperative or postoperative factors.

Preoperatively, the diagnosis of cubital tunnel can be confused with other sources of nerve compression including cervical radiculopathy, thoracic outlet, and ulnar tunnel syndrome. Patient expectations are another factor that can lead to an unsuccessful outcome. Those patients with long standing cubital tunnel, advanced compression, older patients and those with co-morbidities such as diabetes should be counseled that relief following surgery may be partial or incomplete.

Intraoperative factors that can lead to residual symptoms include inadequate decompression, failed transposition (Fig. 14.17), injury to the MABCN (Fig. 14.18), and residual ulnar nerve instability (Fig. 14.19). Rogers noted that recurrent symptoms are often attributed to incomplete decompression or scar [122]. Injury to a branch of the medial antebrachial cutaneous nerve can occur during exposure. The posterior branch of the MABCN is encountered with some frequency and injury will result in neuroma formation and pain. During in situ decompression, the ulnar nerve should be released but not destabilized causing residual ulnar nerve instability. Transposition must be carefully planned and performed to avoid placing the nerve in a location of injury and to prevent making a new site for compression.

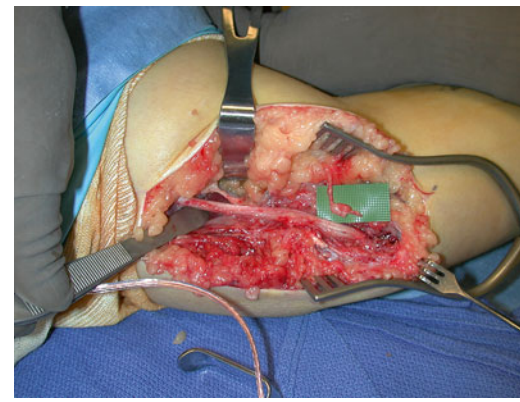


Fig. 14.18 Medial antebrachial cutaneous nerve neuroma following cubital tunnel surgery. Picture taken at time of revision surgery

Dellon and Mackinnon et al. thought the most important factor to obtain successful results after transposition was attention to releasing structures proximal and distal to the region of transfer [102]. Analysis in a primate model showed no evidence of scar after submuscular or intramuscular transposition 3 months following surgery. Broudy reviewed ten patients who had persistent or recurrent symptoms following transposition of the ulnar nerve [100]. Five patients underwent subcutaneous anterior transposition. The ulnar nerve was found surrounded by dense muscular

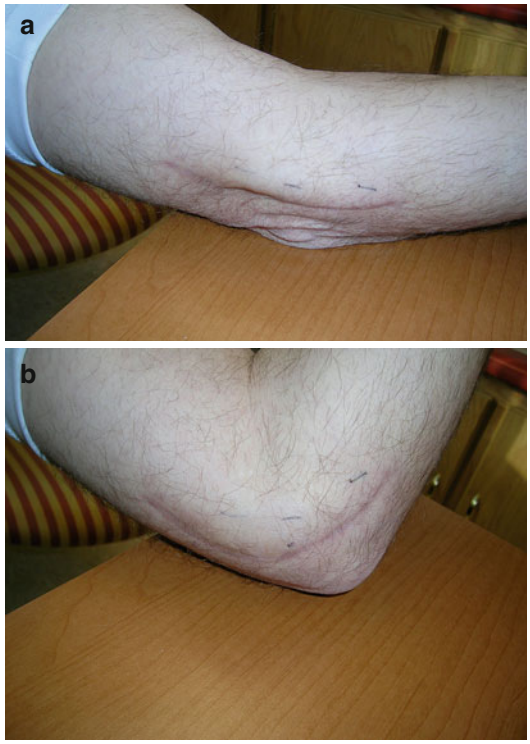


Fig. 14.19 Failed subcutaneous transposition evidenced by residual ulnar nerve instability – subluxation of the ulnar nerve over the medial epicondyle in extension (**a**) and flexion (**b**). Dark dashes on the skin represent the location of the ulnar nerve

scar in four of the five patients and two of the five were found to have a constriction in region of the subfascial sling. Three patients underwent intramuscular transposition. Two developed a dense scar and one was found embedded in muscle with kinking at the cubital tunnel. The one patient that had undergone submuscular transposition had their ulnar nerve compressed beneath the antebrachial fascia overlying the lacertus fibrosis. Only one of the transposed patients showed evidence of ulnar nerve instability; the ulnar nerve was found back in the groove behind the medial epicondyle.

In the acute postoperative period, haematoma can cause recurrent symptoms. Perineural fibrosis and scar can lead to new sites of nerve compression. Immobilisation in the postoperative period has been suggested to increase the potential for cicatricial scar and even contracture formation but studies comparing immobilisation with early mobility have failed to show a difference [42]. Snapping of the medial head of the triceps over the medial epicondyle can occur following ulnar nerve transposition. This is treated by a limited resection of the involved portion of the triceps [123].

Revision Surgery

The decision to perform revision surgery should be offered to patients with persistent or recurrent symptoms who have ulnar nerve symptoms localised to the elbow without significant comorbities. We prefer to have electrodiagnostic studies on all patients considering revision surgery. In these studies we look for persistent or worsening amplitudes, conduction velocities and evidence of motor denervation.

When the primary surgery is decompression without transposition, we favor decompression with subcutaneous transposition when revision surgery is necessary. Goldfarb et al. reviewed 69 extremities in 56 patients who had an in-situ ulnar nerve decompression [124]. Five patients were found to have recurrence of symptoms. All patients with recurrence were treated with anterior submuscular transposition and had relief of their symptoms. The most commonly performed primary procedure is subcutaneous transposition and, as a result accounts, for 60–80 % of failures. Following subcutaneous transposition we will opt for submuscular transposition for the revision procedure.

Conclusion

Ulnar nerve compression neuropathy is common. Diagnosis depends on history, clinical assessment and provocative tests. Role of other modalities like ultrasound and electrodiagnostic studies are useful but await a consensus reference standard to assess their diagnostic utility [46]. If conservative treatment fails, there are many surgical options. In his review of the literature, Dellon stated that the primary factor guiding surgeon choice is "personal bias" [68]. It has been suggested that if surgical outcomes are similar, then least

invasive, least technically demanding procedure should be chosen. Simple decompression offers this advantage. Although endoscopic techniques are becoming an increasingly popular, we prefer to wait for future study to support its reproducibility and delineate its limits. With a proper understanding of the anatomy of the elbow, pathologic locations of cubital tunnel neuropathy, and adherence to surgical principles outlined, good results can be expected with all surgical options we have discussed.

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Ulnar Tunnel Syndrome

15

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Keywords
Ulnar neuropathy • Ulnar tunnel syndrome • Ulnar nerve • Guyon's canal • Compressive neuropathy

Introduction

Ulnar tunnel syndrome results from compression of the ulnar nerve at the wrist. It occurs much less commonly than ulnar nerve compression at higher levels, but should be considered in the differential diagnosis for any patient presenting with signs and symptoms of ulnar neuropathy. The space through which the ulnar nerve passes in the wrist was first broadly described by the French urologist and surgeon Jean Casimir Felix Guyon in 1861 [1, 2], and our understanding of the anatomy and borders of this complex region has subsequently evolved. Unlike carpal tunnel syndrome which is commonly idiopathic, ulnar tunnel syndrome is often secondary to mass effect from another process like ganglion cyst, ulnar artery thrombosis, fracture, anomalous anatomy and etc. For this reason CT or MRI imaging, in addition to plain wrist radiographs and electrodiagnostic studies, is often indicated in the workup. Surgical management addresses the underlying cause of compression. This chapter reviews the clinical presentation of ulnar tunnel syndrome, the relevant patho-anatomy, workup, and longitudinal management.

History

Since ulnar tunnel syndrome is caused by a variety of etiologies that affect virtually all age groups, there is no "typical" age at presentation. The patient will typically present with sensory and/or motor symptoms in the ulnar nerve distribution depending on the location of compression within the ulnar tunnel, as discussed in the Anatomy section. There may be other associated symptoms, depending on the etiology of the compression.

Sensory symptoms include numbness, tingling and parasthesias involving the ulnar nerve sensory distribution. These sensory complaints classically spare the dorsum of the hand, and this detail is helpful in distinguishing ulnar tunnel

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syndrome from cubital tunnel syndrome, which is substantially more common. If sensation on the dorsum of the hand is diminished, the pathology is likely proximal to the dorsal cutaneous branch of the ulnar nerve which emerges about 6–10 cm proximal to the wrist flexion crease [3] – in this case an isolated ulnar tunnel syndrome is considered unlikely. The converse is not true, however; if sensation on the dorsum of the hand is spared, the pathology could be at any level.

Clinical Pearl

	Dorsal sensation intact	Dorsal sensation diminished
Level of compression	Proximal or distal to wrist	Proximal to wrist

Motor symptoms are consistent with intrinsic muscle weakness. Patients may complain of muscle atrophy in the hand, cramps or fasciculations, loss of dexterity, difficulty with fine manipulative tasks, decreased grip strength, weakness specifically when applying torque to a tool or with lateral (key) pinch [4, 5]. In severe cases patients may notice Wartenberg's sign (inability to adduct the small finger), or the development of a claw hand deformity.

Depending on the etiology, onset of symptoms may be sudden or insidious. When associated with fracture or trauma causing compression of the ulnar nerve at the wrist, symptoms develop rapidly; when associated with a degenerative process or a ganglion cyst, symptoms may be intermittent at first, then worsen and become constant.

Associated symptoms are specific to the etiology of the compression. A long history of ulnar-sided wrist pain in an older patient with the insidious onset of an ulnar tunnel syndrome suggests compression related to degenerative pisotriquetral arthritis. Patients with masses compressing the ulnar nerve sometimes complain of fullness in the ulnar aspect of the wrist. Complaints like cold intolerance, pale fingertips or splinter hemorrhages suggest vascular etiologies like hypothenar hammer syndrome or ulnar artery aneurysm. A more quantitative initial and follow-up assessment of cold intolerance is possible with the McCabe cold sensitivity severity scale [6].

A focused social history is useful since understanding the patient's occupation, hobbies and sports can elucidate the diagnosis and direct treatment. Any activity that exposes a patient to vibration or repetitive hand trauma may cause vascular pathologies or fractures. Hypothenar hammer syndrome should be considered for carpenters or laborers, especially when there is evidence of vascular compromise. Cyclists develop compression of the ulnar nerve from prolonged direct pressure. Golfers are prone to acute hook of hamate fractures when the force of impact is transmitted through the club to the hamate hook; in addition to pain, this can lead to symptoms of ulnar neuropathy within the ulnar tunnel [7]. Racquetball and tennis players get chronic hook of hamate fractures by a similar mechanism due to repetitive trauma. The social history should also include an assessment of cigarette smoking, since smokers have an increased incidence of vascular disease, specifically Buerger's disease, which can cause symptoms of ulnar neuropathy in the hand.

Clinical Pearl

Unlike carpal tunnel syndrome, ulnar tunnel syndrome is most commonly the result of compression due to mass effect from another process. Physical examination and imaging focuses on identifying this primary process.

Physical Examination

The physical examination of the hand begins at the neck. Spurling's sign is useful in eliciting symptoms of cervical radiculopathy; Adson's maneuver may be helpful in the diagnostic workup of a suspected thoracic outlet syndrome. The ulnar nerve is examined throughout its course; special attention is paid to the ulnar nerve

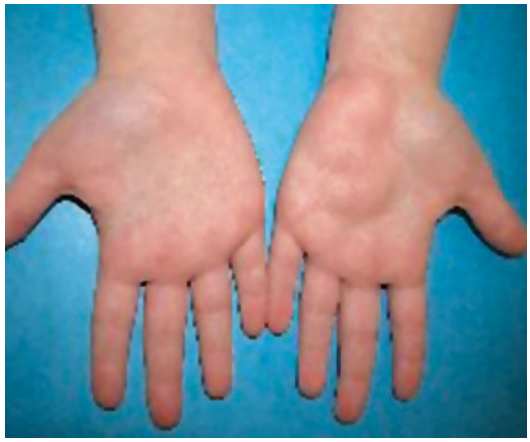


Fig. 15.1 A large mass is demonstrated on inspection of the left palm over the ulnar tunnel in this patient with symptoms of ulnar nerve compression at the wrist. Workup revealed this to be a lipofibroma

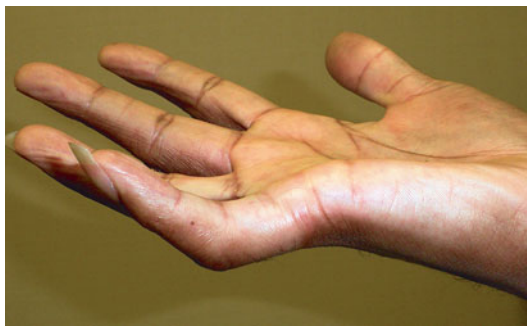


Fig. 15.2 Clawing of the ulnar digits is demonstrated in this preoperative photograph of a patient with long-standing symptoms of ulnar neuropathy

at the elbow and specifically within the cubital tunnel. The relevant techniques and maneuvers are discussed further in applicable chapters. This chapter focuses on the detailed examination of the ulnar nerve at the wrist.

The wrist and hand are inspected dorsally and volarly, with attention to any masses or areas of asymmetry compared to contralateral (Fig. 15.1). The posture of the hand is observed, as advanced ulnar neuropathy may present with a claw-hand deformity (Fig. 15.2) or Wartenberg's sign (Fig. 15.3). Skin changes, bruising, swelling and intrinsic muscular atrophy, especially of the first dorsal interosseus and the abductor digiti minimi,

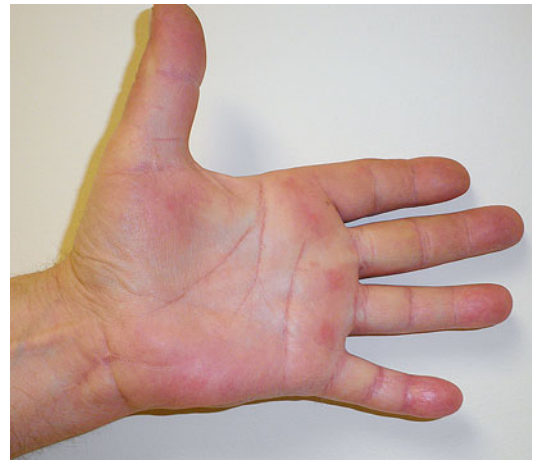


Fig. 15.3 A persistent Wartenberg's sign is demonstrated in this patient with history of ulnar neuropathy treated with decompression. Note the zig-zag incision over the ulnar tunnel

are noted. Splinter hemorrhages or digital pallor suggest a vascular occlusive disease like ulnar artery thrombosis. Advanced vascular insufficiency may cause digital ischemia or gangrene.

A vascular examination is very important when evaluating a suspected ulnar tunnel syndrome. In addition to simple inspection as above, the pulses are palpated, capillary refill is assessed and an Allen's test is performed. If necessary, a handheld Doppler can be used as an extension of the physical examination to evaluate the radial, ulnar and digital arteries, as well as the superficial and deep arch. Decreased signals suggest occlusive pathology. The temperature of the digits is relevant too. Cold fingers, especially if asymmetric, suggest a vascular problem.

The wrist is palpated over Guyon's canal, and any apparent masses are characterized by their texture, mobility, tenderness to palpation, size, location and overlying skin changes. Careful palpation may reveal a pulsatile mass consistent with an ulnar artery aneurysm. The pisiform is palpated, and the pisotriquetral joint is assessed by grinding the pisiform against the triquetrum; tenderness with this maneuver suggests pathology of the pisotriquetral joint that should be considered in the workup. Another exam maneuver relies on the insertion of flexor carpi ulnaris on the pisiform – in a modified apprehension test

similar to the patellar apprehension test, pressure is applied proximal to the pisiform and wrist flexion is resisted. Apprehension with this maneuver suggests pathology of the pisotriquetral joint. The hook of the hamate is palpated. Tenderness here, along with skin changes like calluses, suggests acute or chronic fracture, which may contribute to ulnar nerve symptoms [8].

The ulnar nerve is then evaluated by direct pressure and by percussion over the cubital tunnel and over the ulnar tunnel, in an attempt to elicit a Tinel's sign. First described by Tinel in 1915, this sign is based on the proposition that "pressure on an injured nerve trunk quite often produces a tingling sensation, felt by the patient at the periphery of the nerve and localized to a very precise area of the skin" [9]. Tingling with direct pressure or percussion over the ulnar tunnel suggests pathology at that level.

Sensation in the ulnar distribution is assessed using light touch, pinprick, vibration, and static or moving 2-point discrimination. The exam is performed on the volar and the dorsal surface. If there is sensory loss involving both the volar and the dorsal aspect of the ulnar distribution, then the pathology is proximal to the dorsal cutaneous branch of the ulnar nerve which emerges 6–10 cm proximal to the wrist flexion crease [3] – in this case ulnar tunnel syndrome is considered very unlikely. The converse is not true, however; if sensation on the dorsum of the hand is spared, the pathology could be at any level.

Strength of the hand, wrist, and forearm muscles is assessed and graded on a scale of 1–5. Objective strength measurements can be obtained using a grip meter, such as a Jamar dynamometer, and a pinch meter. Since most of its motor innervation arises distal to the ulnar tunnel, the motor exam is not especially useful in determining the level of compression, however there are two exceptions. At the level of the elbow the ulnar nerve gives off motor branches to the medial half of flexor digitorum profundus (ring and small fingers) and to flexor carpi ulnaris. Asymmetric weakness in these muscles suggests pathology proximal to the motor branches innervating them; however, apparently normal strength on clinical examination could result from the compensatory effect of other muscles like flexor carpi radialis or palmaris longus, and in this case the pathology could be at any level [5].

A variety of special tests have been developed to assess motor function and strength in ulnar-innervated muscles. Froment's sign is compensatory flexion of the thumb interphalangeal joint to compensate for adductor pollicis weakness when attempting a lateral pinch [10]. The basis for this phenomenon is substitution flexor pollicis longus (median-innervated) for adductor pollicis (ulnar-innervated) to produce a lateral pinch. Jeanne's sign is metacarpophalangeal (MP) joint hyperextension to compensate for a weak adductor pollicis during lateral pinch [11].

Strength of the interossei can be assessed by a method described by McPherson and Meals, in which the radial aspects of the patient's abducted index fingers are pressed against one another. If one index finger is overpowered by the other, weakness of the dorsal interosseus is suggested [12]. In the finger flexion sign described by Tsujino and Macnicol, a piece of paper is held between the patient's middle and ring fingers. Interosseus weakness is manifested as metacarpophalangeal flexion, as flexor tendons are recruited to substitute for intrinsic weakness in a mechanism similar to that of Froment's sign [13]. In Wartenberg's sign (Fig. 15.3), "the position of abduction assumed by the little finger" is due to the unopposed action of extensor digiti minimi and extensor digitorum comunis to the little finger, both radially-innervated [14]. The ulnarly-innervated palmar interosseous is weak and cannot counteract the abduction moment. This posture of small finger abduction is most pronounced when the digits are held in extension. In the crossed-finger test, first volar interosseous and second dorsal interosseous weakness is manifested as inability to cross the middle finger over the index finger [15]. Duchenne's sign is caused by weakness of the ulnar-innervated lumbricals, creating a claw deformity of the ring and small fingers due to the unopposed action of the extrinsic digital flexors and extensors [5].

Radiographic Workup and Special Imaging Studies

Unlike carpal tunnel syndrome, imaging studies are essential in the workup of a suspected ulnar tunnel syndrome, since ulnar tunnel syndrome is most commonly secondary to mass effect from another pathology. Radiographic workup begins with plain radiographs including posterior-anterior, lateral, 30° supination view to evaluate the pisotriquetral joint for degenerative or other pathology, and a carpal tunnel view to evaluate the hook of the hamate for fracture.

If bony pathology is suspected or visualized on plain films, CT is useful in better characterizing the bony anatomy. If no bony pathology is suspected, or if soft tissue pathology is suspected or detected on physical examination, MRI is the most useful imaging study after plain films.

If vascular pathology is suspected, a variety of special studies may be considered. Handheld Doppler ultrasound is an extension of the physical examination as described above. Color duplex is useful in distinguishing vascular structures from adjacent masses, and in this way ganglia are distinguished from the adjacent artery, and aneurysms or pseudoaneurysms of the artery can be identified as such. Segmental arterial pressure measurements (expressed as digital brachial index or DBI) and pulse volume recordings are other noninvasive vascular tests used in initial workup. To evaluate a known vascular lesion of the ulnar artery, however, contrast arteriography is the gold standard especially for preoperative planning purposes [16].

Other imaging studies are useful as well. Imaging of the elbow or cervical spine may be indicated in the workup of compressive neuropathies suspected to originate at higher levels. If the patient's presentation includes shoulder pain and a history of smoking, a chest radiograph should be obtained to rule out Pancoast tumor [4].

Electrodiagnostic studies are useful to confirm the diagnosis, to exclude compression at the level of the cervical spine or the cubital tunnel, and to characterize involvement of ulnar nerve motor branches, sensory branches, or both. If muscle wasting is obvious, an EMG should also be done. Increased latencies or decreased nerve conduction velocities across the ulnar tunnel point to pathology at that level.

Clinical Pearl

Bony pathology suspected	CT in addition to plain films
Vascular pathology suspected	Ultrasound exam or contrast arteriography
Soft tissue mass suspected	MRI
Proximal pathology suspected	Elbow XR, chest radiograph or cervical spine imaging
Obvious muscle wasting	EMG

Anatomy: (Figs. 15.4, 15.5, 15.6, and 15.7)

The ulnar nerve arises from the C8 and T1 nerve roots, and emerges as a terminal branch of the medial cord of the brachial plexus. Within the cubital tunnel at the elbow it gives off branches to flexor carpi ulnaris, and to the ulnar half of flexor digitorum profundus. Within the forearm, the dorsal cutaneous branch arises about 6–10 cm proximal to the wrist flexion crease and supplies sensation to the dorso-ulnar hand and fingers. The nerve then passes through the ulnar tunnel where it divides into a sensory branch and a motor branch, as described below. The motor branch then divides to innervate all dorsal interossei, volar interossei, the medial two lumbricals, abductor digiti minimi, flexor digiti minimi brevis, opponens digiti minimi, adductor pollicis, and flexor pollicis brevis – all the small muscles of the hand not innervated by the median nerve [3, 17].

The ulnar tunnel is the space through which the ulnar neurovascular bundle passes at the wrist, and within this confined space the nerve is susceptible to compression. The ulnar tunnel is 4–4.5 cm in length, and unlike the carpal tunnel,

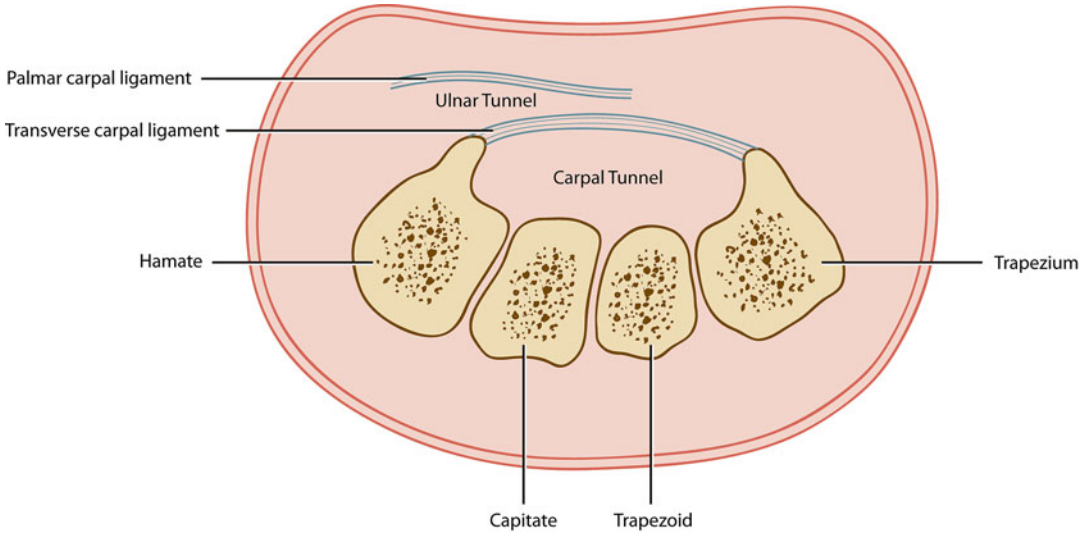


Fig. 15.4 Schematic depiction of the cross-sectional relationship between the ulnar and carpal tunnels in the wrist

the anatomic borders change from proximal to distal. It extends from the proximal edge of the palmar carpal ligament to the fibrous arch of the hypothenar muscles [18]. Within it the ulnar nerve divides into deep and superficial branches. Gross and Gelberman published a landmark anatomical study of this region in 1985, subdividing it into three zones which have since become the standard nomenclature [18].

Zone I begins at the proximal edge of the palmar carpal ligament and ends where the ulnar nerve bifurcates. The floor of Zone I is the transverse carpal ligament proximally and the pisohamate and pisometacarpal ligaments distally; the roof is the palmar carpal ligament proximally and the palmaris brevis distally. Compression of the nerve within Zone I usually presents with combined motor and sensory symptoms, although isolated motor or sensory loss is possible [18].

It is worthwhile noting that the hook of the hamate does not constitute the radial border of the ulnar tunnel, but instead forms part of the floor. Thus the neurovascular structures are not constrained radially by the hook of the hamate [19]. However, pathology of the hamate can still cause compression in the ulnar tunnel [18].

Zone II contains the deep (motor) branch of the ulnar nerve, which courses deep to the sensory branch. The distal extent of Zone II is the fibrous arch between the abductor digiti minimi and the flexor digiti quinti. The innervation to these muscles usually arises just proximal to the arch. The motor branch then passes beneath the arch, pierces and innervates the opponens digiti minimi, then curves around the hook of the hamate. Compression within Zone II results in one of two patterns of motor loss. If the compression is proximal, there is loss of all ulnarly-innervated muscles. If compression is distal, however, the hypothenar muscles are spared because these branches arise proximal to the arch [18].

Zone III contains the superficial branch of the ulnar nerve which courses superficial to the motor branch. The palmaris brevis forms the roof of zone III proximally, and receives its motor innervation from the superficial branch as it passes beneath. From this point on however, the superficial branch is only sensory. Distally, the roof of Zone III is formed by fibrofatty tissue which is less rigid, so isolated compression of the superficial branch is relatively uncommon. Compression within Zone III results in sensory loss to the small finger and the ulnar aspect of the ring finger [18].

Variations in the anatomy of the ulnar nerve have been described. Lindsey and Watumul described different patterns of arborization of the

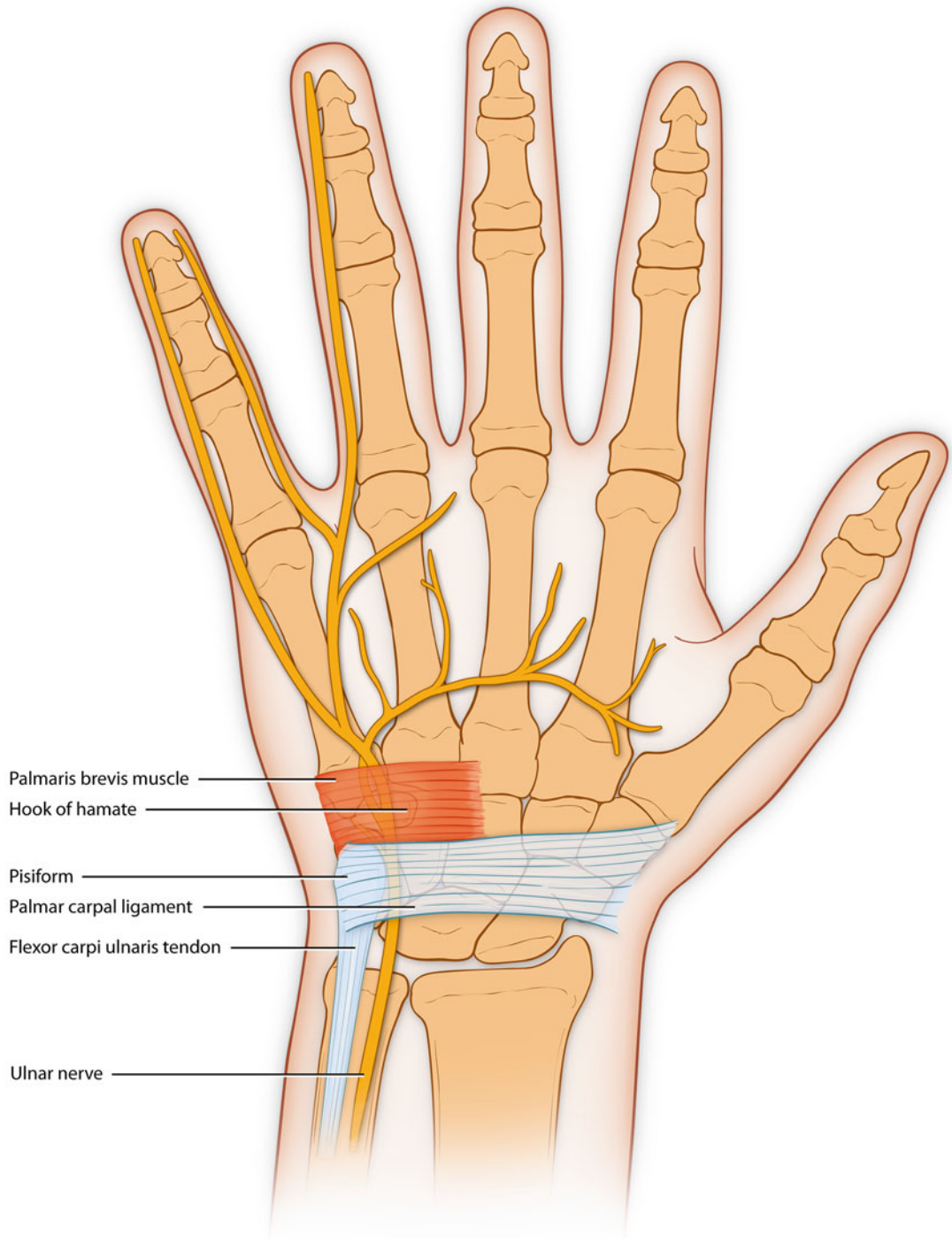


Fig. 15.5 Schematic depiction of the roof of the ulnar tunnel

ulnar nerve within the ulnar tunnel, based on their study of 31 cadaveric upper extremities. In most specimens the nerve bifurcated into a main sensory trunk and a motor branch, however in 6 of 31 specimens the nerve trifurcated into two digital sensory branches and a motor branch. With

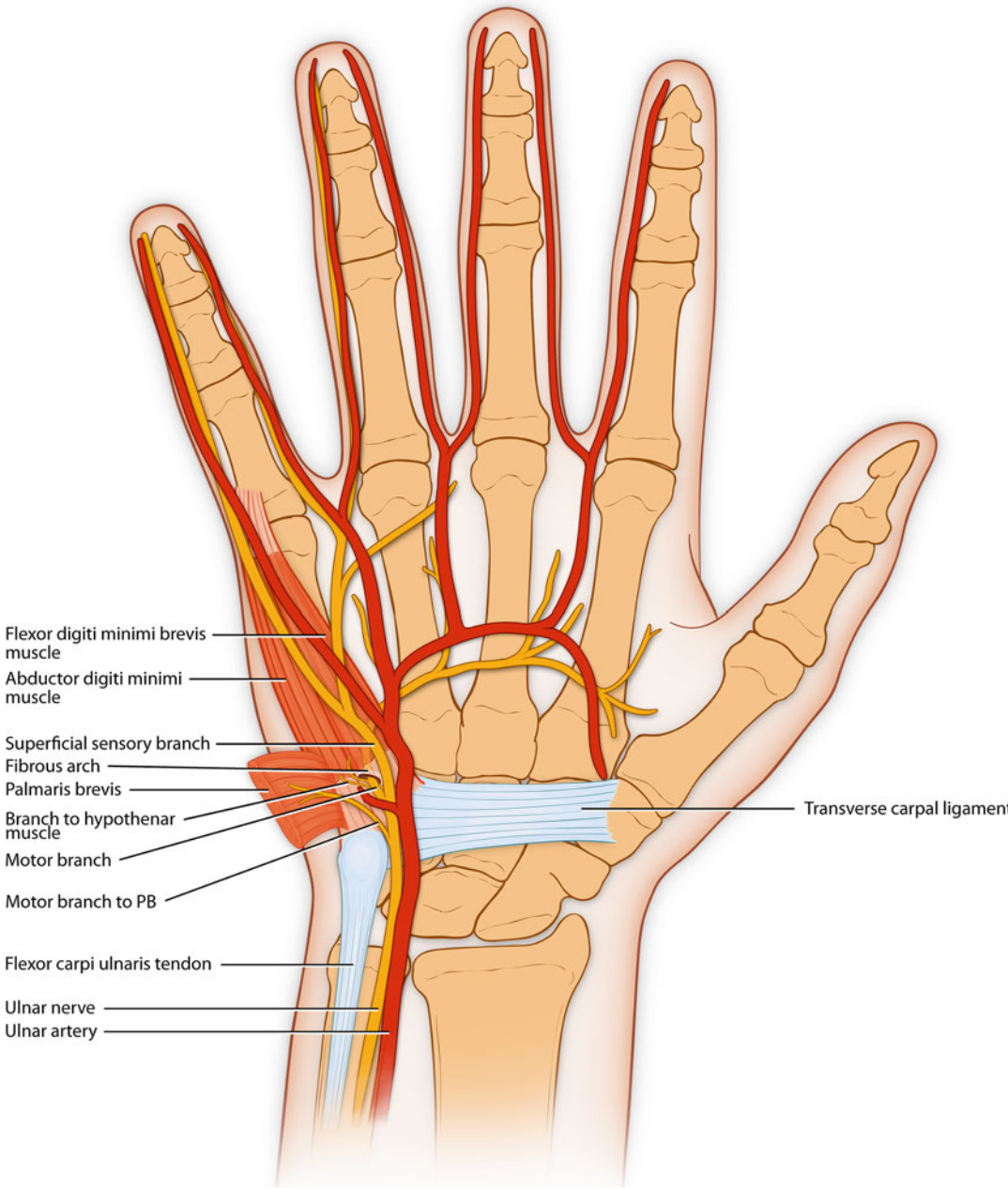


Fig. 15.6 With superficial structures reflected, this illustration depicts the relationship of the ulnar artery and nerve as they bifurcate within the ulnar tunnel

regard to motor innervation, 14 of 31 specimens had two branches innervating the hypothenar muscles; 10 had only one branch; in 7 specimens there were three or more branches innervating the hypothenar muscles [20].

Differential Diagnosis

The most common cause of ulnar neuropathic symptoms in the hand is neurologic compression within the cubital tunnel at the elbow, or at the

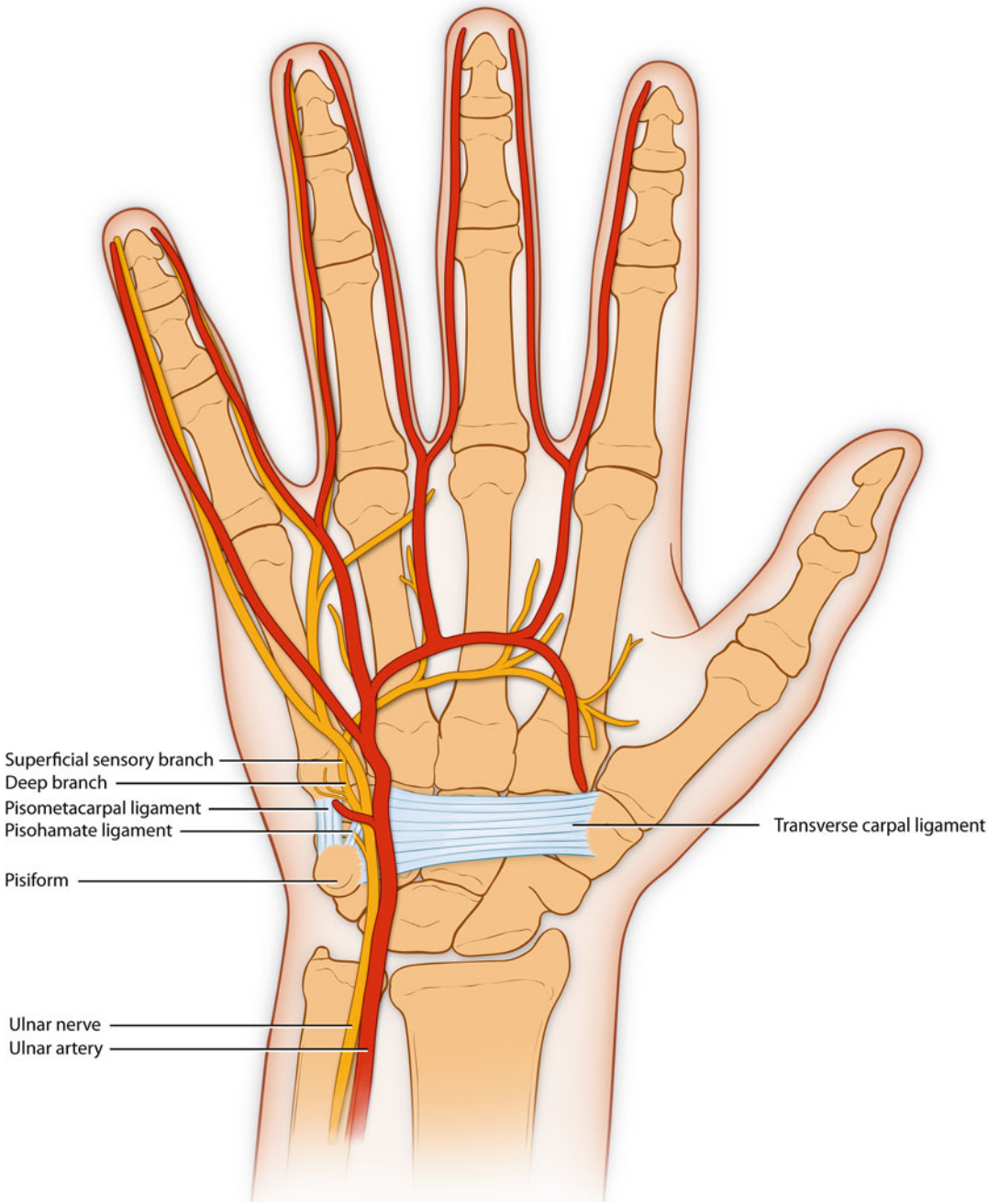


Fig. 15.7 With most soft tissue structures removed, this diagram depicts the floor of the ulnar tunnel and the relationship of the neurovascular structures to the carpus

level of the cervical spine. Thoracic outlet syndrome is another possible etiology. These should be ruled out prior to working up ulnar tunnel syndrome.

The "double-crush" phenomenon is the predisposition of patients with high-level nerve compression to develop a secondary site of compression at a lower level, first hypothesized

by Upton in 1973 [21], and subsequently corroborated by many authors. If a patient has cervical stenosis contributing to symptoms of ulnar neuropathy in the hand, there may be an additional site of compression distally, including the ulnar tunnel. This "double-crush" phenomenon should be considered especially for patients whose symptoms do not resolve after a more proximal pathology has been addressed definitively.

If the source of pathology is the ulnar tunnel itself, symptoms are frequently the result of another process causing mass effect. Table 15.1 is a list of common and uncommon etiologies. Of these, several deserve special mention.

Table 15.1

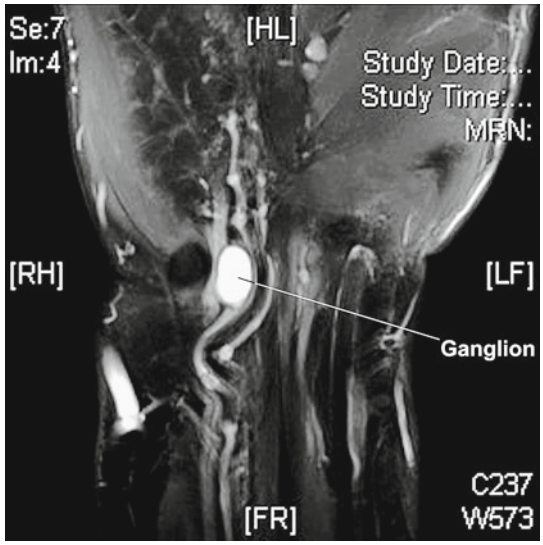
Masses causing external compression
Ganglion cyst
Hemangioma
Lipofibroma
Giant cell tumor
Neurofibroma
Anatomic anomalies
Accessory carpal ossicle
Anomalous hypothenar muscles or palmaris brevis muscle belly
Bipartite hamulus
Thickened pisohamate ligaments
Thickening of the proximal fibrous hypothenar arch
Degenerative
Degenerative disease about the pisiform-triquetral joint
Osteoarthritis of the radioulnar and carpal joints
Iatrogenic
Following opponensplasty
Inflammatory
Edematous fibrous tissue
Hypertrophic synovium
Rheumatoid tenosynovitis
Edema secondary to burn or insect bite
Gout
Neuropathic
Demyelinating focal mononeuropathy
Repetitive trauma neuropathy
Occupation-related
Vibration exposure
Hypothenar hammer syndrome
Direct pressure on ulnar nerve with wrist extended (typing, cycling)
Prolonged hyperextension
Traumatic
Direct trauma
Distal radius fracture with displaced ulnar fragment
Hook of hamate fracture
Fracture of other carpal bones or metacarpals
Vascular
Ulnar artery thrombosis
Hypothenar hammer syndrome
Ulnar artery pseudoaneurysm
Thromboangiitis obliterans (Buerger's disease) or other vasculitis
Other
Idiopathic

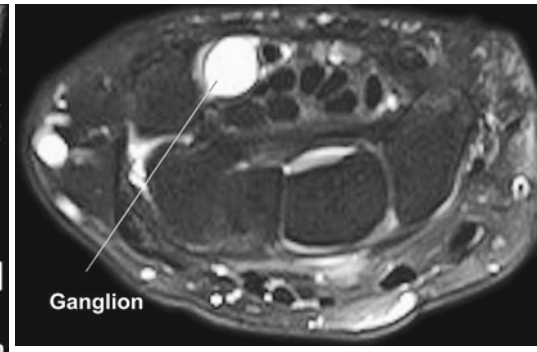
Ganglion Cyst (Figs. 15.8 and 15.9)

Ganglion cyst the most common cause of compression within the ulnar tunnel, and can arise in any of the three zones. The symptoms differ depending on which zone the ganglion compresses, as above. In addition to motor and/or sensory complaints, patients may endorse pain or fullness in the wrist at the level of the ganglion [1].

Ulnar Artery Thrombosis and Hypothenar Hammer Syndrome (Fig. 15.10)

Ulnar artery thrombosis was the most common cause of Zone III compression in Gross and Gelberman's study [18], and it is the most common occlusive disorder of the upper extremity [16]. When caused by repetitive trauma or vibration exposure it is called hypothenar hammer syndrome. When the ulnar artery is completely occluded, perfusion of the hand relies entirely on the radial artery, and this will be detected on physical examination with an Allen's test. Cold, ulcerated fingertips of the ulnar digits and splinter hemorrhages may be evident on examination, secondary to downstream embolization. Conservative treatment including smoking cessation, vasodilators, and cold avoidance, but when DBI <0.7 surgical treatment is indicated. This often entails reconstruction of the segment using a graft [16, 22].





Figs. 15.8 and 15.9 A ganglion cyst compressing the adjacent ulnar nerve is demonstrated on these T2 fat suppression MRI images of the wrist in a 56 year old female with symptoms of ulnar neuropathy

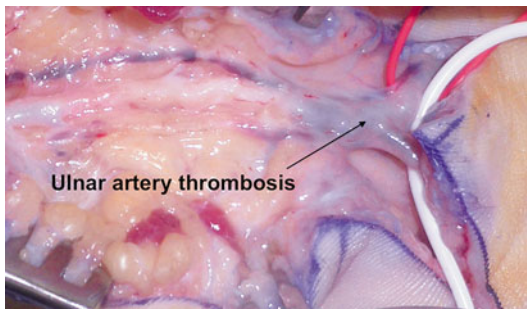


Fig. 15.10 Ulnar artery thrombosis. Note the characteristic corkscrew appearance of the distal artery

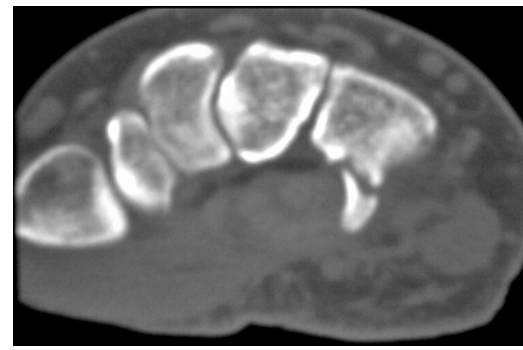


Fig. 15.11 Hook of hamate fracture demonstrated on axial CT images

Ulnar Artery Aneurysm

Ulnar artery aneurysms and pseudo-aneurysms are the most common vascular tumors of the upper extremity. As mentioned above, color duplex is useful in making the diagnosis, but arteriography is best for characterizing the mass prior to surgery. These pulsatile masses are almost always surgically resected and reconstructed to prevent downstream embolization of thrombus [16].

Hook of Hamate Fracture (Fig. 15.11)

Hook of hamate fractures are seen in golfers, tennis players and baseball players, and occurs when the force of impact is transmitted through the bat, club or racquet to the hamate hook at the end of the handle. Since the hamate hook forms part of the floor of the ulnar tunnel, pathologies of the hamate hook can affect the ulnar nerve. Although there are published reports of successful open reduction and internal fixation of these fractures, the preferred treatment is frank excision of the fractured fragment, especially in athletes [7, 8].

Associated with Carpal Tunnel Syndrome

Up to one-third of all patients with carpal tunnel syndrome have a concurrent ulnar tunnel

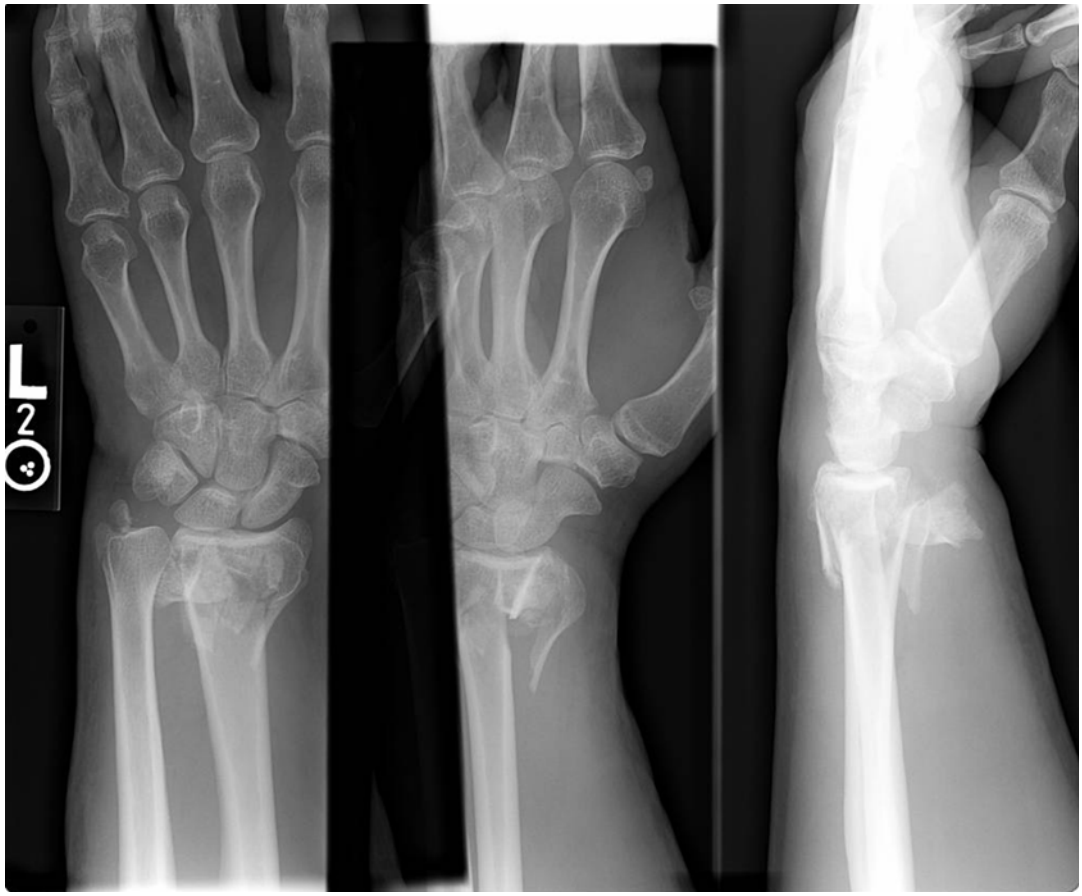


Fig. 15.12 Comminuted distal radius fracture with volarly-displaced ulnar fragment

syndrome, which is indirectly decompressed when the carpal tunnel is released. For this reason a dedicated ulnar tunnel release is usually unnecessary in these patients [1, 23].

Treatment

Management of ulnar tunnel syndrome depends on the etiology. In the case of ulnar tunnel syndrome secondary to repetitive trauma, as in bicyclists, conservative management consists of cessation of the provocative activity and splinting in a neutral position [1]. Injection of the ulnar tunnel is not undertaken since the etiology is rarely inflammatory, and because neurovascular injury can result from introducing a needle into this confined space.

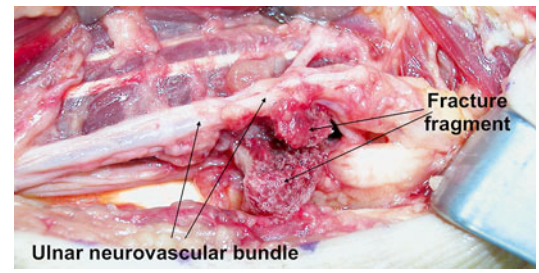


Fig. 15.13 Intraoperative photograph demonstrating the ulnar nerve severely tented by the fracture fragment seen in Fig. 15.12

Surgical management addresses the underlying pathology. If secondary to fracture, the fracture is reduced. If a complete ulnar nerve palsy is present following open or closed fracture reduction, exploration is indicated (Figs. 15.12 and 15.13) [1, 24].

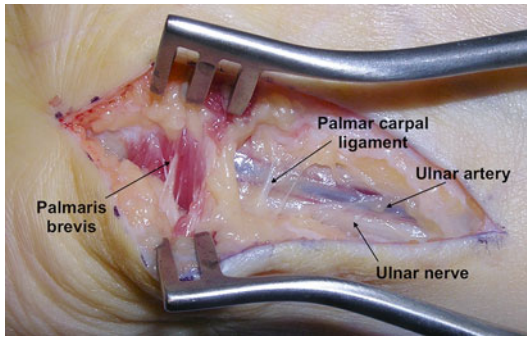


Fig. 15.14 Superficial surgical dissection

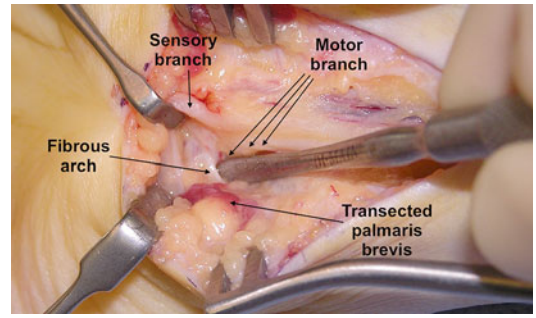


Fig. 15.15 Deep surgical dissection. The superficial and deep branches are demonstrated. The freer elevator has been placed beneath the fibrous arch, in front of the deep branch

Ganglia compressing the ulnar tunnel are resected, and vascular pathologies often necessitate reconstruction as discussed above. Hook of hamate fractures are excised, and idiopathic cases associated with carpal tunnel syndrome respond to isolated carpal tunnel release. Sometimes an underlying etiology is not discovered, and the patient's persistent symptoms may necessitate surgical exploration and release. The following section details the surgical exposure and release of an idiopathic ulnar tunnel syndrome.

Surgical Technique

The patient is positioned supine on the operating table. The extremity is elevated and exsanguinated. The pisiform is palpated and marked. The hook of the hamate is 1 cm distal and 1 cm radial to the pisiform; this is marked as well. A curvilinear or zig-zag incision passing between these two points is carried proximally to the level of the distal wrist flexor crease. The dissection is carried through the subcutaneous tissues until the roof of the ulnar tunnel is identified.

Palmaris brevis crosses the incision distally, and the palmar carpal ligament crosses the incision proximally, forming the roof of the ulnar tunnel (Fig. 15.14).

The palmaris brevis and the palmar carpal ligament are transected and retracted. The superficial and deep branches are identified (Fig. 15.15). A small blunt elevator is placed beneath the fibrous arch in front of the motor branch to protect it; the fibrous arch is then released (Fig. 15.16). The skin is closed and a soft bulky dressing is applied.

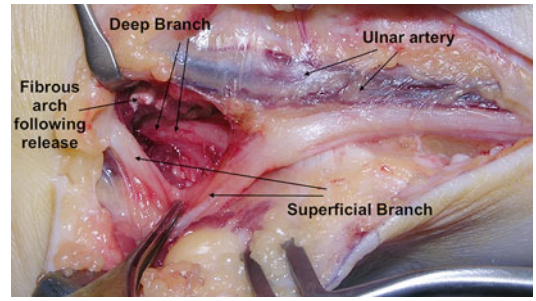


Fig. 15.16 Following release of the fibrous arch. The superficial and deep branches are clearly demonstrated

Summary

Ulnar tunnel syndrome results from compression of the ulnar nerve at the wrist. Within this complex region the ulnar nerve bifurcates into motor and sensory branches, so compression in different regions of the ulnar tunnel creates different combinations of sensory and motor symptoms. Although sometimes idiopathic, ulnar tunnel syndrome is commonly secondary to mass effect from another process; radiographic workup, in addition to careful physical examination, is therefore necessary to identify the associated condition. Treatment addresses the underlying etiology.

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Compressive Neuropathies of the Radial Nerve

16

Bradley C. Carofino, Allen T. Bishop, and Alexander Y. Shin

Keywords
Posterior Interosseous Nerve Compression • Radial Tunnel Syndrome • Wartenberg's Syndrome

Introduction

Compressive neuropathies of the radial nerve are less common than those of the median and ulnar nerves. The annual incidence of all radial nerve entrapments combined is approximately 0.003 % [1]. Compressive neuropathies of the median and ulnar nerves occur 100 times and 10 times more frequently, respectively [2, 3]. Nevertheless, radial nerve entrapment does occur and can produce significant patient morbidity. Most commonly this occurs in the form of radial tunnel syndrome (RTS), posterior interosseus nerve (PIN) entrapment or Wartenberg's syndrome. RTS and PIN entrapment are both compressive neuropathies of the PIN, while Wartenberg's syndrome results from compression of the superficial sensory branch of the radial nerve (SBRN). Although these are the most common clinical entities, radial nerve entrapment can occur at nearly any point along its course, such as at the level of the latissimus tendon, triangular interval, intermuscular septum and triceps [4–6]. These less common possibilities will not be reviewed here but should be remembered in clinical practice.

Radial Nerve Anatomy

The radial nerve arises from the posterior cord of the brachial plexus and receives contributions from the nerve roots of C5–C8. Anterior to the subscapularis muscle the posterior cord divides into the axillary and radial nerves. The axillary nerve passes beneath the subscapularis muscle to enter the quadrangular space while the radial nerve continues distally passing anterior to the teres major and tendinous insertion of the latissimus dorsi [7]. The radial nerve proper then enters the posterior compartment of the arm by traveling through the triangular interval, which is bordered superiorly by the teres major, medially by the long head of the triceps, and laterally by the shaft of the humerus. The profunda brachii artery accompanies it through this region.

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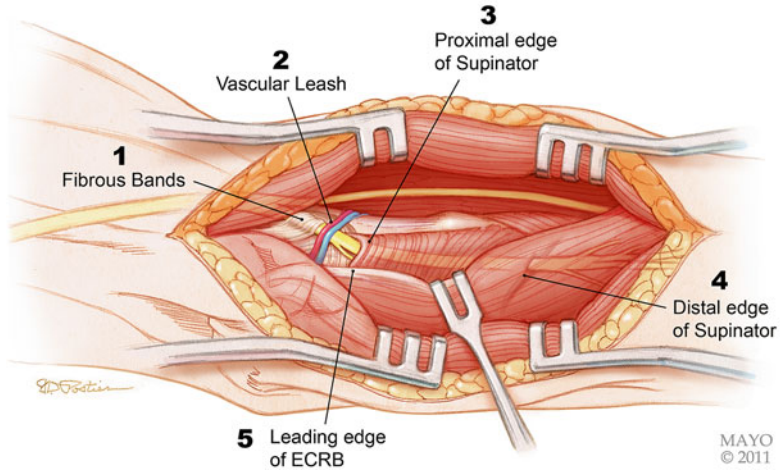


Fig. 16.1 Common PIN compression sites ("Copyright [2011] Mayo Foundation for Medical Education and Research")

The nerve descends the posterior aspect of the arm lying on top of the medial head of the triceps, and does not lie on the bone itself until it crosses the lateral aspect of the humerus within the spiral groove [8]. The nerve then pierces the intermuscular septum to enter the anterior compartment of the arm approximately 12 cm proximal to the lateral epicondyle [8–10]. The nerve lies between the brachioradialis and the brachialis as it travels through the antecubital fossa. One to three centimeters distal to the lateral epicondyle the radial nerve divides into the posterior interosseus nerve and the superficial sensory branch [11]. These travel together for a short distance before the PIN heads posteriorly to enter the radial tunnel and the SBRN continues beneath the brachioradialis.

The radial tunnel is a defined anatomic region which begins at the level of the radio-humeral joint and extends distally for approximately 5 cm [12]. The floor of the tunnel is formed by the radio-capitellar joint capsule proximally and the deep head of the supinator distally. Proceeding from proximal to distal the roof of the tunnel is formed by the fascial bands connecting the brachialis and brachioradialis muscles, the origin of the ECRB, the branches of the radial recurrent artery (Leash of Henry), and the origin of the superficial head of the supinator (Arcade of Frohse). These four structures, as well as the supinator muscle, may compress the posterior interosseus nerve (Fig. 16.1). The most common site of entrapment is the arcade of Frohse, followed by either the tendinous origin of the ECRB or leash of Henry [12, 15].

The Arcade of Frohse, which is the proximal margin of the supinator muscle, forms an opening of variable size for the PIN to pass through. The arcade is anchored laterally at the most lateral aspect of the lateral epicondyle. It then arches distally and reattaches at the medial border of the lateral epicondyle adjacent to the articular surface of the capitellum (Fig. 16.2). The lateral portion of the arcade is always fibrous while the medial portion is typically muscular. However, Spinner found that the medial portion could be fibrous in 30 % of patients [16]. Subsequent studies have shown that this anatomic variation may contribute to PIN compression [17, 18]. The origin of the ECRB muscle is a fascial structure that arises from the common extensor origin, and collateral ligament. It is continuous with the proximal aspect of the supinator muscle.

After exiting the radial tunnel, the PIN passes between the two head of the supinator as it winds around the proximal third of the radius. At the level of the bicipital tuberosity, there is a bare area between the insertions of the deep and superficial heads of the supinator [19]. In this location the nerve lies directly against the radius and is vulnerable to traumatic or iatrogenic injury. After exiting beneath the distal edge of the supinator, the PIN gives off superficial and deep motor branches [19, 21]. The PIN continues distally along the floor of the fourth extensor compartment and provides afferent fibers to the dorsal wrist capsule, and intercarpal joints. The superficial sensory branch of the radial nerve travels down the forearm deep the brachioradialis muscle. It then

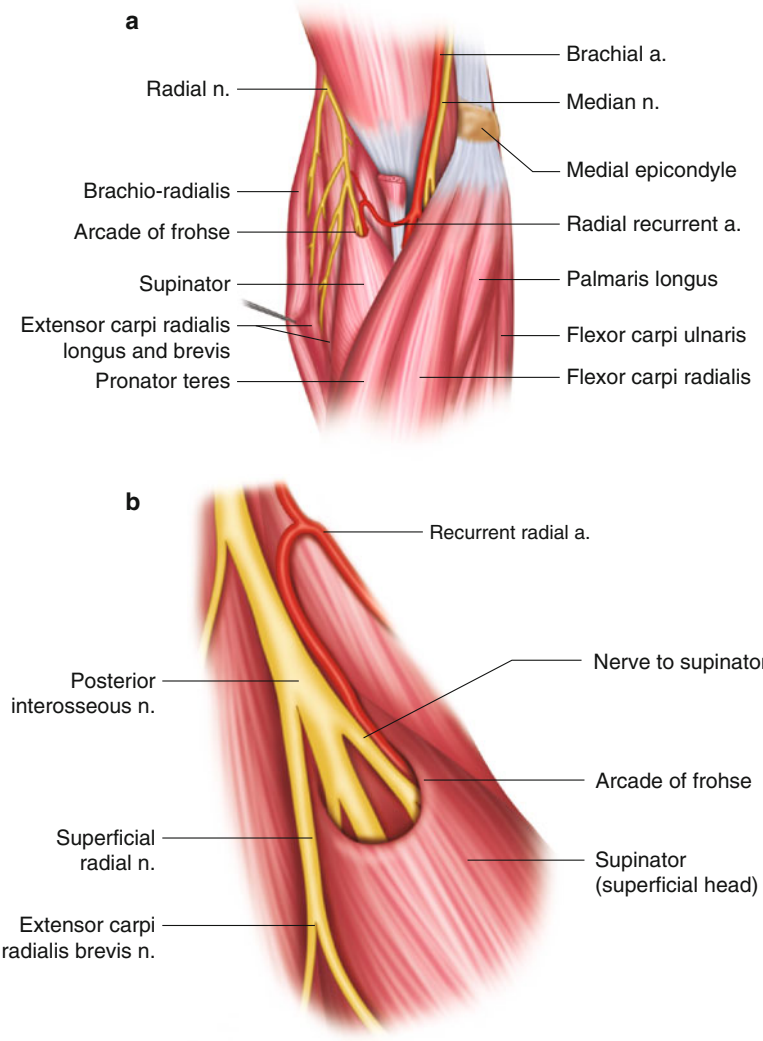


Fig. 16.2 Diagram of arcade of Frohse (Reprinted with permission from Spinner [16])

emerges between the brachioradialis and the ECRL, travels subcutaneously and supplies sensation to the dorsoradial aspect of the hand.

The branches of the radial nerve arise in a predictable pattern which can help localize a site of entrapment or injury. The first branch is the posterior brachial cutaneous nerve which originates in the axilla. As the nerve travels along the posterior aspect of the arm it supplies the triceps and anconeus muscles, then just before crossing the intermuscular septum the inferior lateral brachial cutaneous and posterior antebrachial cutaneous nerves originate. Proximal to the elbow the radial nerve proper supplies the brachioradialis and ECRL; although in some individuals the ECRL is innervated by branches of the PIN [22]. In 50 % of individuals the radial nerve also provides partial innervation to the brachialis along with the musculocutaneous nerve [22]. The branch to the ECRB may also arise in this region. In most individuals the ERCB is supplied by the PIN (45 %), but can arise from the radial nerve proper (24 %) or sensory branch (16 %) [22, 23]. The PIN innervates the supinator as it passes through this muscle, and supplies the remainder of the finger, thumb and wrist extensors upon exiting. The order of innervation from proximal to distal is the extensor digitorum communis (EDC), extensor digiti minimi (EDM), extensor carpi ulnaris (ECU) which are all supplied by the superficial branch, followed by the abductor pollicus longus (APL), extensor pollicus longus (EPL), extensor pollicus brevis (EPB) and extensor indicis proprius (EIP) which are supplied by the deep branch [19–21].

Compressive Neuropathies of the Posterior Interosseous Nerve

Although symptoms are entirely different, RTS and PIN compression syndrome are both compressive neuropathies of the posterior interosseus nerve. PIN compression syndrome results in motor weakness of the PIN innervated finger, thumb and wrist extensors, and is not associated with pain. In contrast, RTS is characterized by pain localized to the proximal lateral aspect of the forearm, and is not associated with motor deficits.

The potential causes of compression are similar for both disorders, and can be secondary to trauma, tumours and constricting anatomy. The most common aetiology for both disorders is compression by one of five anatomic structures: (1) Fibrous bands between the brachialis and brachioradialis, (2) the Recurrent Leash of Henry, which are the branches of the recurrent radial artery that cross over the radial tunnel to supply the extensor musculature (3) the tendinous origin of the ECRB, (4) the Arcade of Frohse, and (5) the supinator muscle. These are often remembered by the mnemonic FREAS. Tumors are more often encountered in PIN compression syndrome than RTS, and reported cases include ganglions, lipomas, periarticular synovitis, and arteriovenous malformations [24–28]. Compression might also result as a sequelae of trauma such as a fracture of the proximal radius or radial head dislocation [29]. Also, iatrogenic injury secondary to overly aggressive retraction or dissection during radial head surgery may cause PIN injury. Finally, microtrauma from repetitive pronosupination has been suggested. To this point, the literature contains a number of case reports describing palsies in occupations that require repetitive motion such as an orchestra conductor, violinist, and swimmer [30].

Clinical Pearl: Aetiology or Radial Tunnel/ PIN Compression ("FREAS")
Fibrous bands
Recurrent leash of Henry
ECRB origin
Arcade of Frohse
Supinator
- Others – tumour, trauma

PIN Compression Syndrome

Clinical Evaluation

This can manifest as either a complete or incomplete paresis of the PIN innervated muscles, and it may be progressive over a period of days to weeks. It is a motor neuropathy only without a sensory component, and it not typically associated with pain. Patients will present with some combination of an inability to extend the metacarpal phalangeal joints of some or all fingers (EDC), extend the thumb IP (EPL) and radially abduct the thumb (APL). Wrist extension may be weakened due to ECU paralysis, and obligatory radial deviation with extension will occur if the ECRB is also affected. This is determined by the innervation of the ECRB which may be derived from the PIN in 45 % of individuals [23]. Forearm supination can be weakened but in order to detect this strength testing should be performed with the elbow extended to eliminate the biceps contribution.

The differential diagnosis for PIN compression includes extensor tendon ruptures such as Vaughn Jackson syndrome, which is an attritional rupture of the extensor tendons of the ring and small fingers caused by the caput ulnae syndrome seen in rheumatoid arthritis. Extensor tendon ruptures can be distinguished from PIN compression by examining for the tenodesis effect, which is lost in the setting of a rupture. A radial nerve injury proximal to the PIN can also produce a similar clinical picture, however sensation in the distribution of the superficial sensory branch will also be affected in that scenario. Other diagnoses to be considered are Parsonage-Turner (Brachial Plexus Neuritis), cervical radiculopathy and systemic disorders associated with neuropathy. These can often be identified by a careful and detailed physical examination and history.

When evaluating these patients plain radiographs of the elbow are obtained to examine proximal radius anatomy, sequelae of old trauma or occult radial head fractures and dislocations. MRI is recommended when clinical examination is suspicious for soft tissue causes of compression such as ganglion, bursa, or tumour. Electrodiagnostic studies can be used to confirm

the diagnosis. Nerve conduction studies will show decreased conduction velocity within the PIN, but normal conduction along the superficial sensory branch. EMG demonstrates denervation changes in the PIN innervated muscles. Electrodiagnostic studies will also assist in differentiating the aetiology of the PIN injury from a brachial plexitis, proximal radial nerve lesion or cervical radiculopathy.

Non Operative Treatment

When an idiopathic or traumatic aetiology of PIN compression is identified, a trial of splint immobilisation may be attempted if the symptoms are not progressive and there is no underlying mass. The elbow should be splinted in a position of flexion and supination to relieve pressure on the nerve [32]. Dynamic splints with outriggers and rubber bands to assist in finger extension are useful in maintaining motion and preventing contractures. If muscle strength improves after a 6 week trial conservative treatment is continued. If there is no improvement, further evaluation, diagnostic studies and exam are performed. At this point, surgical release or tendon transfer surgery should be discussed.

Surgical Management

Multiple surgical approaches have been described for releasing the radial tunnel. Regardless of the approach used the goal is to visualise the nerve throughout its course, remove any masses, and release the five anatomic structures that may cause compression: (1) Fibrous bands, (2) Recurrent Leash of Henry, (3) ECRB, (4) Arcade of Frohse, (5) Supinator muscle.

Anterior Approach of Henry (Figs. 16.3, 16.4, 16.5, and 16.6)

The incision starts over the lateral aspect of the brachialis proximally, the elbow is crossed with either a zig zag or curvilinear course, and distally the incision continues along the medial border of the brachioradialis. The nerve is found in the interval between the brachialis and brachioradialis proximally, and between the brachioradialis and pronator teres distally. This approach has the advantage being extensile, but it may result in scarring particularly where the elbow is crossed. The lateral antebrachial cutaneous nerve can cross the incision site proximally and can potentially be injured.

Brachioradialis Split, BR/ECRL Interval, ECRL/ECRB Interval (Fig. 16.7)

These three approaches provide a similar exposure and may be used interchangeably. They provide visualisation from the radiocapitellar joint to the distal one half of the supinator and there is very limited extensibility. They should not be used if impingement is suspected at the distal exit from the supinator. Of these the brachioradialis split was the first described. The incision is placed directly over the muscle beginning at the elbow crease and extending distally approximately 6 cm. The muscle is bluntly split, and the nerve is identified in a fat stripe beneath. The two interval based approaches (BR/ECRL, ECRL/ECRB) were described to avoid intramuscular dissection and the potential for hematoma formation. When utilising the BR/ECRL interval the incision is placed over the interval beginning at the elbow crease and extending distally 6 cm. It is easiest to identify the correct plain between the muscles distally and then trace back to the common extensor origin where it is less well defined. A similar tactic can be used for the ECRL/ECRB interval, but this incision is typically begun 4 cm distal to the lateral epicondyle. This approach may be useful for combined tennis elbow procedures.

Posterior Approach (Thompson) (Fig. 16.8)

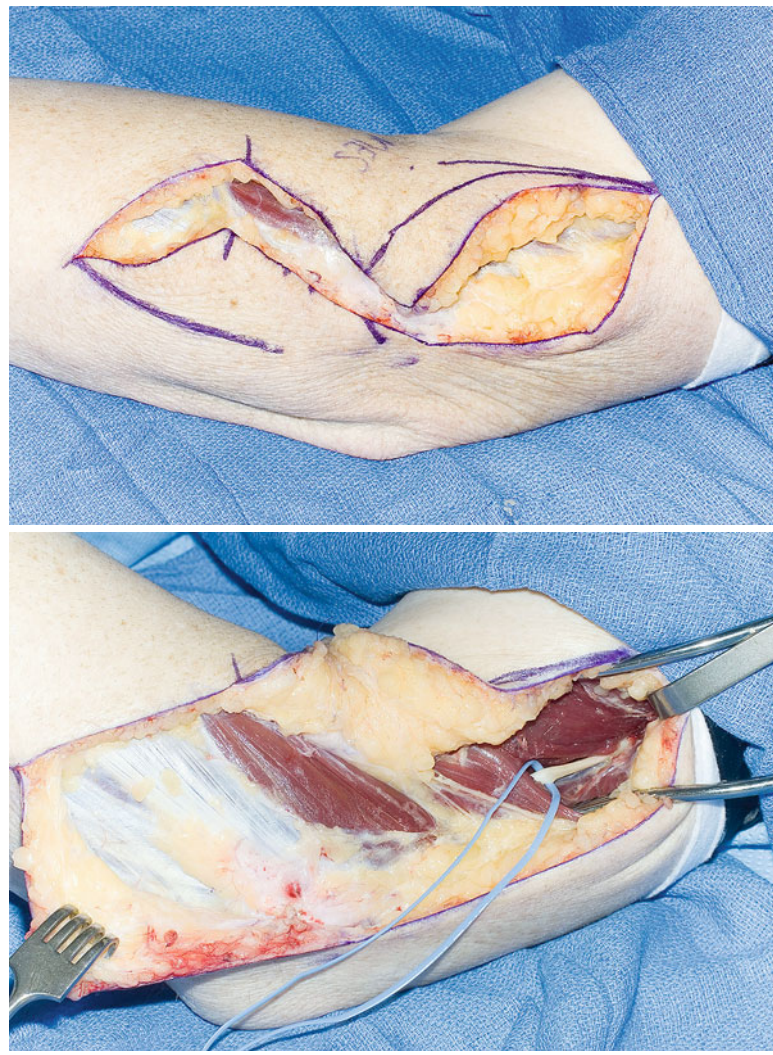
This approach uses the interval between the ECRB and EDC muscles. The incision is placed directly over this interval and extended towards the lateral epicondyle. It provides very good exposure to the distal portion of the supinator but is limited proximally and does not access the radiocapitellar joint.

Selecting an Approach

The anterior approach of Henry provides the broadest exposure and extensile capability. For this reason, it is recommended if mass excision is required or the site of compression is unknown. If surgical treatment of tennis elbow is being performed at the same setting the ECRL/ECRB interval approach provides convenient access for both purposes. Alternatively, an anterior approach may be performed in conjunction with a separate incision over the epicondyle for the tennis elbow release. Finally, surgical approaches can be combined. An anterior incision can be used proximally to identify the radial nerve between the brachialis and brachioradialis and a separate posterior approach can be used distally. By working between these two incisions complete exposure of the nerve can be obtained.

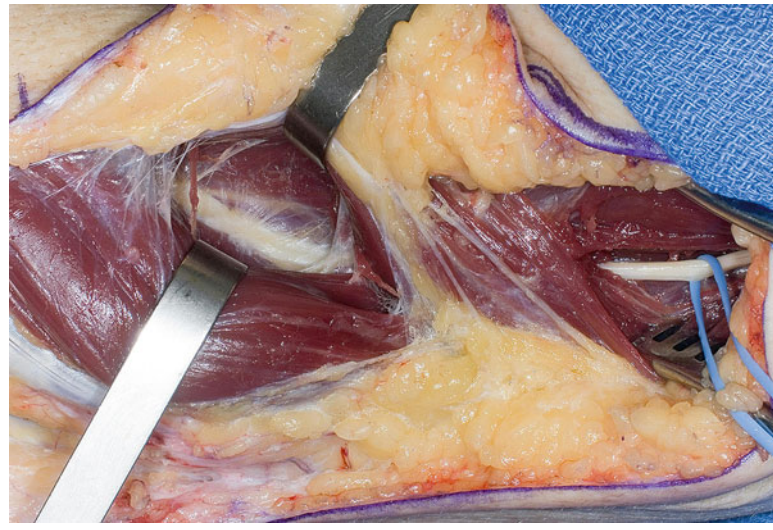
Post Operative Care

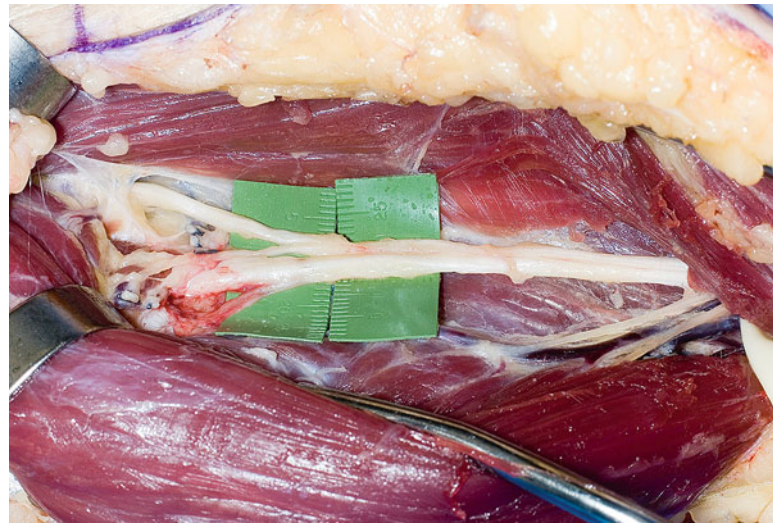
A soft dressing is applied post operatively and active range of motion is allowed as tolerated. Patients are encouraged to perform mobilisation. Lifting is restricted to less than 5 lb initially, but patients are allowed to perform all activities of daily living beginning at 2 weeks. Heavy labour and strenuous exercise is restricted until 6 weeks. If the patient fails to regain strength following



Figs. 16.3, 16.4, 16.5, and 16.6 The anterior approach of Henry. The elbow is crossed in a zig zag fashion, and the incision is placed lateral to the brachialis and medial to brachioradialis. The radial nerve proper is identified between the brachialis and BR proximally and between the BR/ECRL distally. The radial nerve is traced distally releasing the four common compressive structures

Figs. 16.3, 16.4, 16.5, and 16.6 (continued)





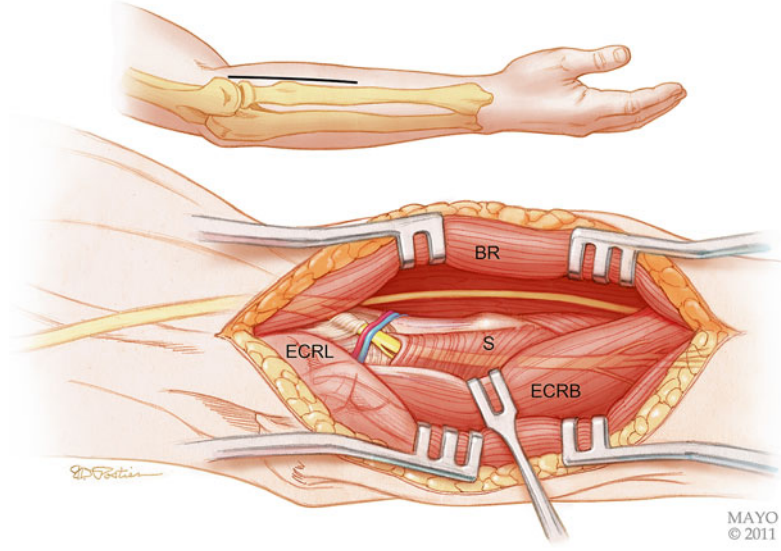


Fig. 16.7 Surgical approach to the radial tunnel through the interval between the brachioradialis and extensor carpi radialis longus muscles achieves exposure to the proximal four sites of possible compression ("Copyright [2011] Mayo Foundation for Medical Education and Research")

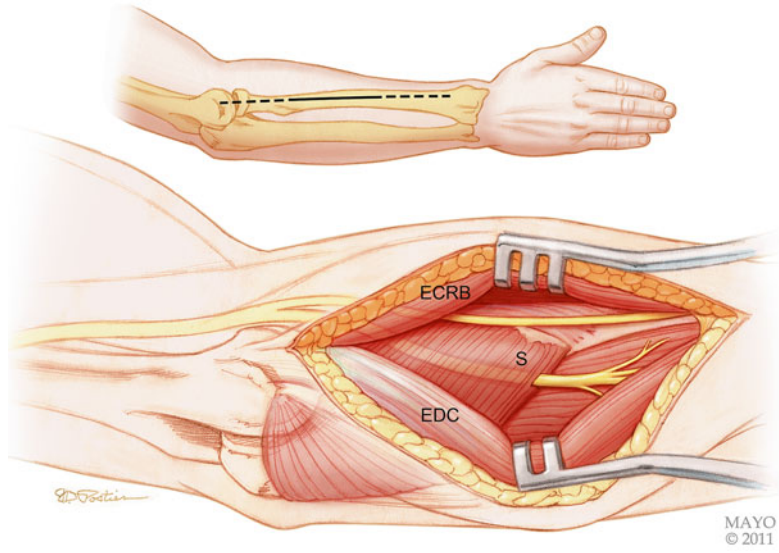


Fig. 16.8 The posterior approach to the posterior interosseus nerve through the interval between the extensor digitorum communis and the extensor carpi radialis brevis muscles provides access to the entire supinator and can extend from the elbow to the wrist ("Copyright [2011] Mayo Foundation for Medical Education and Research")

nerve decompression, tendon transfers can be performed in the future. There are a number of potential surgical options when performing tendon transfers; however a discussion of these is beyond the scope of this chapter.

Radial Tunnel Syndrome

Clinical Evaluation

RTS is characterised by pain along the proximal lateral aspect of the forearm. It is often described as a dull ache or cramping in nature, and tends to be exacerbated by lifting a heavy object, manual labour, and repetitive motion. RTS does not affect motor function but pain inhibition may produce weakness on examination. The clinical presentation is very similar to lateral epicondylitis. Furthermore, these two conditions can coexist. Between 18 and 40 % of patients with RTS will also have tennis elbow, though only 5 % of those with tennis elbow have RTS [13–15, 32]. Additional compressive neuropathies affecting the ulnar or median nerves are present in between 25 and 50 % of patients [13–15, 32].

When considering the diagnosis of RTS it is important to have a systematic and meticulous approach because this disorder does not have a unique character, may be confused with other diagnoses and can be concomitant with other disorders. The point of maximal tenderness is located approximately 5 cm distal to the lateral epicondyle overlying the entrance of the PIN into the supinator [33, 34]. This can be distinguished from tenderness over the epicondyle associated with tennis elbow. Lister described three physical exam findings associated with this disorder: (1) tenderness to palpation over the neck of the radius, (2) pain with resisted extension of the middle finger, (3) pain with resisted supination of the extended arm [12]. He reported that the latter two were pathognomonic for RTS but not 100 % sensitive. Subsequent reports have found this test to be less reliable, but still useful. Mechanistically, resisted extension of the middle finger should cause the ECRB to tighten due to its insertion on

Clinical Pearl – Clinical Signs of Radial Tunnel Syndrome (RTS)

Tenderness over neck of radius (5 cm distal to lateral epicondyle)

Pain with resisted extension of middle finger

Pain with resisted extension of (extended) arm

the third metacarpal, resulting in compression of the PIN beneath the fascial origin of this muscle. Resisted supination may produce dynamic compression by the supinator, but elbow extension is important to eliminate the biceps.

Some authors have advocated for selective injection of steroids and anaesthetics into the radial tunnel and lateral epicondyle to determine which is the primary pain generator [31]. At present there is no diagnostic test to verify or falsify the diagnosis of RTS. Electrodiagnostic studies are not routinely used, and are normal in the majority of reported patients. Techniques involving dynamic supination, and testing with the forearm in various positions of rotation have been reported to be more accurate [35]. However, at present there is no consensus on a technique or diagnostic threshold which produces reliable results [36]. MRI has recently been offered as a new modality to diagnose RTS. Ferdinand et al. found denervation change in 50 % of patients with RTS and none of the asymptomatic controls [37]. Further investigation regarding MRI and RTS will be needed to determine the efficacy of this modality.

Treatment

A trial of non operative management should be attempted before proceeding to surgery. Patients can be splinted with the elbow flexed, forearm supinated and wrist extended to take pressure off the nerve. A trial of anti inflammatory medications may be instituted or an injection of corticosteroids into the radial tunnel may be tried. Activity modification is essential in non operative management and should include avoidance of heavy lifting or repetitive motion. Therapy such as stretching of the extensor musculature may also be tried, as well as modalities such as ultrasound therapy.

If non operative management fails to improve symptoms a surgical release should be considered. The operative approach is the same as previously described for the treatment of PIN compression syndrome. Unfortunately, the outcome of surgical release is somewhat unpredictable. Initial publications reported a very high success rate for surgical release of the radial tunnel. Lister had good results in 95 % of patients, and multiple other authors reported similar success during this time period [12, 38]. However, more recent publications on this topic are less optimistic, with good to excellent results in 10–65 % of patients [13–15]. Also, poor prognostic indicators are concomitant tennis elbow, concomitant compressive neuropathies, and workmen's compensation status [14, 15, 30].

Entrapment of the Superficial Sensory Branch

Overview

Wartenberg is credited with first describing an entrapment syndrome of the superficial sensory branch of the radial nerve in 1932 [39]. He termed this condition "cheiralgia paresthetica" though most subsequent authors have referred to it by his name, Wartenberg's syndrome. The most common site of nerve compression is at Wartenberg's point, where the nerve emerges from beneath the brachioradialis muscle and pierces the fascia connecting this muscle with the ECRL (Fig. 16.9). This site is located between 7 and 10.8 cm proximal to the radial styloid [41]. There are a few plausible mechanisms for entrapment at this position (Fig. 16.10). First, the nerve may be pinched between the tendons of the BR and ECRL during repetitive pronation/supination [42]. During pronation these tendon converge, and the ECRL may actually pass beneath the BR. As a result the nerve can be pinched by this scissoring mechanism. Second, a narrow opening at Wartenberg's point may compress the nerve or cause it be stretched. Longitudinal excursion of the nerve occurs during wrist flexion and ulnar deviation [42]. This requires a smooth gliding pathway. If the nerve is tethered anywhere, such as at Wartenberg's point, undue stretch may occur.

SBRN entrapment can occur at other points along its course such as near the radial styloid.

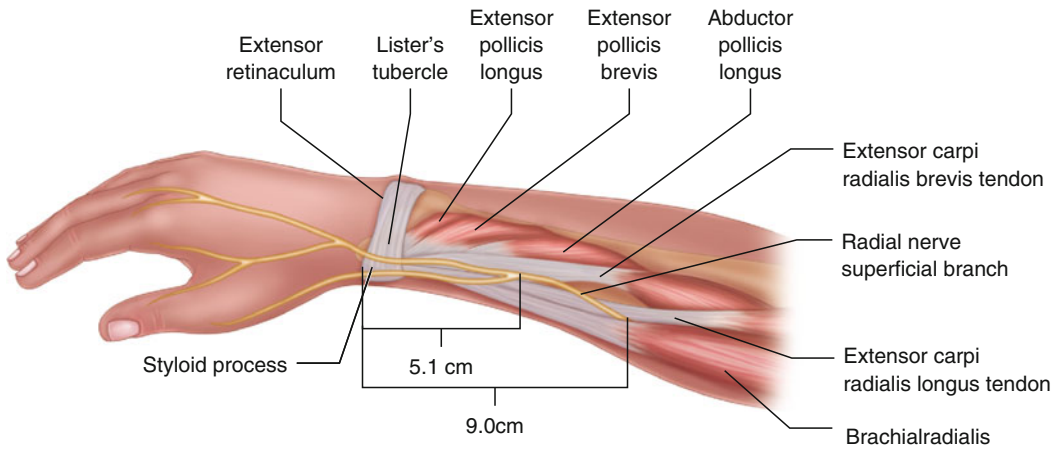


Fig. 16.9 The usual position of the superficial branch of the radial nerve is shown. None of the branches are ulnar to Lister's Tubercle (Reprinted with permission. This article was published in Abrams et al. [41], Copyright Elsevier)

These usually result from oedema or scarring secondary to trauma, wrist bands, implanted Kirschner wires or associated conditions such as DeQuervian's. It is important to clinically differentiate between the classic compression at Wartenberg's point and more distal entrapment sites since these entities have different prognoses.

Clinical Evaluation

Patients typically complain of pain and altered sensation on the dorsoradial aspect of the hand in the distribution of the superficial branch of radial nerve. Often this will be associated with a minor trauma, a wrist watch, or work related repetitive motion. On physical examination, moving two point discrimination and vibratory stimuli can be used for objective documentation. Dellon and MacKinnon reported that all of their patients had abnormal findings in the SBR distribution on these exams [40]. A Tinel's sign can often be elicited and is helpful for defining the zone of entrapment, whether it be at Wartenberg's point or more distal. Lanzetta et al. organized patients into four categories based on the zone of entrapment: Zone 1- classic site between BR/ECRL 8 cm proximal to the styloid, Zone 2- level of wrist watch, Zone 3-level of the first compartment, Zone 4-distal to zone 3 [43].

Symptoms may be evoked by exam maneuvers which place the nerve on stretch such as wrist flexion and ulnar deviation. A positive test on this exam can be confused with De Quervains and is often referred to as a false Finkelstein [44]. However, patients with tenosynovitis of the first extensor compartment should also have focal tenderness over those tendons. Other provocative maneuvers include repetitive pronation/supination, and the pronation-ulnar flexion sign (combined maximum pronation and ulnar flexion).

The differential diagnosis includes De Quervains, cervical spine radiculopathy, a contusion to the SBRN and neuritis of the lateral antebrachial cutaneous nerve (LABC). The LABC and SBRN have a sensory overlap in 75 % of individuals [45]. If LABC neuritis is being considered, selective injections can help determine the pain source. The LABC can be injected in the proximal forearm adjacent to the cephalic vein.

Clinical Pearl: Clinical Features of Wartenberg's Syndrome

Altered sensation/pain dorsoradial aspect hand

Positive Tinel's sign

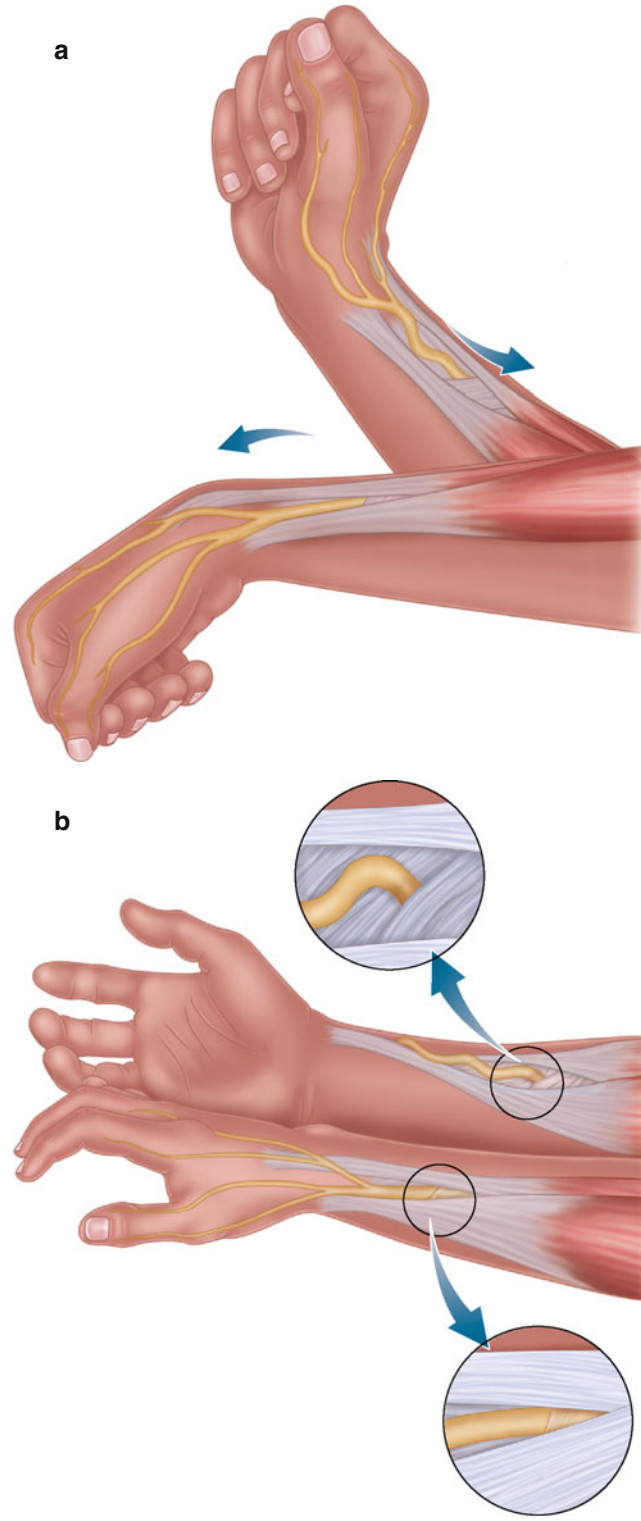


Fig. 16.10 (**a**) With the forearm supinated, the SBR lies beneath the fascia, but without compression between the tendons of the BR and ECRL. When the forearm pronates, the ECRL crosses beneath the BR tendon, creating a scissoring or pinching of the SBR. (**b**) Palmar ulnar flexion of the wrist puts maximum traction on the nerve. Repeated mechanical trauma causes swelling of the SBR deep to the fascia restricting the nerve's normal ability to slide distally during wrist ulnar flexion, resulting in the symptoms of traction pain (Reprinted with permission. This article was published in Dellon and Mackinnon [40], Copyright Elsevier)

Treatment

Conservative management includes a course of anti inflammatory medication as well as therapy modalities to decrease edema. Resting splints can also be attempted and these should place the forearm in supination to prevent the nerve from being pinched between BR/ECRL. If recognised and treated early, conservative management is often successful.

If non operative treatment is unsuccessful surgical management can be considered. The surgical approach should be based on the site of entrapment as determined by physical examination. When performing a release at Wartenberg's point, the incision is centered over this site, or placed just volar to it so that the surgical scar does not lie over the released nerve. The deep fascia connecting the BR and ECRL is identified as well as the SBRN. The fascia is then released both proximaly and distally so that the nerve has a tension free bed. If the entrapment is located in one of the more distal zones, the nerve is explored and neurolysis performed as needed. In revision surgeries where the SBRN may have been previously injured, the SBRN neuroma can be excised and the proximal end implanted in muscle. It is important to inform the patient that whilst such treatment of SBRN neuromas may be beneficial, there is a high incidence of recurrence.

The surgical results depend on the location of entrapment [43]. If nerve compression occurs at Wartenberg's point there is a more favorable prognosis, and good results have been reported in 86 % of these patients following release [40]. On the other hand, entrapment at more distal sites has a less predictable treatment course. Calfee et al. reported improvement in only 54 % of such patients following exploration and neurolysis [46].

Summary

Compressive neuropathies of the radial nerve can be difficult to diagnose and the results of treatment may be unpredictable. In part this is because they are less common and therefore less familiar to hand surgeons. But difficulty also arises because these entities are easily confused with other diagnoses and can present in combination with other neuropathies or tendinopathies. This is epitomised by RTS which is so vague and difficult to pinpoint that some have questioned its existence, while others have attributed it to lateral epicondylitis.

A systematic approach to diagnosis and treatment will maximize clinical results. The physical examination can help localise the site of entrapment, and should also be used to assess for other concomitant diagnoses such as "tennis elbow", DeQuervians or other compressive neuropathies. Imaging modalities may be useful when unusual causes of compression are suspected such as tumours or radial head trauma. EMG is useful in diagnosing PIN compression syndrome but has limited utility in RTS or entrapment of the SBRN. Finally, selective injection can help confirm a diagnosis or elicit the relative contribution of concomitant pathologies.

A first line conservative treatment should always be attempted except in instances where there is progressive motor loss or a compressive mass. Splints, injections, therapy and other modalities may be tried though there is no evidence to support any one in particular. If surgical decompression of the PIN or SBRN is performed it is imperative the surgeon have a thorough understanding of the regional anatomy to ensure that all potential sources of compression are released. There are multiple potential surgical approaches; each has relative merits that may lend itself better to a particular clinical situation. The results of decompression are less predictable than what is experienced with compressive neuropathies of the median and ulnar nerve, and revision surgery is even more unpredictable. The patient should be made aware of this preoperatively to ensure realistic expectations.

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Index

A
Abductor pollicus longus (APL), 36, 49, 71, 163, 285
Allen's test, 249, 269, 276
Anterior interosseous nerve syndrome (AINS)
  aetiology, 236–237
  clinical presentation, 235
  differential diagnosis, 235
  treatment outcomes, 239–240
Avascular necrosis (AVN), 116, 153, 155, 261

B
Ballottement test, 187, 202
Boutonnière deformity. *See* Chronic zone III injury
Brachial plexus (BP)
  Horner's sign, 7
  imaging studies, 7
  lamina test, 7
  preoperative chart, 6

C
Carpal instability
  dyskinematics, 179
  instability patterns, 179–180
  LTq (*see* Lunotriquetral dissociation (LTq))
  osteoarthritis, 179
  SLD (*see* Scapholunate dissociation (SLD))
  wrist dysfunction, 179
Carpal instability adaptive (CIA), 179
Carpal instability complex (CIC), 179
Carpal instability dissociative (CID), 179
Carpal instability non-dissociative (CIND), 179
Carpal tunnel syndrome (CTS)
  acute, 219
  aetiology, 217–219
  chronic compression, 218
  clinical diagnosis, 217
  complications, 227
  differential diagnosis, 234
  endoscopic carpal tunnel release, 226
  open CTR
    Camitz procedure, 225
    Kaplan's cardinal line, 224
    Macdonald's dissector, 225
    nerve damage, 224
    post-operative regime, 225–226
    safe zone, 224
    skin closure, 225
  'O' sign, 219–221
  patient's history and clinical findings, 219
  provocation tests
    Phalen's test, 221, 222
    pronator provocation, 221, 222
    Tinel's sign, 219–221
  recalcitrant and recurrent cases, 227–228
  sensation, 219, 220
  splinting, 222–223
  steroid injection, 223
  surgery, 223
  symptoms, 219
  treatment outcomes, 227
Carpometacarpal joint (CMCJ)
  anatomy, 162
  APL and flexor carpi radialis, 163
  clinical outcomes, 164
  dorsal ligaments, 162
  etiological factor, 162
  extension osteotomy, 163
  extensor carpi radialis longus, 163
  hyperextension, 163
  joint degeneration, 162
  Kirschner wire, 163
  ligament reconstruction, 163
  metacarpal osteotomy, 163
  MRI, 163
  palmaris longus, 163
  primary and secondary instability, 162
  radiographic and arthroscopy, 163
  signs and symptoms, 162
  Wagner approach, 163
Central slip tenotomy, 59–60
Chronic instability
  finger (*see* Finger instability)
  RCL, 167, 169
  thumb instability
    CMCJ (*see* Carpometacarpal joint (CMCJ))
    distal interphalangeal joint, 177–178
    MCPJ, 164
    volar instability, 167, 169

I.A. Trail, A.N.M. Fleming (eds.), *Disorders of the Hand: Volume 2: Hand Reconstruction and Nerve Compression*, DOI 10.1007/978-1-4471-6560-6,

Chronic instability (*cont.*)
UCL
adductor pollicis/aponeurosis, 165, 166
contraindication, 167
gamekeeper's thumb, 164
MCPJ stabilisation, 167, 168
physical examination, 165–167
radiographs, 165
Skier's thumb reflect, 164
Stener lesion, 164, 165
tendon graft, 165
Chronic mallet deformity
aetiology, 58
central slip tenotomy, 59–60
clinical assessment, 58
double breast technique, 60
ORL reconstruction, 60
surgical options, 58–59
tendon plication, 59
tenodermodesis/dermatotenodesis, 59
treatment outcomes, 60–62
Chronic zone III injury
aetiology, 63
central slip re-attachment, 64
central slip reconstruction, 65
clinical assessment, 63
Curtis' staged reconstruction, 65
free tendon graft reconstruction, 65
Littler-Eaton technique, 64
Matev procedure, 65
scar excision, 64
surgical options, 63–64
terminal tendon tenotomy, 64
treatment outcomes, 65–66
Chronic zone 6, 7, and 8 injuries
aetiology, 69
clinical assessment, 69
free tendon interposition graft, 69–70
tendon buddying, 70
treatment options, 69
treatment outcomes, 70
Clawing, 42
anti-claw procedure, 44
Bouviere's test, 44
dynamic operations, 45, 46
knuckleduster splint, 44
proximal interphalangeal joint flexion, 44
static operations, 45
Zancolli Lasso procedure, 46, 47
Compression neuropathies
CTS (*see* Carpal tunnel syndrome (CTS))
PIN (*see* Posterior interosseus nerve (PIN))
RTS
clinical evaluation, 290–291
non operative management, 291
SBRN
clinical evaluation, 292
conservative management, 294
entrapment, 291
nerve compression site, 291, 294
scissoring mechanism, 291, 293
Wartenberg's syndrome, 291
ulnar nerve
elbow (*see* Elbow)
wrist (*see* Ulnar tunnel syndrome (UTS))
CTS. *See* Carpal tunnel syndrome (CTS)
Curtis' staged reconstruction, 65

D
Derby test, 187
Distal radio-ulna joint (DRUJ)
capitate neck fractures, 200
capsular and ligamentous laxity, 200
congenital abnormalities, 200, 204, 205
differential diagnosis, 200
distal radial fractures, 200, 201
evaluation, 202–204
fracture
dorsal and palmar subluxation, 208
Essex-Lopresti injury, 207, 208
Galleazzi fracture, 206, 207
interosseous membrane reconstruction, 208, 210
Monteggia fracture, 206, 207
pre-operative planning, 207–209
sigmoid notch osteotomy, 211
single bone forearm, 208, 210
TFCC insertion, 208, 210
ulna styloid fractures, 208
functional anatomy, 196–197
Keinböck's disease, 202
kinematics and biomechanics
dry bone articulation, 198
forearm rotation, 197, 199
tensioning and de-tensioning, 197, 198
ulno-carpal ligaments and capsule, 199
Lichtman and Collins classification, 199
Madelungs deformity, 199, 200
piano key sign, 202
press test, 202
rheumatoid arthritis, 200, 204–206
sigmoid notch fractures, 200
soft tissue injury, 211–213
stability, 195
Distal radius malunion
dorsal angulated malunion (*see* Dorsal angulated malunion)
indications, 131–132
intraarticular malunion, 143
pathomechanics
carpal zigzag pattern, 130
DISI type pattern, 131
dorsal slide, 130
midcarpal and distal radio-ulnar joint, 129–130
physiologic volar tilt, 130
radiocarpal joint, 129
surgical outcomes, 148
ulnar side procedures
distal radio-ulnar joint instability, 145
elongated styloid, 145, 146

joint incongruity/degeneration, 146, 148–149
TFC detachment technique, 144
TFC reattachment technique, 146–147
trampoline effect and positive hook test, 144, 145
ulnar head impaction symptoms, 144–145
volar angulated malunion
osteotomy, 141
sagittal malrotation, 141–142
Smith's type malunion, 141
ulna and volar sagging, 140
Dorsal angulated malunion
Fernandez's method, 132, 134
opening wedge osteotomy
correction angle, 134, 135
disadvantages, 134
FCR sheath, 135
Lanz's technique, 134
osteotomy, 135, 136
soft tissue procedures, 135–138
synovectomy, 135–138
ulnar styloid excision, 135–138
sagittal malrotation, 132, 133
Wada's osteotomy, 138–140
Dorsal radio-ulna ligament (DRUL), 196–199, 212
Double crush syndrome (DCS) theory, 236
DRUJ. *See* Distal radio-ulna joint (DRUJ)
Duchenne's sign, 270
Durkan test, 232

E
Elbow
anatomy, 243–244
arcuate/Osborne's ligament, 244
blood supply, 244
compression sites
anconeus epitrochlearis, 245, 247
Arcade of Struthers, 245
cubitus valgus deformity, 245, 246
intermuscular septum, 245
medial epicondyle and epicondylar groove, 245
Osborne's ligament and fascia, 247
space occupying lesions, 245, 247
cubital tunnel retinaculum, 243, 244
diagnosis
acromegalic ulnar nerve, 249
atrophy, 250
crossed fingers test, 250
double crush phenomenon, 249–250, 252
elbow flexion test, 251
hypoesthesia, 250
intrinsic minus posture/ulnar clawing, 251
Masse's sign, 251
thoracic outlet syndrome, 249
Tinel's test, 250
ulnar tunnel syndrome, 249
failed operative treatment
MABCN, 260
subcutaneous transposition, 260, 261
submuscular transposition, 260
MABCN, 244–245
non-operative treatment, 252–253
operative treatment, 253–254
pathology, 248
provocative tests, 243
revision surgery, 261
staging, 252
surgical technique
anterior subcutaneous transposition, 256–257
endoscopic cubital tunnel release, 259
intramuscular transposition, 257–258
medial epicondylectomy, 258–259
in situ decompression, 255–256
submuscular transposition, 258
Extensor carpi ulnaris (ECU), 188, 200, 285
Extensor compartment artery (ECA), 154
Extensor digiti minimi (EDM), 285
Extensor digitorum communis (EDC), 68, 82, 285
Extensor indicis proprius (EIP), 285
Extensor pollicus brevis (EPB), 285
Extensor pollicus longus (EPL) reconstruction, 285
aetiology, 70
APL to EPL transfer, 71
clinical assessment, 70–71
EIP to EPL transfer, 71
free tendon interposition graft, 71–72
surgical options, 71
treatment outcomes, 72
Extensor tendon reconstruction
chronic mallet deformity
aetiology, 58
central slip tenotomy, 59–60
clinical assessment, 58
double breast technique, 60
ORL reconstruction, 60
surgical options, 58–59
tendon plication, 59
tenodermodesis/dermatotenodesis, 59
treatment outcomes, 60–62
chronic zone III injury
aetiology, 63
central slip re-attachment, 64
central slip reconstruction, 65
clinical assessment, 63
Curtis' staged reconstruction, 65
free tendon graft reconstruction, 65
Littler-Eaton technique, 64
Matev procedure, 65
scar excision, 64
surgical options, 63–64
terminal tendon tenotomy, 64
treatment outcomes, 65–66
chronic zone 6, 7, and 8 injuries
aetiology, 69
clinical assessment, 69
free tendon interposition graft, 69–70
reconstructive procedures, 69
tendon buddying, 70
treatment outcomes, 70

Extensor tendon reconstruction (*cont.*)
EPL reconstruction
aetiology, 70
APL to EPL transfer, 71
clinical assessment, 70–71
EIP to EPL transfer, 71
free tendon interposition graft, 71–72
surgical options, 71
treatment outcomes, 72
sagittal band reconstruction
aetiology, 66–67
clinical assessment, 67
EDC tendon slip, 68
junctura tendinum, 68
pulley construction, 68
repair/imbrication, 67
scar excision, 67
soft tissue procedures, 67
treatment outcomes, 68
Extra-articular metacarpal malunions
angulatory deformity
clinical manifestation, 100
surgical techniques, 102–103
metacarpal shortening
clinical manifestation, 100
surgical techniques, 103
rotational malunion
clinical manifestation, 100
surgical techniques, 102

F
Finger instability
interphalangeal joints
dorsal chronic instability, 172, 174–177
hyperextension deformity, 173–174, 177
lateral chronic instability, 174–175
ligaments, 171–172
posterior-anterior and radiographs, 172, 174
volar chronic instability, 175, 177
MCPJ
arthroscopy, 170
clinical testing, 170
conservative treatment, 170–171
gross instability/deviation, 170–171
ligament avulsion and degenerative changes, 170
radial collateral ligament, 170, 171
ultrasound and MRI, 170
Flexor carpi radialis sheath (FCR), 134, 135
Flexor carpi ulnaris (FCU), 188
Flexor digitorum profundus (FDP), 232, 244
Flexor tendon reconstruction
in children, 93
donor grafts, 93–94
one-stage, 79–80
primary repair, 75
pulley reconstruction, 76
tenolysis
adhesions, 76
complications, 78–79
knife, 77
local anesthesia, 77
MRI, 76
outcomes, 78
patient selection, 76
postoperative immobilization, 77
range of motion (ROM), 76, 77
step by step technique, 77
steroids deposition, 78
traction flexor check, 77–78
ultrasonography, 76
thumb injury, 92–93
two-stage, 79–80
zone I injuries (*see* Zone I flexor tendon reconstruction)
zone II injuries (*see* Zone II flexor tendon reconstruction)
zone III injuries (*see* Zone III flexor tendon reconstruction)
zone IV injuries (*see* Zone IV flexor tendon reconstruction)
Froment's sign, 250, 270

H
Hereditary liability to pressure paralysis (HLPP), 218–219

I
In situ decompression
complications, 256
contraindications, 255
curvilinear incision, 254
lax, 255, 256
nerve proximally and distally, 254–255
post-operative management, 255
1,2 intercompartmental supraretinacular artery (ICSRA), 122, 125, 154
Intercostal nerve (ICN) transfer, 22, 24
Interposition nerve grafting, 11, 12, 21, 27
Intra-articular metacarpal malunion
classification, 100
surgical techniques, 103

J
Jeanne's sign, 43, 269

K
Kienböck's disease, 155–156
Kiloh-Nevin syndrome, 231
Kleinert/Weilby method, 89, 90

L
Littler-Eaton technique, 64
Lunotriquetral dissociation (LTq)
ballottement test, 187
Derby test, 187
Gilula's line 1, 187

SL gap and positive ring sign, 187
treatment
complex spiral tenodesis, 188
ECU and FCU, 188
3LT, 188
Mayo Clinic ligament reconstruction, 188, 189
radiolunate fusion/midcarpal, 188
spiral tendon reconstruction, 188, 191
Wrightington method, 188, 190

M
Malunions
definition, 97
metacarpal (*see* Metacarpal malunions)
phalanges (*see* Phalanges malunions)
Masse's sign, 251
Matev procedure, 65
Mayo Clinic ligament reconstruction, 188, 189
Medial antebrachial cutaneous nerve (MABCN), 244–245, 254, 260
Medial femoral condyle (MFC), 119, 125, 126
Median nerve entrapment forearm
anatomy, 231–232
anterior interosseous syndrome
aetiology, 236–237
clinical presentation, 235
differential diagnosis, 235
treatment outcomes, 239–240
complications, 240
history, 231
non-operative management, 237–238
operative technique, 238
physical examination, 232–233
pronator syndrome
aetiology, 236–237
clinical presentation, 233
differential diagnosis, 234–325
treatment outcomes, 239
Median nerve palsy
anatomy, 50
clinical assessment, 50–52
high median nerve palsy, 53
low median nerve palsy, 50–53
Metacarpal malunions
extra-articular
classification, 100
surgical techniques, 102–103
intra-articular
classification, 100
surgical techniques, 103
Metacarpophalangeal joint (MCPJ)
finger instability
arthroscopy, 170
clinical testing, 170
conservative treatment, 170–171
gross instability/deviation, 170–171
ligament avulsion and degenerative changes, 170
radial collateral ligament, 170, 171
ultrasound and MRI, 170
thumb instability, 164
Midcarpal instability, 186–187

N
Nerve conduction studies (NCS), 219
Nerve grafting
donor-nerve characteristics, 11
gold standard, 11
interposition nerve grafting, 11, 12
intraneural topography, 11
proximal and distal nerve stumps, 11
saphenous nerve, 19
sural nerve, 19, 20
ulnar nerve, 17, 19
vascularized nerve graft
blood supply, 11
clinical indication, 13
cross chest graft, 13, 17
example of, 13–16
Terzis technique, 13, 17
ulnar nerve graft, 13, 17
vascularized fascia, 17, 18
Nerve transfer, 19
Neurogenic thoracic outlet syndrome, 234
Non-unions
complications, 111
definition, 97
and delayed unions, 99
metacarpal
aetiology, 98
surgical techniques, 107–109
treatment outcomes, 109–110
open reduction and fixation, 99
phalanges
aetiology, 97–98
surgical techniques, 107–109
treatment outcomes, 110
wait and see policy, 99
Non-vascularized bone graft
cortical part removal, 120, 121
DISI deformity, 120, 122
Herbert screw, 120, 123
hump back/flexion deformity, 116, 118–120, 122
mid-third and dorsal approach, 119, 120
revision surgery, 120, 124

O
Oblique retinacular ligament (ORL) reconstruction, 60
Okutsu triple loop technique, 89
Open carpal tunnel release (CTR)
Camitz procedure, 225
Kaplan's cardinal line, 224
Macdonald's dissector, 225
nerve damage, 224
post-operative regime, 225–226
safe zone, 224
skin closure, 225

P
Palmar radio-ulnar ligament (PRUL), 196
Parsonage-Turner syndrome, 234
Peripheral nerve injury
  aetiology, 4
  angiography, 7, 8
  axonotmesis, 5
  BP injuries
    Horner's sign, 7
    imaging studies, 7
    lamina test, 7
    preoperative chart, 6
  compound action potential (CAP), 21
  four roots avulsion, 22
  global avulsion, 22
  intraoperative electrophysiologic recordings, 21
  microsurgical management, 8
  neurapraxia, 5
  neurotmesis, 5
  one/two root avulsions, 22
  pathophysiology, 4–5
  preoperative electrophysiological study, 21
  Preston dynamometer set, 6
  repair
    carbonic anhydrase staining, 10
    complications, 28
    end-to-end-repair, 10
    end-to-side nerve repair, 10–11
    epineurial repair, 9
    intraoperative diagnostic tool, 9
    nerve end coaptation, 9
    nerve grafting (*see* Nerve grafting)
    nerve transfer, 19
    rehabilitation, 25–26
    tensionless repairs, 9
    timing, 8–9
  sensory evaluation, 6
  severity score, 21
  surgical techniques
    C5 root transfer, 23
    C7 root transfer, 23
    ICN transfer, 24
    phrenic nerve transfer, 24–25
    ulnar-to-musculocutaneous nerve transfer, 25
    XI transfer, 23–24
  three roots avulsion, 22
  Tinel's sign, 5
  treatment outcomes, 26–28
Phalanges malunions
  angulatory deformity
    clinical manifestation, 101
    surgical techniques, 104–106
  intra-articular malunion
    anatomic reduction, 106
    clinical manifestation, 101–102
    closing wedge osteotomy, 107
    condylar advancement osteotomy, 107
    dental pick, 106
    midlateral incision, 106
    Swanson implant, 107, 109
    thin oscillating saw/osteotome, 107, 108
  phalangeal shortening
    clinical manifestation, 102
    surgical techniques, 107
  rotational malunion
    clinical manifestation, 101
    surgical techniques, 104
Phalen test, 221–222, 232, 234, 251
Piano key sign, 202, 204
Posterior interosseus nerve (PIN)
  Arcade of Frohs, 284–286
  causes, 286
  clinical evaluation, 286–287
  compression sites, 284
  ECRB origin, 286
  fibrous bands, 286
  non operative treatment, 287
  post operative care, 288, 290
  recurrent Leash of Henry, 286
  superficial and deep motor branches, 284
  supinator muscle, 286
  surgical management
    anterior approach of Henry, 287–289
    brachioradialis split, 287, 289
    BR/ECRL interval, 287, 289
    ECRL/ECRB interval, 287, 289
    posterior approach of Thompson, 287, 290
Preiser's disease, 155–157
Pronator syndrome (PS)
  aetiology, 236–237
  clinical presentation, 233
  differential diagnosis, 234–325
  treatment outcomes, 239
Proximal interphalangeal joints (PIPJ)
  dorsal chronic instability, 172, 174–177
  hyperextension deformity, 173–174, 177
  lateral chronic instability, 174–175
  ligaments, 171–172
  posterior-anterior and radiographs, 172, 174
  volar chronic instability, 175, 177

R
Radial collateral (RCL) instability, 167
Radial nerve palsy
  anatomy, 36
  extensor function, 36, 37
  high radial nerve palsy, 36–38
  low radial nerve palsy, 38
  marked radial deviation, 36, 37
  surgical techniques
    C-shaped incision, 39
    FCR, 38, 39
    Henry approach, 38
    Lasso technique, 40
    spiral technique, 40
    S-shaped curvy linear incision, 38, 39
    timing, 40–41
    transverse incision, 38
Radial tunnel syndrome (RTS)
  clinical evaluation, 290–291
  non operative management, 291

Reduction-Association of the SL Joint (RASL), 183, 186
Rheumatoid arthritis (RA), 70, 204, 205

S
Sagittal band reconstruction
  aetiology, 66–67
  clinical assessment, 67
  EDC tendon slip, 68
  junctura tendinum, 68
  pulley construction, 68
  repair/imbrication, 67
  scar excision, 67
  soft tissue procedures, 67
  treatment outcomes, 68
Scaphoid non union
  avascular bone, 116, 118
  avascular necrosis, 118–119
  bony union, 115
  clinical results, 126
  diagnosis, 115
  Herbert modified classification, 116, 117
  non-vascularized bone graft
    cortical part removal, 120, 121
    DISI deformity, 120, 122
    Herbert screw, 120, 123
    hump back/flexion deformity, 116, 118–120, 122
    mid-third and dorsal approach, 119, 120
    revision surgery, 120, 124
  postoperative treatment, 126
  SNAC wrist pattern, 115
  stability and blood supply, 119
  T1-weighted image, 116, 117
  variables, 119
  vascularized bone graft
    headless bone screw, 125
    ICSRA, 122, 125
    proximal pole nonunion, 116, 122, 123
    standard palmar approach, 124
Scaphoid shift test, 180
Scapho-lunate advanced collapse (SLAC), 183, 185
Scapholunate dissociation (SLD)
  increased SL angle, 181
  scaphoid ring sign, 181
  scaphoid shift test, 180
  tendon reconstruction
    bone-ligament-bone grafts, 186
    3LT method, 184–186
    radio-scapho-lunate fusion plus distal scaphoidectomy, 186–187
    RASL, 186
    scaphotrapezoid-trapezial arthrodesis, 186
  Terry Thomas sign, 181
  treatment
    carpal malalignment, 182
    cartilage, 182
    complete SL ligament injury, 183
    dorsal capsulodesis, 182–183
    dorsal SL ligament, 182
    four corner/lunocapitate fusion, 183
    intracarpal fusion, 183
    mid-substance ligament rupture, 182
    reconstruction, 183
    secondary scaphoid stabilizers, 182
Silicone rod
  chronic zone 6, 7, and 8 injuries, 70
  zone I flexor tendon reconstruction, 83, 84
  zone II flexor tendon reconstruction, 87, 88
Spike osteotomy, 104
Spinal accessory nerve (XI) transfer, 23–24
Spurling's sign, 268
Static and dynamic stabilisers, 196
Superficial sensory branch of radial nerve (SBRN)
  clinical evaluation, 292
  conservative management, 294
  entrapment, 291
  nerve compression site, 291, 294
  scissoring mechanism, 291, 293
  Wartenberg's syndrome, 291
Synergism, 35

T
Tendon transfers
  definitive transfer, 34
  donor excursion, 34
  donor strength, 34
  energy loss minimisation, 35
  insertion points, 35
  median nerve palsy (*see* Median nerve palsy)
  radial nerve palsy (*see* Radial nerve palsy)
  surgical algorithm, 35–36
  synergism, 35
  tissue equilibrium, 34
  ulnar nerve palsy (*see* Ulnar nerve palsy)
  wrist flexors and extensors, 35
Terry Thomas sign, 181
Terzis technique, 13, 17
Three-ligament tenodesis (3LT) method, 184–186
Tinel's sign, 5, 7, 219–221, 234, 270
Tinel test, 232
Tissue equilibrium, 34

U
Ulnar collateral (UCL) instability, 164–165
Ulnar nerve palsy
  anatomy, 41
  clawing, 42
    anti-claw procedure, 44
    Bouviere's test, 44
    dynamic operations, 45, 46
    knuckleduster splint, 44
    proximal interphalangeal joint flexion, 44
    static operations, 45
    Zancolli Lasso procedure, 46, 47
  Froment's sign, 43
  Jeanne's sign, 43
  lateral key pinch, 42–43, 49
  power grip, 42, 47–48
  sensory loss, 42
  unbalanced flexion, 42
  Wartenberg's sign, 43

Ulnar paradox, 42
Ulnar tunnel syndrome (UTS)
anatomy
hook of hamate, 272
palmar carpal ligament, 272
sensory and motor branches, 272, 273
soft tissue structures, 275
superficial structures, 274
transverse carpal ligament, 272
variations, 272–273
deep surgical dissection, 279
differential diagnosis
"double-crush" phenomenon, 275–276
ganglion cyst, 276, 277
hook of hamate fracture, 277
hypothenar hammer syndrome, 276, 277
ulnar artery aneurysms, 277
ulnar artery thrombosis, 276, 277
electrodiagnostic studies, 271
etiology, 276
history, 267–268
physical examination
Duchenne's sign, 270
Froment's sign, 270
Jeanne's sign, 269
nerve compression, 269
splinter hemorrhages/digital pallor, 269
Spurling's sign, 268
Tinel's sign, 270
ulnar neuropathy, 269
vascular examination, 269
Wartenberg's sign, 269, 270
radiographic studies, 271
superficial surgical dissection, 279
surgical management, 278

V
Vascularized bone graft (VBG)
complications, 158
corticocancellous graft, 155
ECA, 154
headless bone screw, 125
ICSRA, 122, 125
Kienböck's disease, 155–156
local, pedicled/free grafts, 153
outcomes
lunate pathology, 158
scaphoid pathology, 157–158
Preiser's disease, 155
proximal pole nonunion, 116, 122, 123
rehabilitation, 156
scaphoid injury, 154–155
standard palmar approach, 124
surgical techniques, 156
Vascularized nerve graft
blood supply, 11
clinical indication, 13
cross chest graft, 13, 17
example of, 13–16
Terzis technique, 13, 17
ulnar nerve graft, 13, 17
vascularized fascia, 17, 18
VBG. *See* Vascularized bone graft (VBG)
Volar angulated malunion
osteotomy, 141
sagittal malrotation, 141–142
Smith's type malunion, 141
ulna and volar sagging, 140

W
Wartenberg's sign, 250, 269, 270
Wrightington method, 188, 190
Wrist drop. *See* Radial nerve palsy

Z
Zancolli Lasso procedure, 46, 47
Zone I flexor tendon reconstruction
clinical presentation, 80, 81
complications, 85
one stage tendon grafting, 82–83
outcomes, 81, 84–85
primary repair, 81
tenodesis/arthrodesis, 81
two stage tendon grafting, 83–84
Zone II flexor tendon reconstruction
clinical presentation, 80, 81, 85
complications, 90
one-stage tendon grafting, 86–87
outcomes, 89–90
pulley reconstruction, 88–90
two-stage tendon grafting, 87–88
Zone III flexor tendon reconstruction
clinical presentation, 80, 81, 90–91
complications, 91
outcomes, 91
treatment, 91
Zone IV flexor tendon reconstruction
clinical presentation, 80, 81, 90–91
complications, 91
outcome, 91
treatment, 91