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

Disorders of the Hand

Volume 3: Inflammation, Arthritis and Contractures



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

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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!

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Part I

Inflammation and Arthritis

Tendinopathy at the Wrist

Zafar Naqui and Ian A. Trail

Keywords

Tendinopathy • Tenosynovitis • Tendovaginitis • Rheumatoid Arthritis • Gout • Pseudogout • de Quervains's Disease • Finklestein's test • Intersection Syndrome • Extensor Carpi Ulnaris • Extensor Pollicis Longus • Flexor Carpi Ulnaris • Amyloidosis • Sarcoidosis

Introduction

Twelve extensor and 11 flexor tendons cross the wrist. Disorders afflicting these tendons as they traverse the wrist are a frequent cause of symptoms and make up a significant proportion of the workload of the hand surgeon. Broadly, tendons may be affected by either tenosynovitis or, more commonly, due to entrapment.

Tenosynovitis technically refers to inflammation of the synovial sheath around the tendon. Causes can be grouped as either (i) inflammatory arthropathies, such as rheumatoid arthritis (RA), (ii) deposition diseases, such as gout, calcific ten-

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I.A. Trail, MBCHB, MD, FRCS (Edin), FRCS (Lon), ECFMG Upper Limb Unit, Department of Orthopaedics, Wrightington Hospital, Wigan, UK donitis and amyloid, (iii) infection or (iv) due to retained foreign body or implant.

Tendon entrapment is referred to as tendovaginitis. It can also somewhat erroneously be referred to as stenosing tenosynovitis, which does not accurately reflect the pathology being primarily that of a tendon being entrapped within its retinacular sheath. Histologically, there is a characteristic non-inflammatory hyperplasia of the retinacular sheath and corresponding thickening and nodularity in the tendon itself, as opposed to any synovial inflammation [1]. The most commonly entrapped tendons are those of the first extensor compartment.

Pertinent Anatomy

An understanding of tendon anatomy at the wrist is prudent to understanding the pathogenesis and management of tendonitis (Fig. 1.1). The 12 extensor tendons traverse the wrist through 6 fairly tight fibro-osseous membranes constituting the 6 extensor compartments. On the flexor

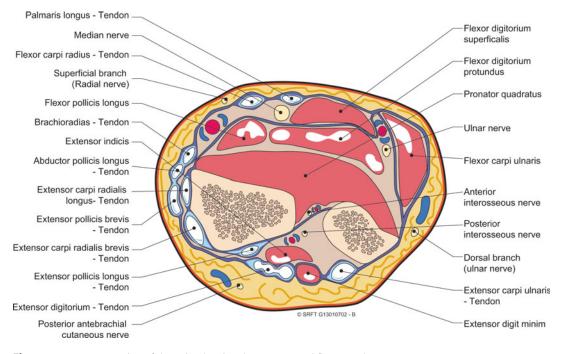


Fig. 1.1 Transverse section of the wrist showing the extensor and flexor tendons

aspect of the wrist, the carpal tunnel contains the 4 flexor digitorum profundus, 4 flexor digitorum superficialis and the flexor pollicis longus tendons. Flexor carpi radialis and flexor carpi ulnaris travel within their own tunnels.

Tenosynovitis

Rheumatoid Arthritis

Tenosynovitis at the wrist is a relatively common manifestation in RA patients, although the rate of presentation is now perceived to be subsiding due to improved medical management of the disease [2]. There is hypertrophy of the synovium, which may go on to infiltrate adjacent structures and unchecked can lead to tendon rupture. Patients may present with a history of pain when actively contracting or passively stretching the tendons. Often they can present with a relatively mobile, painless swelling, more evident on the dorsal side of the wrist, due to its expansile skin. There may be tenderness and crepitus felt on palpation of the swelling, which follows the course of the tendon and often there is a bulge on either side of the restrictive extensor retinaculum. This can help differentiate the tenosynovitic swelling from that of a ganglion or other tumour.

Inflammatory tenosynovitis can affect any dorsal compartment, but is most often present on the ulnar aspect of the wrist. Similar symptoms develop on the flexor surface, but presentation may be by sway of symptoms of carpal tunnel syndrome and/or flexor tendon triggering at the wrist level, due to the relatively rigid flexor retinaculum acting to entrap the median nerve and flexor tendons in the presence of synovial swelling.

The management of RA is covered in Chap. 7.

Deposition Diseases

The deposition of any material within the tendon sheath can precipitate an inflammatory response. There are a number of diseases which can lead to deposition within the tendon sheath, including gout, pseudogout, calcific tendonitis and amyloid. Patients present with intense pain, erythema and swelling.



Fig. 1.2 Clinical photo of gouty tophi affecting thumb and index finger DIPJ (Courtesy of Mr. Lindsay Muir, Salford Royal Hospital, Manchester, UK)

Gout

In gout, a disorder where urate metabolism leads to an overproduction of uric acid, hyperuricaemia and deposition of monosodium urate (MSU) crystals within the joints and soft tissues, including the tenosynovium. In longstanding, poorly controlled disease, gouty tophi may also be present. These are palpable, cheesy white, multilobular swellings formed by the deposition of crystals in the subcutaneous tissues of the digits (Fig. 1.2). Tophi in the hand are not usually seen without preceding deposits elsewhere in the body, typically the great toe and the pinna of the ear, the hand being an unusual primary site [3]. Gouty tendosynovitis will present in the extensor tendons in a similar way to RA and involvement of the flexor tendons can also present as carpal tunnel syndrome. Symptoms of sudden onset pain, erythema, warmth and swelling need to be differentiated from acute infection, which can also present in a similar manner. Conversely, it must also be kept in mind that deposition disease can simultaneously be present alongside infection. Unchecked, gouty tenosynovitis can lead to tendon rupture and skin ulceration at the sites of tophi [4].

A high uric acid level in the blood is not diagnostic for gout, as this is found in a small percentage of the normal population. Aspiration of the joint or tenosynovium is the most helpful diagnostic test. Examination of the synovial aspirate under a polarized light microscope will reveal strongly negatively birefringent crystals. Radiography may reveal intra-articular degenerative arthritic changes if the gout has been longstanding, whilst the use of ultrasound has recently been described in the detection of MSU crystals in subclinical gout [5].

The mainstay of management is medical and acute episodes usually respond well to a combination of colchicine and non-steroidal antiinflammatory drugs. Surgery may be indicated by way of tendon synovectomy for adherent tendons, tendon transfers for tendon rupture, median nerve decompression for carpal tunnel and excision of painful tophi [4]. Peri-operative colchicine cover is recommended to prevent a flare up of gout post-operatively.

Other Deposition Diseases CPPD

Calcium pyrophosphate dehydrate, or pseudogout, has been reported to cause an inflammatory synovitis. Symptoms and signs are similar to those already described for gout. Polarised light microscopy of aspirate reveals weakly positive bifringent rhomboid-shaped crystals. Debridement of the synovial sheath may be necessary [6].

Acute Calcific Tendonitis

As with other sites of calcific deposition in the body, such as the bursae of the shoulder, the aetiology of deposition is poorly understood. Calcific hydroxyapatite deposits are chalky white and their deposition within the tendon sheath can cause an intense, acute painful tenosynovitis. There is concomitant localised fluctuance, erythema and warmth. Diagnosis may be missed or delayed due to the presentation mimicking infection [7]. The patient is systemically well. Any wrist tendon may be affected and carpal tunnel syndrome in flexor tenosynovitis secondary to calcific deposition has been described [8].

The blood profile for infection will be negative and calcium levels in the blood are normal. Radiographs may reveal speckles of calcification within the soft tissues.

Acute episodes usually settle and are selflimiting and do not damage the tendon proper. Management can be confined to splintage and anti-inflammatory drugs. Surgical debridement, however, can expedite the resolution phase.

Amyloidosis

In patients with renal failure undergoing dialysis, the serum protein β 2-microglobulin is not filtered and can accumulate in soft tissues and bones. Amyloid deposition can occur along the tenosynovium of flexor tendons. Patients can present with reduced motion of the tendons, wrist tendon triggering and tendon rupture. Patients undergoing renal dialysis and who have carpal tunnel syndrome may well have amyloid plaques upon the flexor tendons. There are usually no signs of inflammation. Radiographs may reveal concomitant cystic lesions in the carpus and interphalangeal joints. Treatment is by way of the surgical debridement of thick amyloid plaques from tendon sheaths [9].

Sarcoidosis

Sarcoid tenosynovitis at the wrist is a rare but recognised condition. Both flexors and extensors at the wrist can be involved. Presentation is similar to other tenosynovitis as described above. Multiple non-caseating granulomatous nodules are found in this immune mediated condition, the aetiological trigger being unclear. Radiographs may reveal characteristic cystic lesions in the phalanges. Management is through the administration of corticosteroids and tenosynovectomy [10].

Infection

Acute or chronic infection within the tendon sheath can cause tenosynovitis. Pyogenic, gonococcus, tuberculosis, atypical mycobacterium and fungal infections all need to be considered.

Foreign Body and Implants

Any foreign body within the tendon sheath can incite an inflammatory response. Cases of plant thorns causing aseptic tenosynovitis have been described. In the absence of a clear history, ultrasound imaging may help localise the foreign body. Over the last decade, with the widespread use of volar locking plates for open reduction internal fixation of distal radius fractures, there has been an increase in reported extensor tendonitis, attrition and rupture. The surgeon must be careful of screw placement when performing this procedure and also have a low threshold of suspicion for this complication when presented with a patient complaining of pain, tenderness or reduced tendon motion on the extensor surface post-operatively. Management should be directed at surgical removal of the irritant.

Tendovaginitis

Quite distinct from tenosynovitis, tendovaginitis is a mechanical process caused by entrapment of the tendon within its retinacular sheath. Of note, inflammatory tissue is not found within the tenosynovium. However, an unduly thickened and degenerate sheath has been described in several histological studies. This has led authors to speculate on a more intrinsic, degenerate mechanism in these conditions, as opposed to an extrinsic, inflammatory one. The term stenosing tenosynovitis is thus felt not to reflect the pathological process [11].

It is not entirely clear why tendon entrapment occurs in some patients. Epidemiological trends are recognised, with patients often suffering from several conditions of the upper limb, including trigger finger, carpal tunnel syndrome, de Quervain's disease, epicondylitis and subacromial bursitis. This would suggest an, as yet, unrecognised underlying systemic process or predisposition. The condition is more prevalent in women and diabetics. Perhaps most controversial is the possible role that occupational factors may play in inducing tendon entrapment. Repetitive desk-based, computer and some manual work, sports or "overuse" have all been associated with tendovaginitis in some studies, but not in others. Whilst there may be some contribution by repetitive motions in the workplace and there has been some reported success with the modification of these activities, the condition may not fully be explained by this alone and other intrinsic factors may play a role [12].

De Quervain's Disease

The commonest site of tendon entrapment at the wrist is within the first dorsal compartment. Entrapment of the tendons of Extensor Pollicis Brevis (EPB) and Abductor Pollicis Longus (APL) was first described in 1895 by the Swiss surgeon Fritz de Quervain. It has also been referred to as washerwoman's sprain, gamer's thumb, mother's wrist or mother's thumb.

The two tendons run within a synovial sheath and enter a tunnel, the roof of which is formed by the relatively unyielding fibro-osseous extensor retinaculum (ER), with the EPB lying dorsal and ulnar to the APL. The ER acts to prevent bowstringing and thereby give the tendons a mechanical advantage. The floor of the tunnel is the groove of the radial styloid and the walls are the fibrous septae running up vertically from the radius to the ER. However, both the number of tendon slips and number of tunnels within the first extensor compartment have been found to be variable. Multiple slips of APL (1-7), multiple slips of EPB (0-3) and multiple tunnels have all been found in numerous anatomical studies [13, 14]. The literature suggests that the presence of a single EPB and APL within one sheath is present only in a minority of patients. This wide anatomical variation has significant implications in the conservative and surgical management of the condition.

Patients present with a history of a variable duration of pain, exacerbated by thumb movement, thumb pinching, grasping or twisting with the hand. They may complain of a general difficulty with movement around the radial aspect of the wrist. They may also notice slight swelling over the radial border of the wrist and infrequently complain of clicking or snapping in this region. The condition is most common in people in their sixth decade and far more common in women than men. The incidence is also higher in diabetics. The most striking subset of patients are post-partum, lactating mothers who classically present with de Quervain's during this period, postulated to be due to the new, repetitive positioning of their wrist whilst holding their infant. There may be a history of repetitive wrist movements involving abduction of the thumb with

ulnar deviation of the wrist. This movement is thought to put the tendons under most tension and a greater anatomic angulation of these tendons in women has been thought to account for the increase prevalence. Examination usually reveals swelling and tenderness overlying the tendons approximately 1-2 cm proximal to the radial styloid. The location of tenderness is important diagnostically, as a number of conditions which present on the radial side of the wrist need to be differentiated. Patients may present with intersection syndrome, classically 4-5 cm proximal to the radial styloid. Distal to the radial carpo-metacarpal styloid, thumb arthritis, scaphotrapezial-trapezoid arthritis, scaphoid fracture or radio-carpal arthritis may all cause pain in this region. Careful palpation of the radial border of the wrist can often elucidate the exact site of tenderness, which would correspond to the respective pathology. A positive Finklestein's test is diagnostically helpful. The test is correctly performed by holding the affected thumb in abduction and then forcibly ulnar deviating the hand away from the thumb to reproduce the symptoms. It is often incorrectly attributed to a test originally described by Eichhoff, in which the patient clasp's their thumb within their palm and then forces the wrist into ulna deviation - a test which may well invoke pain within an asymptomatic wrist. Other signs include triggering of the APL, pain on resisted active extension of the thumb and loss of ulnar deviation.

Radiographs may reveal a bony osteophyte within the first compartment, proximal to the styloid and will also help differentiate radio-carpal arthritis. Ultrasound (US) and magnetic resonance imaging (MRI) modalities have been used to diagnose de Quervain's. Characteristic tendon thickening, synovial sheath thickening and peritendinous oedematous changes are visible with both modalities. Useful information about the number of tendon slips and intertendinous septae can also be gleaned [15].

Non-operative Management

Rest alone, analgesia, splintage and steroid injection have all been advocated as methods of treating the condition.

Studies of lactating mothers have found complete resolution of symptoms following cessation of lactation without any intervention [16]. A number of papers have attempted to address the relative efficacy of splintage alone versus steroid injection. Although controlled clinical trials are few [17], consensus is that injection is by far more successful in achieving speedier resolution of symptoms, with success being quoted as up to 86 % [18, 19]. Splintage alone has not been shown to give lasting relief with failure rates as high as 70 % [20]. The surgeon should allow a minimum of 6 weeks before evaluating the success of injection. Further trials of injections may be considered, although the risks of steroid extravasation in this superficial, sensitive part of the wrist must be borne in mind. For this reason we advocate only one further injection attempt. Failure of complete relief of symptoms may be due to the presence of multiple septae within the first dorsal compartment. Recent studies have described the use of ultrasound to help identify differing sub-compartments in which to place the needle and to avoid superficial placement of the needle as a useful therapeutic adjunct [21, 22].

Technique

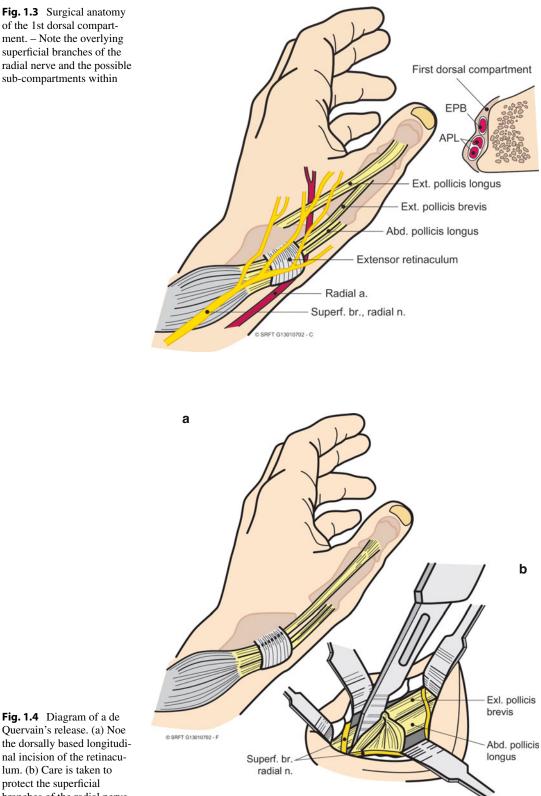
The patient should be advised of both the risk of steroid extravasation and of incomplete relief of symptoms. Using an aspetic technique, a 2 ml syringe with a 22-guage needle is used to administer a mixture of local anaesthetic and corticosteroid. The exact mixture may be the surgeon's preference as to what is locally available. The more soluble and short acting solutions of steroid may be safer and thereby reduce the likelihood of fat necrosis, subcutaneous atrophy and depigmentation. We use 1 ml of 1 % lidocaine and 40 mg of methylprednisolone acetate. The needle is placed 1 cm proximal to the radial styloid at a 45° angle to the longitudinal axis of the radial border of the forearm. To avoid a too superficial placement of the needle and the risk of steroid extravasation, we advocate inserting the needle into to the APL tendon and gently pressing on the syringe plunger, at which resistance will be felt. Whilst maintaining the pressure on the plunger and without trying to force the solution into the

tendon proper, the syringe is slowly withdrawn. As the syringe exits the tendon, a flush of fluid may be seen to pass proximally and distally within the first compartment. An attempt should also be made to infiltrate the EPB subsheath by injecting part of the solution dorso-ulnarly. The patient should get immediate relief of symptoms due to the local anaesthetic.

Surgery

In cases of failed steroid injection, release of the first compartment is the recommended treatment. In getting consent from the patient, specific mention needs to made of the risks of superficial radial nerve injury and incomplete relief of symptoms. A thorough understanding of the surgical anatomy is important to minimise the risk of complications (Figs. 1.3 and 1.4). Most at risk are the superficial branches of the radial nerve, which overlie the extensor retinaculum. Over zealous retraction, accidental laceration or suture of these branches can lead to exquisitely painful neuroma, sensitivity or numbness. Underlying the snuff box, just distal to the styloid is the radial artery, which is at risk if the surgeon strays too deep or distal.

The procedure is performed under local anaesthetic, with an inflated tourniquet for the duration. A 2 cm transverse incision is made 1 cm proximal to the styloid, taking care not to incise too deeply (Fig. 1.5). Use of skin hooks to elevate the skin allow it to be teased off from the subcutaneous fat by blunt dissection, which will allow for better visualisation and protection of the branches of the radial superficial nerve. The retinaculum can then be visualised and incised longitudinally at its most dorsal aspect (Fig. 1.6). At this point a thorough attempt for all possible tendon slips and compartments should be undertaken; all of which should be decompressed. All tendons are gently delivered out of the wound to confirm complete release (Fig. 1.7). Traction on the EPB will be confirmed by extension at the thumb MCP. If this does not occur, re-check that all sub-compartments have been identified and released. The patient is then asked, in turn, to actively abduct and then extend the thumb to again confirm full release. The proximity of the nerve is illustrated by it's position following



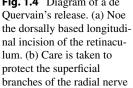




Fig. 1.5 Photograph showing surgical markings for release of 1st compartment. The radial styloid is marked with a *dot* and the site of the transverse incision is 1 cm proximal



Fig. 1.6 Photograph demonstrating release of the 1st dorsal compartment. The nerve is retracted and the retinaculum incised longitudinally on it's dorsal aspect



Fig. 1.7 The three tendons of 1st Compartment – a fleshy looking EPB dorsally and two slips of APB volarly (the most frequent pattern)

release (Fig. 1.8). After haemostasis is achieved, the skin is closed with a subcuticular 3/0 mono-filament suture. A thick bulky dressing is applied



Fig. 1.8 Superficial branch radial nerve intact following full release of the 1st compartment

for 48 h and mobilisation is commenced immediately to avoid any tendon adherence. Heavy lifting with the hand is avoided for the first 6 weeks. No formal hand therapy is usually required.

Open surgery is reported to have a high success rate with a resolution of symptoms quoted as high as 91 % with a patient satisfaction rate of 88 % in a long term review [23].

Complications

There are three main complications to consider; nerve injury, incomplete compartmental release and volar subluxation of the tendons.

By far the most troublesome is injury to the superficial branches of the radial nerve, which can lead to numbness, or worse; a sensitive and painful neuroma. Some authors advocate a longitudinal but less aesthetic incision, to offer safer exposure. Scheller et al. reported no neuromas in a long term follow up of 96 cases, reporting only four cases of transient nerve lesions using a longitudinal approach [24, 25]. Care must be taken, not only during the initial dissection and retraction, but also skin closure when the nerve is at risk of either laceration or retraction. Management of an established neuroma can be challenging and is addressed in Vol 2, chapter 1.

Failure of release of all possible subcompartments is a well recognised complication. In particular, failing to release the EPB subsheath is cited as one reason for incomplete relief of symptoms. For this reason, the surgeon should



Fig. 1.9 The larger volar leaf of the extensor retinaculum is identified buttressing the tendons of the 1st Compartment

recognise, as previously discussed, the anatomical variations found within this compartment. Suspicion of incomplete release may be investigated further by way of US or MRI and, if positive, the patient should be re-explored. Patients who have had no resolution of symptoms should be also assessed for co-existing pathologies on the radial border of the wrist, as previously discussed.

Finally, volar subluxation of the APL and EPB following release has been cited as an infrequent but problematic complication. To avoid this complication, the majority of authors advocate incising the retinaculum as dorsal as possible and not to excise the entire retinacular sheath (Fig. 1.9). Some suggest suturing of the retincular flap to the subcutaneous tissue to act as a ledge upon which the tendons can sit, thus preventing subluxation [26]. Various techniques have been described to address established volar subluxation by pulley reconstruction using a part of the extensor retinaculum or brachioradialis, reporting good resolution of symptoms [27, 28].

Alternative techniques to the classic open de Quervain's release have been described to achieve relief of symptoms and mitigate against complications of nerve injury and tendon subluxation. Kang et al. reported success using an endoscopic technique [29], whilst Okada and Kutz described a technique of removal of accessory APL tendon slips to successfully decompress the compartment [30].

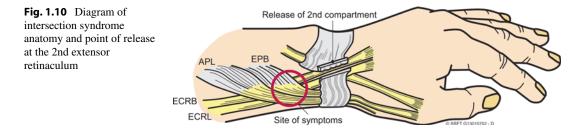
Clinical Pearls

- Initially incise the skin gently to avoid direct laceration of the superficial branches of radial nerve.
- Incise the ER on its most dorsal aspect to avoid tendon subluxation
- Undertake a thorough search for all tendon slips and sub-compartments, incising each septum encountered.
- Retract released tendons gently to visualise passive, smooth abduction and extension of the thumb.
- Ask the patient to actively abduct and extend the thumb to confirm full release.

Intersection Syndrome

The second dorsal wrist compartment contains the tendons of extensor carpi radialis longus (ECRL) and extensor carpi radialis brevis (ECRB). Intersection syndrome describes the symptoms and signs of pain and swelling that occur 4-5 cm proximal to the radial styloid as the muscle bellies of EPB and APL cross over the wrist extensors in the second compartment. However, this syndrome is not due to any friction between the groups of tendons crossing over. The pathology itself is distal and ulnar to this location, within the tendon sheath of the wrist extensors underlying the tight extensor retinaculum of the second compartment. As there is not much space under the retinaculum, swelling extends proximally to the point of "intersection" [31].

The syndrome has been linked to wrist "overuse", usually in athletes performing activities such as rowing, weight-lifting or racquet sports. The use of MRI can help diagnose the condition, with peri-tendinous oedema evident on imaging [32]. Conservative management is the first line treatment and patients are encouraged to modify their activities and rest the wrist in a splint. An injection of local anaesthetic and steroid can also be inserted. A recent study successfully used soft tissue taping in five athletes to resolve symptoms in all at 3 weeks, with patients being symptom



free for 1 year [33]. Conservative treatment is usually sufficient for resolution of symptoms. In resistant cases, the second compartment is released by a longitudinal incision overlying the ECRL and ECRB (Fig. 1.10). Post-operatively the wrist is splinted in slight dorsiflexion [31]. Care is taken to protect superficial branches of the radial nerve and avoid damage to the extensor pollicis longus tendon which is adjacent in the third compartment (Fig. 1.5).

Extensor Pollicis Longus

The EPL travels within the third dorsal compartment, through a relatively lengthy fibro-osseous tunnel, which angles sharply in a radial direction around Lister's tubercle as the tendon travels towards the thumb's inter-phalangeal joint. Within it's synovial tunnel, the tendon is relatively avascular, being dependant on synovial fluid for it's nutrition. Entrapment and subsequent rupture of the tendon is sometimes seen following undisplaced extra-articular distal radius fractures. It is thought that in an undisplaced fracture a haematoma builds up and exerts pressure within the fibro-osseous tunnel leading to ischaemia to the avascular portion of the tendon [34]. EPL rupture has also been reported secondary to synovitis of the extensor carpi radialis tendons [35].

Patients present with pain and swelling, exacerbated by thumb IP joint movement. Triggering of the tendon at Lister's tubercle is also a recognised symptom [36]. Tenderness and crepitus may be noted on palpation. As the EPL has a tendency to rupture, patients may present 6–7 weeks on following their fracture, or in clinic following removal of their cast, with an inability to retropulse the thumb.

As there is a definite risk of rupture, when the patient presents with swelling and pain, urgent surgical debridement is the first line of treatment and steroid injection is contra-indicated. The tendon is approached through a small, dorsal incision just ulnar to Lister's tubercle. Superficial branches of the radial nerve must be protected. The extensor retinaculum is divided and the EPL tendon gently lifted. The tunnel is debrided of any osteophytes. The tendon can then be transposed radial to the tubercle and the tunnel closed to prevent the tendon from slipping back. The thumb can be mobilised immediately. In the case of acute rupture, a palmaris longus interposition graft can be used and in delayed presentation, extensor indicis proprius to EPL tendon transfer is the reconstruction of choice [37].

Forth and Fifth Dorsal Extensor Compartments

The tendons of EIP and EDC travel within the fourth compartment, with extensor digiti minimi travelling in its own fifth fibrous compartment. Far less common than at other sites, patients can present with symptoms of swelling, pain, crepitus, tenderness, snapping and triggering within these compartments. Extensor indicis proprius syndrome is well recognised and thought to be due to hypertrophy of its muscle belly extending distally under the tight retinacular sheath. The patient will complain of pain, particularly in wrist flexion and asking the patient to actively extend the index finger against resistance whilst maintain the wrist in flexion will induce pain just radial to Lister's tubercle. Anomalous, additional tendons within these compartments are a well recognised cause of dorsal wrist pain and swelling

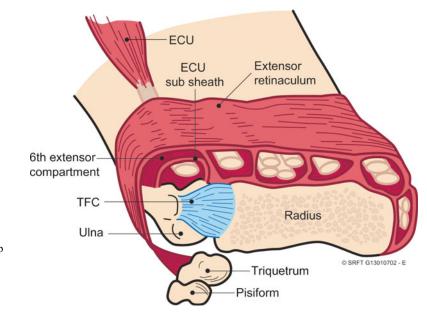
and may be mistaken for a dorsal wrist ganglion [38]. If conservative measures fail, one should consider the possibility of anomalous tendons. Partial release of the extensor retinaculum, or in the case of anomaly, a partial excision of tendon can be performed to relieve symptoms [39].

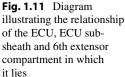
Extensor Carpi Ulnaris Tendonopathy

An understanding of the anatomy of the ECU as it crosses the wrist is imperative, both in diagnosis and management. The ECU muscle becomes tendinous approximately 2 cm proximal to the ulna head and at this point passes through a fibroosseous tunnel on its way to attach to the base of the 5th metacarpal. The fibro-osseous tunnel is composed of an investing fascia of the forearm, termed the ECU subsheath, which fully encircles the tendon sitting in the osseous grove of the ulna. This osseous groove is situated on the dorsal aspect of the distal ulna, between the ulna head and it's articular facet with the radius at the DRUJ. The subsheath itself is tightly adherant to the osseous groove. The subsheath ends distally, by blending with the dorsal capsule over the dorsum of the triquetrum. The extensor retinaculum forms the 6th dorsal compartment as it arches over this fibro-osseous tunnel, extending ulnarward, draping over the ulna, going on to attach to the triquetrum and pisiform. The retinaculum does not attach to the ECU, ECU subsheath or the ulna (Fig. 1.11). This allows for the wrist to prono-supinate. The relative adherence of the ECU subsheath within the fibro-osseous groove results in the ECU tendon being both bound tightly to the ulna and being placed under significant stresses when the wrist goes from pronation to supination. This anatomical arrangement is thought to account for the tendons preponderance to tendonitis, entrapment and instability.

Tendinopathy of the ECU is not uncommon and is regarded as second only to de Quervain's disease in frequency of tendinopathy at the wrist [40]. As the symptoms are often poorly localised, it may well be that it is more prevalent than perceived.

Tendonitis can be secondary to overuse, simple soft-tissue trauma following a fall or twist, or be related to instability of the ECU. There is a propensity for those involved in stick or racquet sports to develop ECU tendinopathy, due to the forceful wrist, flexion and supination movements involved, leading to tension and abrasion of the tendon [41]. It may also occur in association with either degeneration or injury of an adjacent





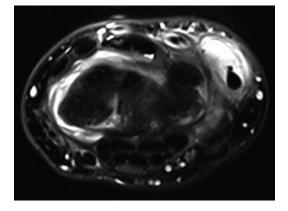


Fig. 1.12 Gadolinium enhanced MRI demonstrating florid tendonitis of the ECU within its sub-sheath (*top right* of the image) (Courtesy of Mr. Lindsay Muir, Salford Royal Hospital, Manchester, UK)

structure, such as the luno-triquetral ligament, TFCC, ulnar styloid or distal radial ulnar joint. As in other dorsal compartments, tenovaginitis may also be secondary to an anomalous accessory tendon slip within the 6th extensor compartment [42]. The proximity of adjacent structures, which may be involved in isolation or in combination with the ECU, as mentioned above, make this condition diagnostically challenging [40, 43].

Patients may present with an acute onset of diffuse ulnar-sided swelling and pain. However, the pain may be difficult to pinpoint accurately. The proximity of branches of the ulna nerve may give rise to patchy paraesthesia or dysaesthesia. There may also be tenderness and crepitus on palpation of the tendon. All active movements of the wrist are painful, in particular extension and ulnar-deviation [40]. Passive prono-supination of the wrist may elicit subluxation or "snapping" of an unstable ECU as the wrist is moved into flexion and supination. Injection the ECU sheath with local anaesthetic will confirm the diagnosis, if the symptoms settle [43].

Dynamic ultrasound can be very useful in diagnosing an unstable ECU. MRI is also particularly useful in evaluating tendinopathy, tendonitis and tendon rupture (Fig. 1.12) [44].

Conservative management by way of rest, splintage and analgesia is the first line of treatment and has been shown to be effective. In resistant cases, injection of corticosteroid may be sufficient to resolve symptoms [45]. If conservative measures fail, a release of the tunnel can be performed. This is done through a dorsal approach, centred over the ulnar groove, just radial to the ulna head. The superficial branches of the ulna nerve are identified and carefully preserved. The extensor retinaculum is divided to access the tunnel, which is then released fully. The osseous groove is inspected and debrided if necessary. The retinaculum is then repaired for ECU stability, although some authors have found no adverse effects from leaving the retinaculum open [45, 46].

Flexor Carpi Radialis

The flexor carpi radialis (FCR) tendon is lined with synovium as it passes through a fibro-osseous tunnel on the volar radial aspect of the wrist (Fig. 1.13). The tendon ultimately inserts onto the base of the second metacarpal. The distal aspect of the tunnel wall is composed mainly of the medial facet of the trapezium. Proximal to the trapezium, to the radial side of the tendon, is the tubercle of the scaphoid. As the tendon travels through this narrow fibro-osseous tunnel it is prone to stenotic tendovaginitis, particularly if there is a degenerative process involving the trapezium, scaphoid or scaphotrapezial joint [47, 48].

It is more frequent in women of late middle age, who are likely to have concomitant scaphotrapezial osteoarthritis. Sporting activities involving strenuous wrist flexion may also bring on symptoms [49].

Patients will present with pain on the volar radial aspect of the wrist. On examination there may be swelling and tenderness along the course of the tendon and pain may be reproduced by resisted flexion. The surgeon needs to differentiate FCR tendonditis from de Quervain's disease, scaphotrapezial arthritis, scaphoid fracture, base of thumb arthritis and ganglion cysts. An injection of local anaesthetic into the FCR sheath may assist diagnosis by transient relief of symptoms.

Conservative management by analgesia, rest and splintage is again the first line treatment. If this is not successful, corticosteroid injection of

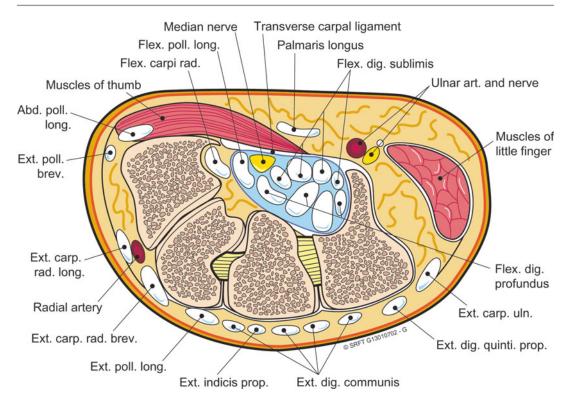


Fig. 1.13 Cross-sectional diagram showing journey of FCR sitting adjacent and ulnar to the side wall of the trapezium which roofs over it

the sheath may resolve symptoms. If this is ineffective then the tunnel is released. An incision is made over the tubercle of the scaphoid. The palmar cutaneous branch of the median nerve is avoided, as the tendon is identified and a release is performed from proximal to distal, opening up the entire fibro-osseous tunnel. The tendon is retracted gently to allow for inspection and debridement of the tunnel, which may well contain sharp osteophytes, secondary to the degenerative processes at the trapezium and/or scaphoid [48].

References

- Drossos K, Remmelink M, Nagy N, de Maertelaer V, Pasteels J, Schuind F. Correlations between clinical presentations of adult trigger digits and histologic aspects of the A1 pulley. J Hand Surg Am. 2009;34(8): 1429–35.
- Chung K, Pushman A. Current concepts in the management of the rheumatoid hand. J Hand Surg Am. 2011;36(4):736–47.

- Moore JR, Weiland AJ. Gouty tenosynovitis in the hand. J Hand Surg Am. 1985;10:291–5.
- Tang C, Fung B. The last defence? Surgical aspects of gouty arthritis of hand and wrist. Hong Kong Med J. 2011;17(6):480–6.
- Dalbeth N, Doyle A, McQueen F. Imaging in gout: insights into the pathological features of the disease. Curr Opin Rheumatol. 2012;24(2):132–8.
- Gerster J, Lagier R. Upper limb pyrophosphate tenosynovitis outside the carpal tunnel. Ann Rheum Dis. 1989;48(8):689–91.
- Dilley D, Tonkin M. Acute calcific tendinitis in the hand and wrist. J Hand Surg Br. 1991;16(2):215–6.
- Harris AR, McNamara TR, Brault JS, Rizzo M. An unusual presentation of acute calcific tendinitis in the hand. Hand (N Y). 2009;4(1):81–3.
- Fitzpatrick DC, Jebson PJL, Madey SM, Steyers CM. Upper extremity musculoskeletal manifestations of dialysis-associated amyloidosis. Iowa Orthop J. 1996;16:135–8.
- Wang H, Sunil T, Kleinert HE. Multiple unusual complications after extensive chronic sarcoid tenosynovitis of the hand: a case report. J Hand Surg Am. 2005; 30(3):610–4.
- Clarke MT, Lyall HA, Grant JW, Matthewson MH. The histopathology of de Quervain's disease. J Hand Surg Br. 1998;23(6):732–4.

- Waersted M, Hanvold T, Veiersted K. Computer work and musculoskeletal disorders of the neck and upper. BMC Musculoskelet Disord. 2010;11:79.
- Gonzalez M, Sohlberg R, Brown A, Weinzweig N. The first dorsal extensor compartment: an anatomic study. J Hand Surg Am. 1995;20(4):657–60.
- Shiraishi N, Matsumura G. Anatomical variations of the extensor pollicis brevis tendon and abductor pollicis longus tendon-relation to tenosynovectomy. Okajimas Folia Anat Jpn. 2005;82(1):25–9.
- Diop AN, Ba-Diop S, Sane JC, Tomolet Alfidja A, Sy MH, Boyer L, et al. Role of US in the management of de Quervain's tenosynovitis: review of 22 cases. J Radiol. 2008;89(9 Pt 1):1081–4.
- Avci S, Yilmaz C, Sayli U. Comparison of nonsurgical treatment measures for de Quervain's disease of pregnancy and lactation. J Hand Surg Am. 2002;27(2): 322–4.
- Peters-Veluthamaningal C, van der Windt DAWM, Winters JC, Meyboom-de Jong B. Corticosteroid injection for de Quervain's tenosynovitis. Cochrane Database Syst Rev. 2009;(3):CD005616.
- Richie 3rd CA, Briner Jr WW. Corticosteroid injection for treatment of de Quervain's tenosynovitis: a pooled quantitative literature evaluation. J Am Board Fam Pract. 2003;16(2):102–6.
- Mehdinasab SA, Alemohammad SA. Methylprednisolone acetate injection plus casting versus casting alone for the treatment of de Quervain's tenosynovitis. Arch Iran Med. 2010;13(4):270–4.
- Weiss AP, Akelman E, Tabatabai M. Treatment of de Quervain's disease. J Hand Surg Am. 1994;19(4):595–8.
- Jeyapalan K, Choudhary S. Ultrasound-guided injection of triamcinolone and bupivacaine in the management of De Quervain's disease. Skeletal Radiol. 2009; 38(11):1099–103.
- McDermott JD, Ilyas AM, Nazarian LN, Leinberry CF. Ultrasound-guided injections for de Quervain's tenosynovitis. Clin Orthop Relat Res. 2012;470(7):1925–31.
- Ta KT, Eidelman D, Thomson JG. Patient satisfaction and outcomes of surgery for de Quervain's tenosynovitis. J Hand Surg Am. 1999;24(5):1071–7.
- Bouras Y, El Andaloussi Y, Zaouari T, Touil N, Fnini S, Chikhaoui N, et al. Surgical treatment in De Quervain's tenosynovitis. About 20 cases. Ann Chir Plast Esthet. 2010;55(1):42–5.
- Scheller A, Schuh R, Hönle W, Schuh A. Long-term results of surgical release of de Quervain's stenosing tenosynovitis. Int Orthop. 2009;33(5):1301–3.
- Le Viet D, Lantieri L. De Quervain's tenosynovitis. Transversal scar and fixation of the capsular flap. Rev Chir Orthop Reparatrice Appar Mot. 1992;78(2):101–6.
- Ramesh R, Britton JM. A retinacular sling for subluxing tendons of the first extensor compartment. A case report. J Bone Joint Surg Br. 2000;82(3):424–5.
- McMahon M, Craig SM, Posner MA. Tendon subluxation after de Quervain's release: treatment by brachioradialis tendon flap. J Hand Surg Am. 1991;16(1):30–2.
- Kang HJ, Hahn SB, Kim SH, Choi YR. Does endoscopic release of the first extensor compartment have

benefits over open release in de Quervain's disease? J Plast Reconstr Aesthet Surg. 2011;64(10):1306–11.

- Okada M, Kutz JE. Excision of aberrant abductor pollicis longus tendon slips for decompression of de Quervain's disease. J Hand Surg Eur Vol. 2011;36(5):379–82.
- Grundberg AB, Reagan DS. Pathologic anatomy of the forearm: intersection syndrome. J Hand Surg Am. 1985;10(2):299–302.
- Lee RP, Hatem SF, Recht MP. Extended MRI findings of intersection syndrome. Skeletal Radiol. 2009;38(2): 157–63.
- 33. Kaneko S, Takasaki H. Forearm pain, diagnosed as intersection syndrome, managed by taping: a case series. J Orthop Sports Phys Ther. 2011;41(7):514–9.
- Engkvist O, Lundborg G. Rupture of the extensor pollicis longus tendon after fracture of the lower end of the radius–a clinical and microangiographic study. Hand. 1979;11(1):76–86.
- Bonatz E, Kramer TD, Masear VR. Rupture of the extensor pollicis longus tendon. Am J Orthop. 1996;25(2):118–22.
- Kardashian G, Vara AD, Miller SJ, Miki RA, Jose J. Stenosing synovitis of the extensor pollicis longus tendon. J Hand Surg Am. 2011;36(6):1035–8.
- Skoff HD. Postfracture extensor pollicis longus tenosynovitis and tendon rupture: a scientific study and personal series. Am J Orthop. 2003;32(5):245–7.
- Baker J, Gonzalez MH. Snapping wrist due to an anomalous extensor indicis proprius: a case report. Hand (N Y). 2008;3(4):363–5.
- Tan ST, Smith PJ. Anomalous extensor muscles of the hand: a review. J Hand Surg Am. 1999;24(3):449–55.
- Nachinolcar UG, Khanolkar KB. Stenosing tenovaginitis of extensor carpi ulnaris: brief report. J Bone Joint Surg Br. 1988;70(5):842.
- Montalvan B, Parier J, Brasseur JL, Le Viet D, Drape JL. Extensor carpi ulnaris injuries in tennis players: a study of 28 cases. Br J Sports Med. 2006;40(5):424–9; discussion 429.
- Allende C, Le Viet D. Extensor carpi ulnaris problems at the wrist–classification, surgical treatment and results. J Hand Surg Br. 2005;30(3):265–72.
- Ruland RT, Hogan CJ. The ECU synergy test: an aid to diagnose ECU tendonitis. J Hand Surg Am. 2008;33(10):1777–82.
- Coggins CA. Imaging of ulnar-sided wrist pain. Clin Sports Med. 2006;25(3):505–26, vii.
- Crimmins CA, Jones NF. Stenosing tenosynovitis of the extensor carpi ulnaris. Ann Plast Surg. 1995;35(1):105–7.
- Kip PC, Peimer CA. Release of the sixth dorsal compartment. J Hand Surg Am. 1994;19(4):599–601.
- Bishop AT, Gabel G, Carmichael SW. Flexor carpi radialis tendinitis. Part I: operative anatomy. J Bone Joint Surg Am. 1994;76(7):1009–14.
- Gabel G, Bishop AT, Wood MB. Flexor carpi radialis tendinitis. Part II: results of operative treatment. J Bone Joint Surg Am. 1994;76(7):1015–8.
- Soejima O, Iida H, Naito M. Flexor carpi radialis tendinitis caused by malunited trapezial ridge fracture in a professional baseball player. J Orthop Sci. 2002;7(1):151–3.

Tendon Conditions in the Thumb

David Warwick

Keywords

Thumb • Tendonitis • Tendinosis • Tenosynovitis • Attrition rupture • Anatomy • Peritendonitis crepitans • Rheumatoid arthritis • De Quervain's syndrome • Trigger thumb

Introduction

Box 2.1: Intrinsic and Extrinsic Control of the Thumb

Intrinsic Muscles to the Thumb

- Adductor Pollicis
- Flexor Pollicis Brevis
- Abductor Pollicis Brevis
- Opponens Pollicis Extrinsic Tendons to the Thumb
- APL: Abductor Pollicis Longus
- EPL: Extensor Pollicis Longus
- EPB: Extensor Pollicis Brevis
- ED: Extension Formers Drevis
- FPL: Flexor Pollicis Longus

The thumb is the crucial pillar of the hand; the opposable thumb makes the hand a tool. The sliding basal joint, with the hinged metacarpo-phalangeal joint (MCPJ) and inter-phalangeal joint (IPJ), require a balanced set of extrinsic tendons and intrinsic muscles to provide co-ordinated and accurate movement (see Box 2.1). The extrinsic tendons pass towards the thumb across the carpus, constrained by fascial compartments and sheaths. The tendons and surrounding sheaths are prone to pathological processes, not always inflammatory, which are the subject of this chapter.

Anatomy

Extensor Compartments

The extrinsic extensor tendons to the thumb pass beneath the extensor retinaculum. The extensor retinaculum is a consolidation of the deep dorsal forearm fascia and the fascia between the dermis and tendons over the back of the hand. The retinaculum is secured to the underlying radius with vertically-descending pillars, thus forming individual compartments

- First compartment–EPB, APL
- Second Compartment–ECRB, ECRL
- Third Compartment-EPL

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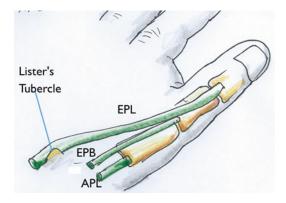


Fig. 2.1 Extensor and abductor tendons of the thumb

Extrinsic Extensor Tendons

The APL, EPB and EPL each attach just distal to a separate sequential joint in the thumb-trapeziometacarpal (TMJ), MCPJ and IPJ respectively (Fig. 2.1).

APL moves the metacarpal base into extension and also palmar flexion. It has an essential role in stabilising the thumb base in the position of function (extension-flexion), providing a stable platform for pinch grip.

EPB extends the proximal phalanx of the thumb. It antagonises FPL in power pinch, preventing excessive flexion at the MCPJ. There can be anomalous insertions into the extensor hood or the APL, or even entire absence. The tendon may duplicate, or even triplicate, within the first dorsal compartment [1]; recognition of which is essential to ensure thorough release during a de Quervain's operation.

EPL has a contributory role in extending the distal phalanx of the thumb (the thumb intrinsics have a complimentary role through the extensor hood). EPL predominantly pulls the thumb backwards and ulnarwards such that the palmar-ulnar corner of the thumb IPJ opposes the radial side of the index MCPJ. This position opens the hand in preparation for subsequent opposition and prehension. Even if the EPL is ruptured or denervated, the thumb DIP joint will still extend, animated through the extensor hood by the adductor pollicis (ulnar nerve) and abductor pollicis brevis (median nerve).

FPL primarily flexes the tip of the thumb. To prevent "bowstringing" across the thenar emi-

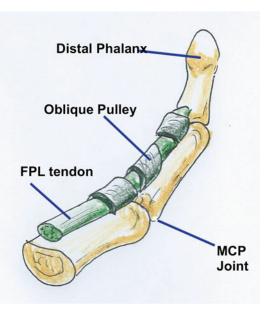


Fig. 2.2 Flexor sheath of the thumb

nence in power-pinch, it runs through a fibrous sheath. The sheath commences at the level of the MCPJ and ends at the level of the IPJ (Fig. 2.2).

The tendon commences in the distal quarter of the forearm, motivated by powerful unipennate muscle fibres. The tendon runs through a fibrous sheath immediately palmar to the scaphoidtrapezoid-trapezium joint, then runs freely through the carpal tunnel radial-dorsal to the median nerve, then between adductor pollicis and the flexor pollicis brevis bellies, to reach the flexor sheath. It attaches distally into the base of the distal phalanx, often through a small sesamoid bone which is visible on a lateral radiograph and easily confused with a small avulsion fracture.

Terminology and Pathology of Thumb Tendon Disorders

Conditions of the tendon system should be named with care to avoid misleading inferences on aetiology or treatment.

Tendonitis: The term *tendonitis* should refer to an inflammatory condition of the tendon itself – a very unusual pathological occurrence. *Tendinosis*: The tendon itself may occasionally be affected by a pathological condition, such as gout or abrasion beneath a tight sheath.

Tenosynovitis: this refers to an inflammatory process affecting the synovium. To substantiate this diagnosis, there must be inflammatory cells present, with consequent physical signs of swelling, tenderness and crepitus along the tendon sheath. There may be an inflammatory, infective or depository causes including:

- mycobacterial infection
- fungal infection
- · foreign body
- sarcoidosis
- gout
- amyloid

Tendon sheath disorders: these are a separate group – de Quervain's disease and trigger thumb – and are discussed below.

Rheumatoid Arthritis

The FPL and long extensor tendons are surrounded by a synovial sheath and so may be affected by rheumatoid arthritis. Nodules may also form within the tendon. Synovitis and even tendon rupture used to be quite common, both FPL and EPL being particularly vulnerable. Fortunately, these problems are becoming more rare with the advent of effective diseasemodifying agents. Trigger thumb may occur from impingement of thickened synovium within the sheath.

Treatment

- medical management of the rheumatoid arthritis
- cortisone injection for synovitis
- synovectomy
- sheath release (trigger thumb, EPL synovitis)
- tendon transfer (e.g. EIP into EPL, FDS (IV)) into FPL or interposition grafting
- fusion (IPJ for FPL rupture or MCP for EPB rupture when grafting or transfer unsuitable or failed).

Attrition Disorders

Surgical fixation is hazardous to the tendons of the thumb.

Kirschner Wires

APL, EPB and EPL are all prone to irritation by Kirschner wires passed percutaneously for stabilisation of the distal radius fracture. This may present with pain, stiffness, infection and even rupture. Careful surgical technique (equally important to avoid cutaneous nerve damage) is essential.

Dorsal Plates

Earlier designs had a prominent distal edge. This was a potent cause of tenosynovitis or tendon rupture of the finger extensors and especially EPL (Fig. 2.3). Lower profile plates with a distal bevel are safer but not infallible. Placing the plate beneath the ECRL and ECRB will preserve EPL, if the fracture repair or osteotomy reconstruction allows.



Fig. 2.3 EPL tendon rupture from dorsal plate



Fig. 2.4 (a, b) Volar plate causing attrition tendon rupture of APL (a) and FPL (b)

Volar Plates

The edges of the plate can cause attrition synovitis and rupture of APL/EPB and FPL (Fig. 2.4a, b). A further problem with volar plating is protrusion of the screws dorsally, eroding EPL. A true lateral x-ray may give false reassurance, due to the obliquity of the dorsum of the distal radius either side of Lister's tubercle (Fig. 2.5a–c).

FPL Attrition from STT Arthritis

The FPL is a direct anterior relation of the scaphoid-trapezoid-trapezium joint (STT or Triscaphe joint).

A combination of osteophytes and synovial thickening from this joint may erode the FPL, causing tendinopathy and eventual rupture. There is pain in the wrist on moving the thumb tip and eventually either painful or unexpected painless rupture (Mannerfelt Syndrome). Occasionally, FCR tendon is involved. Radiographs show an advanced STT arthritis (Fig. 2.6). Treatment requires judicious removal of the distal pole of the scaphoid (never more than one fifth, to avoid secondary midcarpal collapse [2]) and interposition grafting.

De Quervain's Disease

Box 2.2: Key Points: de Quervain's

- Usually constitutional changes in sheath
- Finklestein's and Hitch-hiker's tests positive
- Usually responds to injection
- Beware extra tendons and hidden compartments during surgery
- Beware superficial radial nerve during surgery

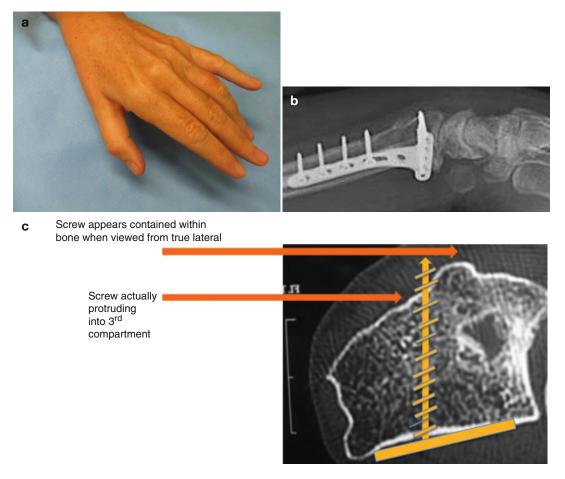


Fig. 2.5 (a) EPL rupture; (b) parallax error on lateral radiograph; (c) oblique radiograph showing long screw impinging in third compartment



Function of the First Compartment

This condition affects the first dorsal extensor compartment. This contains the Abductor Pollicis Longus (APL) and Extensor Pollicis Brevis (EPB) tendon, which attach to the base of the metacarpal and proximal phalanx respectively. The APL stabilises the thumb base in lateral extension and palmar flexion, preventing collapse on power pinch and enabling some span of the thumb web. EPB extends the MCP joint, considerably opening the span of the thumb web. Together, the tendons put the thumb into the "hitchhiker pose". The first compartment of the extensor retinaculum prevents bowstringing of these tendons in pinch, or in extending the thumb into full span.

Fig. 2.6 STT arthritis

Anatomical Variants

APL tendon has many variations, frequently more than one slip within the compartment. Further slips may pass across ulnarwards to the APB or trapezium. The tendon may also fuse with EPB.

The EPB tendon runs in a separate subcompartment of the first compartment in 30–60 %; this must be specifically excluded during surgery to avoid persisting symptoms.

Pathology

The cause is unknown. There is an increase in extracellular matrix, thickening of the collagen fibrils and areas of fibrocartilage metaplasia. The tendon surface may be fibrillated or delaminated. There is no pathological evidence of inflammation – so the term "tenosynovitis" is illogical [3, 4].

Clinical Features

The great majority of cases occur spontaneously.

It is more common in females than males; occurrence in very late pregnancy or soon after delivery is characteristic. It may occasionally follow unaccustomed overuse, but "repetitive strain" as an aetiology is at best speculative.

The typical complaint is pain aggravated by thumb use. The patient points to the tip of the radial styloid. Occasionally there is even triggering of the APL tendon [5] or a small hard tender ganglion over the first compartment.

Physical signs include swelling and tenderness over the tip of the radial styloid, severe pain on resisted active extension of the thumb, loss of ulnar deviation and a positive Finkelstein's test. Finkelstein described reproduction of pain by 'grasping the patient's thumb and quickly abducting the hand ulnarward'. There is another test, which was originally described by Eichhoff but is erroneously attributed to Finkelstein, in which pain is provoked by deviating the wrist ulnarward while the thumb is held in the palm beneath the flexed fingers [6]. The differential diagnosis of de Quervain's includes intersection syndrome (see below), a volar wrist ganglion and degenerative arthrosis of the trapezio-metacarpal, scapho-trapezial, or radio-scaphoid joints.

Treatment

Rest is helpful, if the symptoms have been provoked by a brief period of unaccustomed repetitive and forceful activity. This can be supplemented with a course of oral, non-steroidal, antiinflammatory *drugs* and a *splint*. A splint can help but is cumbersome.

Physiotherapy (ultrasound, frictions) might help some, but the local pressure can be very uncomfortable.

Cortisone injection can be very effective (80 % success), especially if there is frank tenosynovitis (i.e. crepitus, linear tenderness) [7–9]. Half a millilitre of preparation is administered into the sheath through a fine-bore needle, the skin having been sterilised with alcohol and then frozen with ethyl chloride. The patient should be warned of the tiny risk of damage to the superficial radial nerve, which can provoke a dystrophic response. If there is any neuralgic or severe pain on needle insertion, the compound must not be injected. Instead, the needle should be repositioned. Skin discoloration and subcutaneous fat atrophy may also occur.

Surgery cures the majority. Surgical loupes are mandatory. The arm is exsanguinated and a tourniquet inflated. Under a local anaesthetic, a transverse skin-crease incision is made at the level of the tip of the radial styloid. The subcutaneous tissues are very gently spread with fine tenotomy scissors, sliding the fat containing the superficial radial nerve fibres dorsally and palmarwards on the plane of the retinaculum. The retinaculum is clearly exposed on its outer surface. The retinaculum is divided along its dorsal third, thus preserving an anterior margin to minimise anterior subluxation of the tendons, when the wrist is subsequently flexed. A very careful search is made for one or more hidden compartments. Thirty to sixty per cent have a septum separating APL and EPB [10]; multiple tendons are not uncommon. If there is synovitis, this is removed and, if necessary, specimens are sent for analysis. The wound is washed out thoroughly and then the tourniquet deflated. Once haemostasis is secured with gentle pressure (not diathermy, because of the vulnerability of the cutaneous nerves to inadvertent damage), the wound is closed with a continuous subcutaneous soluble suture. Splinting is not advised – early restoration of gliding planes is always recommended in hand surgery.

EPL Tendon

Anatomy

It runs through the 3rd dorsal compartment. Lister's tubercle pulls the tendon ulnarwards. This imparts a vector so that contraction of the muscle pulls the thumb dorsally and ulnarwards – *retroposition*.

Tenosynovitis

The EPL tendon can be affected by an inflammatory tenosynovitis. This is usually provoked by unaccustomed overuse. It presents with the cardinal features of a tenosynovitis – pain, swelling, tenderness and crepitus along the course of the tendon. Colour duplex ultrasonography is a very sensitive test for confirmation.

Treatment involves rest, non-steroidal, antiinflammatory tablets and an accurate cortisone injection. Surgical release for intractable cases is very rarely needed, but nevertheless likely to be effective.

EPL tendon can be affected in rheumatoid arthritis. This most commonly presents with silent rupture and an inability to retropose the thumb. EIP transfer is very effective [11].

Ischaemic Rupture

EPL tendon can rupture spontaneously following a distal radius fracture. This occurs a few weeks after

the injury. Undisplaced fractures are usually affected; EPL rupture is rare after comminuted displaced fractures. The cause is likely to be ischaemia within the tight sheath, provoked by swelling [12].

Peritendonitis Crepitans

Box 2.3: Key Points: Peritendonitis Crepitans

- Provoked by unaccustomed overuse
- Usually responds to rest and steroid injection
- Surgical attention- second dorsal compartment

Pathology

This condition is sometimes known as intersection syndrome, or crossover syndrome (Fig. 2.7). It is a true tenosynovitis around the radial wrist extensor tendons within the second extensor compartment. Although the pathology was previously attributed to a frictional bursitis between the APL/EPB and ECRL/ECRB tendons, this has been revised. Grundberg and Reagan [13] found tenosynovitis in each of the 13 cases in the second extensor compartment; the symptoms were

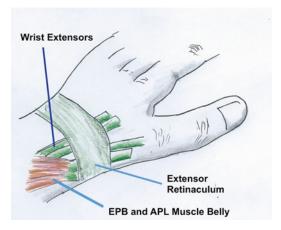


Fig. 2.7 Crossover syndrome (peritendonitis crepitans)

relieved by decompressing only the second compartment decompression alone. The explanation is that the swollen tenosynovium cannot distend the stout 2nd compartment and so presents just proximal, where the EPB and APL cross over.

Clinical Features

This condition usually follows a period of unaccustomed overuse. The patient complains of pain over the back of the distal forearm, pointing to an area proximal to that of de Quervain's. On examination there is pain, swelling and crepitus at the point where EPB and APL run obliquely across the longitudinally orientated second dorsal compartment.

Treatment

Rest, anti-inflammatory drugs and a splint can help, especially if there is an acute onset after overuse. A steroid injection can be very effective.

For persisting cases, surgery is recommended. A longitudinal incision is made over the intersection. The plane between the EPB/APL and underlying wrist extensors is developed. The 2nd dorsal compartment is opened.

Adult Trigger Thumb

Pathology and Aetiology

The pulley system in the thumb differs from the finger. There are two transverse pulleys separated by an oblique pulley. The first transverse pulley is involved in trigger thumb.

The pathology is similar to de Quervain's disease, namely primary stenosis of due to alteration of the sheath [14, 15], The nodule in the tendon is probably secondary to the constriction by the edge of the sheath, rather than primary pathology itself.

It is usually an idiosyncratic, constitutional condition. It is more common in diabetes (due to metabolic alterations in the sheath) [16]. It is also

more likely in those with amyloidosis and mucopolysaccharidoses. Several digits may be affected in these groups of patients.

It also occurs in rheumatoid arthritis, due to a thickening of the synovial sheath. Relationship to working activities is controversial. Trigger thumb is not related to occupation [17], although repetitive gripping activities may cause acute transient triggering [18].

Clinical Features

The thumb is affected less commonly than the middle and ring fingers (which are the most usually affected) [19]. The patient may present with locking of the interphalangeal joint during flexion, often more noticeable on awakening. The patient may also complain of significant pain over the front of the thumb. Sometimes the complaint is of an inability to bend the thumb properly, rather than locking.

On examination, there is a tender nodule at the opening of the sheath, directly anterior to the MP joint. Locking may or may not be demonstrable – there may just be a restriction in active flexion.

Treatment

Trigger thumb may resolve spontaneously – patients with early mild symptoms just need reassurance. The thumb can be splinted or taped straight at night. Corticosteroid injection into the tendon sheath cures around 70 %, although is less reliable in type I diabetes [16].

Surgery is almost universally effective [20]. Surgical loupes are mandatory. Under local anaesthetic infiltration, a 1 cm oblique incision is made over the skin crease in front of the MP joint. The digital nerves, which are close to the midline, are gently exposed and meticulously avoided. The thickened opening of the sheath is divided longitudinally under direct vision with a scalpel for about 8 mm. Free excursion is confirmed by asking the patient to actively flex and extend the thumb tip. Percutaneous release with a needle is an alternative [21]; the proximity of the digital nerves in the thumb, rather than the finger, demands caution.

Trigger Thumb in Children

Box 2.4: Key Points: Trigger Thumb in Children

- Acquired rather than congenital
- May resolve spontaneously by age 3
- Outcome good even if surgery delayed
- Try splinting if tolerated

Pathology and Aetiology

The cause is unknown. The term "congenital" is inappropriate, because children rarely present before the age of 6 months; no case was found amongst 4,719 newborn infants [22]. The differential diagnosis is congenital clasped thumb, in which there is absence or hypoplasia of the thumb extensors.

Clinical Features

This is the commonest surgical hand problem in a child. The parents of a child from 6 months to 3 years notice that the thumb tip is flexed, or that the child cannot bend down the thumb [23, 24]. Proper triggering is not usually reported by the parents. The thickened flexor tendon sheath is palpable at the level of the MP joint ("Notta's node").

Treatment

Trigger thumb may resolve spontaneously in nearly half of children [25–27]. Fifty per cent of thumbs responded to night splintage in extension [28, 29], with only 2 of 43 digits requiring surgery. Cortisone injection can also be effective in children, but would be confined to much older children able to tolerate the injection in an outpatient clinic. Spontaneous improvement after

Clinical Pearls

- Take great care with distal plate position (FPL) and screw length (EPL) when using a volar locking plate.
- Intra-operative dorsal skyline views during volar plate surgery should exclude dorsal screw protrusion
- Cortisone injections can solve most tendinopathies in the thumb
- Beware the hidden tendon when releasing de Quervain's
- The thumb tip will still extend after EPL rupture due to intrinsic pull through the extensor hood. Inability to put the pulp of the thumb against the dorso-radial corner of the index MP joint is the key finding.

Age 3 is uncommon and so surgery might now be considered [30]. However, the results of surgery are still good, even if delayed after 5 years of age [31]. At this age a general anaesthetic is required and exquisite surgical technique is mandatory. Percutaneous release, rather than open surgery, is an option in children [32].

Conclusions

The thumb has an array of tendons controlling its precise yet powerful function. These tendons are subject to a range of pathologies and are vulnerable to iatropathic injury. Restoration of function is usually possible with therapy, injections and surgery.

References

- Brunelli GA, Brunelli GR. Anatomy of the extensor pollicis brevis muscle. J Hand Surg Br. 1992;17B: 267–9.
- Corbin C, Warwick DJ. Midcarpal instability after excision arthroplasty for scapho-trapezial-trapezoid (STT) arthritis. J Hand Surg Eur Vol. 2009;34:537–8.
- Ippolito E, Postacchini F, Scola E, Bellocci M, De Martino C. De Quervain's disease. An ultrastructural study. Int Orthop. 1985;9:41–7.
- Clarke MT, Lyall HA, Grant JW, Matthewson M. The histopathology of de Quervain's disease. J Hand Surg Br. 1990;22B:732–4.

- Alberton GM, High WA, Shin AY, Bishop AT. Extensor triggering in de Quervain's stenosing tenosynovitis. J Hand Surg Am. 1999;24:1311–4.
- Dawson C, Mudgal CS. Staged description of the Finkelstein test. J Hand Surg Am. 2010;35:1513–5.
- Harvey FJ, Harvey PM, Horsley MW. De Quervain's disease: surgical or nonsurgical treatment. J Hand Surg Am. 1990;15A:83–7.
- Ilyas AM. Nonsurgical treatment for de Quervain's tenosynovitis. J Hand Surg Am. 2009;34:928–9.
- Peters-Veluthamaningal C, van der Windt DA, Winters JC, Meyboom-de Jong B Corticosteroid injection for de Quervain's tenosynovitis. Cochrane Database Syst Rev. 2009 :CD005616.
- Leslie BM, Ericson Jr WB, Morehead JR. Incidence of a septum within the first dorsal compartment of the wrist. J Hand Surg Am. 1990;15A:88–91.
- Magnussen PA, Harvey FJ, Tonkin MA. Extensor indicis proprius transfer for rupture of the extensor pollicis longus tendon. J Bone Joint Surg. 1990;72B:881–3.
- Hirasawa Y, Katsumi Y, Akiyoshi T, Tamai K, Tokioka T. Clinical and microangiographic studies on rupture of the EPL tendon after distal radial fractures. J Hand Surg Br. 1990;1990(15B):51–7.
- Grundberg AB, Reagan DS. Pathologic anatomy of the forearm: intersection syndrome. J Hand Surg Am. 1985;10A:299–302.
- Sampson SP, Badalamente MA, Hurst LC, Seidman J. Pathobiology of the human A1 pulley in trigger finger. J Hand Surg Am. 1991;16A:714–21.
- Boretto J, Alfie V, Donndorff A, Gallucci G, Carli P. A prospective clinical study of the A1 pulley in trigger thumbs. J Hand Surg Eur Vol. 2008;33:260–5.
- Baumgarten KM, Gerlach D, Boyer MI. Corticosteroid injection in diabetic patients with trigger finger. A prospective, randomized, controlled double-blinded study. J Bone Joint Surg. 2007;89A:2604–11.
- Trezies AJ, Lyons AR, Fielding K, Davis TR. Is occupation an aetiological factor in the development of trigger finger? J Hand Surg Br. 1998;23B:539–40.
- Giles SN, Gosling T, Hay SM. Acute transient bilateral trigger fingers. J Hand Surg Br. 1998;23A: 253–4.

- Ryzewicz M, Wolf JM. Trigger digits: principles, management, and complications. J Hand Surg Am. 2006;31A:135–47.
- Lim M-H, Lim K-K, Rasheed MZ, Narayanan S, Beng-Hoi Tan A. Outcome of open trigger digit release. J Hand Surg Eur Vol. 2007;32E:457–9.
- Gilberts EC, Beekman WH, Stevens HJ, Wereldsma JC. Prospective randomised trial of open versus percutaneous surgery for trigger digits. J Hand Surg Am. 2001;26A:497–500.
- Slakey JB, Hennrikus WL. Acquired thumb flexion contracture in children: congenital trigger thumb. J Bone Joint Surg. 1996;78B:481–3.
- Cardon LJ, Ezaki M, Carter PR. Trigger finger in children. J Hand Surg Am. 1999;24A:1156–61.
- Moon WN, Suh SW, Kim IC. Trigger digits in children. J Hand Surg Br. 2001;26B:11–2.
- Mulpruek P, Prischasuk S. Spontaneous recovery of trigger thumbs in children. J Hand Surg Br. 1998;23B: 255–7.
- Dunsmuir RA, Sherlock DA. The outcome of treatment of trigger thumb in children. J Bone Joint Surg. 2000;82B:736–8.
- Ogino T. Trigger thumb in children: current recommendations for treatment. J Hand Surg Am. 2008;33A: 982–4.
- Nemoto K, Nemoto T, Terada N, Amako M, Kawaguchi M. Splint therapy for trigger thumb and finger in children. J Hand Surg Br. 1996;21B:416–8.
- Lee ZL, Chang CH, Yang WY, Hung SS, Shih CH. Extension splint for trigger thumb in children. J Pediatr Orthop. 2006;26:785–7.
- Bae DA, Sodha S, Waters PM. Surgical treatment of the pediatric trigger finger. J Hand Surg Am. 2007; 32A:1043–7.
- Han SH, Yoon HK, Shin DE, Song DG. Trigger thumb in children: results of surgical treatment in children above 5 years of age. J Pediatr Orthop. 2010; 30:710–4.
- Sevencan A, Inan U, Köse N, Omeroğlu H, Seber S. Percutaneous release for trigger thumbs in children: improvements of the technique and results of 31 thumbs. J Pediatr Orthop. 2010;30:705–9.

Tendon Inflammation of the Fingers Including Trigger Finger

Jonathan W.G. Lohn and Andrew N.M. Fleming

Keywords

Tendinopathy • Tenosynovitis • Tenovaginitis • Crystalline arthropathy • Gout • Pseudogout • Sarcoid • Amyloid • Steroid injection • Surgery • Tendon inflammation • Trigger finger • Diabetes mellitus

Introduction

This common group of hand disorders have a diverse and often confusing nomenclature. The term tendonopathy is perhaps a better description for a group of conditions affecting the whole tendon apparatus which includes true inflammatory synovitis (also called "-vaginitis") of the tendon sheath (infective and non-infective), infiltrative tendonoses of the tendon (gout, calcific, amyloid, sarcoid – all of which may produce secondary, true inflammation) and rarely, isolated tendonitis of the tendon itself.

In the fingers the flexor sheath is more commonly affected although the extensor tendon synovial sheaths may be involved in true triggering and non-rheumatoid synovitis. Truly inflammatory, non-infective, conditions of the tendons and/or tendon sheaths of the fingers and thumb

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may affect all anatomical layers of the hand and the aetiology includes the common systemic conditions such as rheumatoid arthritis, psoriasis, gout, SLE and scleroderma. These systemic inflammatory conditions are mostly managed medically and few now need surgical attention. Diabetic cheiroarthropathy will cause similar stiff, thickened fingers with limited joint mobility, although this is due to joint contracture and non-specific thickening of all soft-tissue layers rather than a true tenosynovitis. By far the commonest cause of "inflammation" of the finger flexor sheath is trigger finger or "stenosing tenosynovitis, or -vaginitis, of the flexor sheath" to give this condition it's more flamboyant title. This is, in fact, not a true inflammatory process but a metaplasia of elements of the tendon sheath resulting in a mechanical mis-match between the smooth gliding tendon and it's constraining tendon sheath.

Whatever the aetiology (Table 3.1) of the mismatch between tendon size and the diameter of the flexor sheath, the end result is restricted tendon gliding, inflammatory signs including pain, crepitus and swelling and functional restriction in the movement of the affected finger or digits.

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 Table 3.1
 Aetiology of tendon inflammation

1. Infective	
Acute suppurative tendon sheath infection	
Chronic non-suppurative tendon sheath infection	
Mycobacterial - marinum, tuberculosis, etc.,	
Fungal	
2. Non-infective	
Idiopathic – trigger finger	
Mechanical – partial tendon injury, SOL's	
Systemic inflammatory	
Rheumatoid arthritis	
Psoriasis	
Crystalline tendonopathy	
Gout	
Pseudogout	
Calcific tendonitis	
Scleroderma	
Systemic lupus erythematosis	
Diabetic cheiroarthropathy	
Sarcoidosis	
Amyloid	

We will not discuss acute or chronic infections although mycobacterial infections, particularly, are great mimickers and must always be included in the differential diagnosis of the chronically swollen, painful digit or hand with poor function. Likewise rheumatoid, gout and psoriatic synovitis are covered elsewhere. We will, however, describe the approach to flexor synovectomy, in the rheumatoid finger as it provides the model surgical procedure for any flexor synovectomy of whatever aetiology.

Trigger Finger

Idiopathic trigger finger is an acquired condition in which the sheath of the flexor tendon of a finger (or thumb) thickens and narrows at the A1 pulley level such that the flexor tendon cannot glide freely through it. Described by Notta in 1851 [1], it is extremely common, leading to significant morbidity: pain, intermittent snapping ("triggering") or actual locking (in flexion or extension) of the affected digit. Any of the other non-infective inflammatory conditions listed in Table 3.1 may also cause triggering including

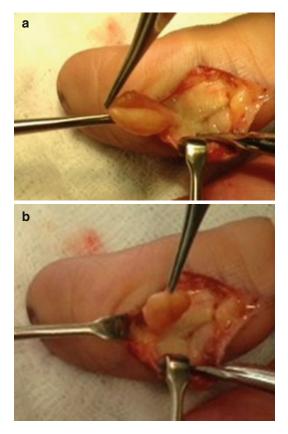


Fig. 3.1 (a) Intra-synovial lipoma of Flexor Pollicis Longus sheath and (b) post excision

intra-thecal space-occupying lesions such as partial tendon rupture or division, foreign bodies or tumours including lipoma (Fig. 3.1a, b).

Anatomy

The tendon flexor sheath is composed of a rigid retinacular pulley system and a membranous synovial portion. The retinacular pulley system acts as a physical restraint upon the flexor tendons to prevent bowstringing in flexion thereby conferring favourable biomechanics upon grip strength. It is the A1 pulley area that is usually responsible for the signs and symptoms of trigger finger and thumb. The A1 pulley is normally 8–10 mm wide and overlies the metacarpophalangeal joints. The flexor sheath apparatus begins at this point forming a tight fibro-osseous tunnel bound by the A1 pulley and a groove in the volar surface of the metacarpal neck [2].

Pathology

A study by Sampson et al. [3] compared normal and trigger A1 pulleys and they showed that the normal A1 pulley is composed of two layers: an outer convex vascular layer and an inner, concave, so-called friction layer which is avascular and allows smooth gliding. They also demonstrated that this inner, gliding surface of the A1 pulley contains chondrocytes and a matrix which has fibro-cartilaginous characteristics. Histological examination of pulleys from trigger digits revealed that the number of chondrocytes and surrounding extracellular matrix in the friction layer was significantly increased. Other studies have confirmed these histological findings at the A1 pulley in cases of triggering. They also suggest, however, that another connective tissue layer forms adjacent to the flexor tendon [4, 5] which then degenerates as the triggering progresses. The outer vascular layer also hypertrophies. Synovial cells were not observed in any pulley sample.

The pathological change in the flexor sheath is thus fibro-cartilaginous, or "chondroid", metaplasia and hypertrophy of the A1 pulley which clinically produces a tender thickening at the base of the finger in the palm. A secondary nodule, called "Notta's node" when associated with trigger thumb in children, may develop within the flexor tendons as a result of chronic impingement against the A1 pulley. This will spontaneously resolve as the A1 pulley constriction is removed.

These studies all confirm that inflammatory cells are not a component of triggering. Chondroid metaplasia is well described in other tissues in response to changed or abnormal mechanical stresses and may explain why triggering may appear a few months after carpal tunnel release [5].

Thus, the traditional term of stenosing tenosynovitis is inaccurate – in the non-rheumatoid digit, the underlying pathology of trigger finger is chondrocyte-dependent, rather than synovial cell.

Epidemiology

The overall lifetime incidence of trigger digit is 2-3 % [6]. It commonly affects the thumbs, but rarely the fingers, of young children causing a flexion deformity of the inter-phalangeal joint. It also occurs throughout adulthood, most frequently in the 5th–6th decades [7], and is then more common in women than men by a ratio of up to 6:1. The most commonly affected digit is still the thumb, followed by the ring, middle, little, and then index fingers.

Diabetes and thyroid disease are independent risk factors for trigger digit and it may also occur in association with rheumatoid arthritis, kidney disease and other rarer disorders. While occupation has been postulated as a risk factor, a study by Trezies et al. [8] looked at 178 idiopathic trigger fingers and concluded that there was no link.

Clinical Pearl

Trigger finger is said to occur in 2-3 % of the population at some stage.

It commonly occurs in the 5th to 6th decade and more frequently in women than men.

The most commonly affected digit after the thumb is the ring finger.

Presentation

Triggering may affect single digits, including the thumb, or multiple digits and the diagnosis is made on clinical grounds based on a history of locking or stiffness of the affected digit(s), tenderness in the palm of the hand at the base of the affected finger and palpable clicking or locking of the affected digit during active movement. Pain may also be referred to the dorsum of the digit. These symptoms are commonly worse in the early morning and can lead to significant disability.

Differential diagnoses include Dupuytren's contracture, post-traumatic joint contracture or intermittent locking of the metacarpophalangeal joint following either sagittal band attrition or rupture or a loose body within the joint.

Grade	
1. Pretriggering:	Pain; history of catching; no clinical signs; tender over A1 pulley
2. Active:	Demonstrable catching; active extension still present
3. Passive:	
3a	Demonstrable catching requiring passive extension
3b	Inability to actively flex
4. Contracture:	Demonstrable catching with fixed flexion deformity of the PIP joint

 Table 3.2
 Grading of Trigger finger severity – Wolfe [7]

Severity of Trigger Finger

This has been classified into Types 1–4 [7] (Table 3.2). Wolfe, however, states that this grading system has been shown to have no therapeutic or measurable outcome benefit but merely provides a means of recording the severity of triggering [7].

Treatment Options and Outcomes

Conservative Treatment

Spontaneous without recovery splinting occurred in 83 % of thumbs studied by Schofield et al. [9], but only after an average of 7 months. Conservative treatment with nocturnal splinting of the metacarpophalangeal joint of the long digits has also been used with reported success rates of 66 % after 6 weeks of splintage with a minimum review at 1 year [10], whilst others are less optimistic quoting 20 % recovery [6]. There is no good evidence to suggest that work-place modification (ergonomic adjustments) or hand physiotherapy help in the management of trigger finger. The apparent difference in natural history between triggering in the thumb and fingers has no explanation.

Steroid Injection is an effective treatment for trigger finger and thumb. In 1953, Howard et al. [11] described 12 cases of trigger finger treated with injection of steroid into the flexor sheath, with resolution of the condition in 11. Subsequent studies, however, revealed a wider variation in response rates. Rather surprisingly there appears to be no difference in response between an

intra-sheath injection and one into the subcutaneous tissues of the hand [12]. In 1995, Lambert et al. [13] published their results from a controlled double-blind prospective study of the injection of methyl-prednisolone and local anaesthetic versus an injection of local anaesthetic alone for the treatment of idiopathic trigger finger. They showed a 60 % success rate for the steroid injection against 16 % for local anaesthetic used alone. A further Level 1 study supports this outcome [14].

Marks and Gunther used triamcinolone in a large, prospective study with a 44 month followup [15]. They showed slightly better outcomes in females and an overall response rate of 84 % with one injection and 91 % with a second. Newport et al. undertook a large retrospective review of 338 digits [16]. They showed a response in 49 % after one injection, 72 % after a second and 77 % after a third. They concluded that there were certain markers for a favourable prognosis: specifically single digit involvement and less than 6 months duration of symptoms.

There is some evidence that insoluble Triamcinolone may have a more rapid effect than soluble Dexamethasone with a slightly less durable effect at 3 months [17], Overall, however, steroid injections have few complications and are safely given in the outpatient clinic setting [18].

Some studies have excluded patients with diabetes mellitus [14]. While the natural history of trigger finger in diabetes is unclear, it is established that the prevalence is much higher -23 % in type I and 16 % in type II [19]. The response rates to steroid injection in patients with diabetes is also lower than in those with no risk factors for trigger digit. However up to 60 % of diabetic patients are successfully treated with steroid injections, though the success rate is lower if multiple digits are involved. There is no evidence that simple sheath dilatation with saline or lignocaine offers any therapeutic benefit [20, 21].

Sato et al. [22] in a prospective trial of 150 trigger digits allocated three groups of 50 patients randomly to either methylprednisolone injection, percutaneous A1 release or surgical release. Cure rates at 6 months were 86 % in the steroid group (up to 2 injections allowed) and 100 % in both the surgical groups with no complications in any group. Despite the evidence for non-surgical treatment of trigger digits, a recent Cochrane review [23] concluded that the effectiveness of steroid injections were "only studied in two small randomised controlled trials of poor methodology". It concludes, however, that steroid injection is a safe treatment that may last for up to 4 months.

Clinical Pearl

The diagnosis of triggering is made on clinical grounds.

- The results of conservative treatment, that is splinting, can result in a resolution of symptoms.
- The most effective conservative treatment is local anaesthetic and steroid injection.

Surgery

Surgical Release of the A1 Pulley is indicated for the following reasons:

- · patient choice
- failed conservative treatment
- for recurrent triggering after one to two injections of steroid
- if there are severe symptoms at presentation locked finger
- in diabetics who are unlikely to benefit from steroid injections – multiple digits and severe symptoms

Surgical division of the A1 annular pulley of the flexor sheath of the digit, either by conventional open, or percutaneous, release is an effective treatment with a high success rate, minimal complication and short recovery period. It is usually done as a day-case under local anaesthetic and often provides a permanent cure when performed by an appropriately trained surgeon.

Percutaneous trigger finger release has been undertaken for nearly two decades and is a well established, safe, office-based treatment [22, 24–27]. A variety of needles, hooks and micro-knives have been described to achieve the same result – percutaneous division of the A1 pulley under local anaesthetic control. This technique is generally not used in the thumb and index finger due to the close proximity of the digital nerves to the flexor sheath. **Procedure**: the point of triggering is confirmed clinically and marked; the metacarpo-phalangeal joint is hyper-extended to retract the nerves dorsally and, under local anaesthetic, a 19 or 21 gauge needle is inserted centrally into the tendon and its position confirmed by asking the patient to gently flex the finger. The bevel of the needle is then aligned with the tendon and the tip of the needle is withdrawn from the tendon substance and then swept back and forth longitudinally scoring the A1 pulley with the tip until division of the pulley is confirmed. A second needle placement is sometimes necessary to completely divide the pulley. Active flexion by the patient then confirms a full release.

The safety of this technique has been established in both cadaveric studies [25] and case series [22, 26, 27] showing it to be a good officebased alternative to open surgical release with few reported complications.

Open surgery is usually carried out under local anaesthesia with a pneumatic arm tourniquet in situ. Local anaesthesia is infiltrated subcutaneously, usually via the thinner web skin or from dorsally, into the palm over the flexor sheath. An incision is then made over the proximal A1 pulley. This incision can be transverse the author's preferred method, a chevron or axial. Neurovascular bundles are protected as longitudinal spreading dissection down to the flexor sheath proceeds. The A1 pulley is released axially and completely using a knife and taking care not to release the A2 pulley. The patient is then requested to actively flex the finger to demonstrate full active range of movement. Some authors suggest releasing the sheath on the radial side, leaving an ulnar cuff of A1 pulley, to prevent subsequent ulnar translation of the tendons. The tourniquet is then deflated, haemostasis achieved and the skin closed with absorbable interrupted suture material. Any dressing should be light to permit immediate mobilisation of the fingers. All patients should be taught active and blocked active mobilisation exercises.

Complications

There have been no reported vascular or nerve injuries with corticosteroid injection. It is a safe treatment for trigger digit [23] although some patients will find it painful. We commonly infiltrate local anaesthetic prior to our steroid injection. The commonest error is to deliver the steroid subcutaneously and not directly into the flexor sheath although the evidence suggests that this does not seem to affect the response rate [12]. Cutaneous hypo-pigmentation is not usually a problem at this level.

Percutaneous trigger digit release appears to have a low complication rate. Eastwood et al. describe a 94 % response rate with no adverse incidents [24]. In another study, pain was significantly lower when compared to steroid injection and no major complications were described [28]. Ragoowansi et al. [29] in a series of 180 cases had 10 patients with recurrent triggering at 3 months; all of these were treated by open release. However, they had no cases of tendon or nerve injury.

Nerve injury, both traction neuropraxia and neurotmesis are rare complications of open trigger release. Neuropraxia will settle within weeks. Nerve division necessitates immediate repair and will usually lead to an incomplete recovery. Bowstringing of the tendon may occur with overenthusiastic division of the pulley system, particularly if it includes the proximal A2 pulley. This may ultimately require pulley reconstruction. Incomplete release will also lead to incomplete resolution or recurrence of symptoms.

A single surgeon series by Will et al. [30] reviewed 78 trigger finger releases in 43 patients and showed only two major complications - a synovial fistula requiring re-operation and an arthro-fibrosis requiring serial splintage. Otherwise, there was minor infection and scar tenderness in four patients. There was no nerve injury, no bowstringing and no recurrence. In another series by Lange-Riess et al. [31] looking at long term follow up of 254 trigger finger releases, 249 regained full range of movement on-table (the remaining 2 cases had had long standing pathology). They had nine perioperative complications - two superficial infections, six neuropraxias of a digital nerve and one instance of delayed wound healing. No bowstringing or recurrence was seen.

Clinical Pearl

- Surgical release is indicated in cases of failed conservative treatment, a locked finger, or in diabetic multiple joint involvement. Currently there is no reported difference in
- outcome between percutaneous trigger finger release and open surgery. Complications are uncommon.

Personal View

In our practice, a corticosteroid (typically 10 mg Triamcinolone) injection in the office or clinic, under local anaesthetic, is the first line of treatment in almost all cases, the exceptions being patient choice, the diabetic with multiple digit involvement and the longstanding stuck trigger digit that is unlikely to resolve with conservative management. If this first injection fails or there is early recurrence (less than 2 months) then most patients will receive a further injection. If this fails, then surgery is the next step and this is performed in an ambulatory day-case setting. In our practice, we close using a 5/0 dissolving polyglactin 910 suture (Vicryl Rapide, Ethicon Inc.). This enables the patients to avoid suture removal. They remove their own dressing at 7 days and thereafter use and bathe the hand normally. They are not routinely followed up if this is an isolated pathology. Formal hand therapy including dynamic splinting is only indicated in cases of long-standing contracture.

Trigger Finger in Children

This is much less common than paediatric trigger thumb and the aetiology is different both from trigger thumb in children and trigger finger in the adult [32]. Suggested aetiologies include aberrant tendon connections and FDS decussation, intra-tendinous nodules, abnormal tendon insertions, storage disorders and simple idiopathic triggering. Whilst idiopathic trigger finger may occur, high recurrence rates after simple A1 pulley release suggest that a different clinical approach needs to be employed [33].

Clinical Presentation

The usual presentation is that of a finger flexed at the proximal inter-phalangeal joint level, which is commonly attributed to trauma by the parents. This may have been present for a long time, even since birth. Important differential diagnoses include: "failure of differentiation of parts", part of a generalised athrogryposis picture [32] – typically presenting as a thumb-inpalm deformity - or a more localised condition such as camptodactyly, paediatric fibromatoses and true post-traumatic finger contracture. Close examination for the absence of flexor and/or extensor creases will often provide the clue to the congenital nature of a flexed finger. In the presence of normal flexure creases and where no subcutaneous nodularity is palpable or any history of trauma identified then it can be assumed that a true trigger exists.

Investigation

Rreal-time ultrasound investigation is the most paediatric friendly, non-invasive procedure to assist with the diagnosis of the flexed finger in childhood although more difficult in a recalcitrant child with a locked finger.

Treatment

This is directed at the cause, although injection of steroid is not indicated in the paediatric population. Surgical exploration of the finger should be performed under general anaesthetic with loupe magnification. Initial manipulation indicates whether the flexion deformity is unlockable and palpation may reveal an obvious abnormality. We will then almost invariably use a longitudinal chevron or zig-zag incision which allows sequential extension down the finger. After initial A1 pulley release further exploration of the flexor sheath, proceeding distally, is undertaken with exposure of any anatomical abnormalities. These are dealt with until a full passive range of tendon gliding is demonstrable [33]. Rarely, resection of slip(s) of the FDS tendon may be necessary.

Extensor Tendon Triggering

If the more common De Quervain's tenosynovitis and Rheumatoid synovitis cases are excluded, then this is a rare condition compared to triggering of the flexor tendons and typically occurs at the level of the distal extensor retinaculum [34, 35]. Clinical presentation is with dorsal pain and swelling, snapping and a sensation of incomplete extension. Trauma is implicated in the aetiology [36] and the usual treatment is surgical exploration and either debridement of a frayed tendon, removal of intratendinous ganglion or synovectomy.

Crystalline Tendonopathy

Our intention here is to dwell briefly on the crystalline tendonopathies as these are seldom seen in isolation.

Bullocks et al [37]. described two processes occurring in the patient presenting with crystalline tenosynovitis: firstly the physical deposition of crystals and subsequently an inflammatory reaction within the synovium. This inflammation is secondary to phagocytosis of the deposited crystals. The inflammatory mediators released then activate the well known pathways that lead to swelling, pain and erythema. This can make crystalline tenosynovitis at least clinically very difficult to distinguish from acute infection [37].

Gout

Gout is characterised by the deposition of urate crystals in multiple sites in the body including joints and tendons. Gout is caused by an excess of serum urate – hyperuricemia – due to excess ingestion or production or insufficient secretion. Gouty tophi are a late complication of



Fig. 3.2 (a) Painful gouty tophi of middle finger distal inter-phalangeal joint (b) exposure via dorsal "H" flaps and (c) post debridement around extensor tendon and joint

uncontrolled hyperuricemia and consist of collections of monosodium urate crystals in the soft tissues. It is rare for tophi to occur without a previous history of acute gout [38].

Signs of flexor tendon involvement include pain, erythema and swelling and the differential diagnosis must include acute suppurative infection of the flexor sheath [39, 40]. Tophaceous deposits may also occur around the metacarpophalangeal and interphalangeal joints of the finger. Both computed tomography and magnetic resonance imaging scans have been used to show local tophaceous involvement of the tenosynovium [41].

If neglected, intra-tendinous infiltration may occur along with contractures, skin ulceration and superficial infection leading to infective arthitis. While the majority of cases of gout can be controlled medically, surgery is indicated for topahecous gout where infection and ulceration occur, to improve function by debulking and for removal of painful tophi (Fig. 3.2a–c). With extensive flexor or extensor tendon involvement sequential, longitudinal exposure of the tendon(s) with synovectomy (Fig. 3.3a–c), intratendinous debulking and even excision of a slip of the FDS tendon plus release of A1 pulley may lead to improved pain-free range of movement.

Pseudogout

Pseudogout is a clinical term used to describe a peri-articular inflammatory process, which resembles the urate deposition of true gout. This disease, also known as chondrocalcinosis or calcium pyrophosphate deposition disease (CPDD), is characterised by calcium pyrophosphate deposition and rarely involves the hand. It may also be associated with haemochromatosis and hyperparathyroidism [42]. It should, however, be suspected whenever a calcified soft tissue mass is identified clinically

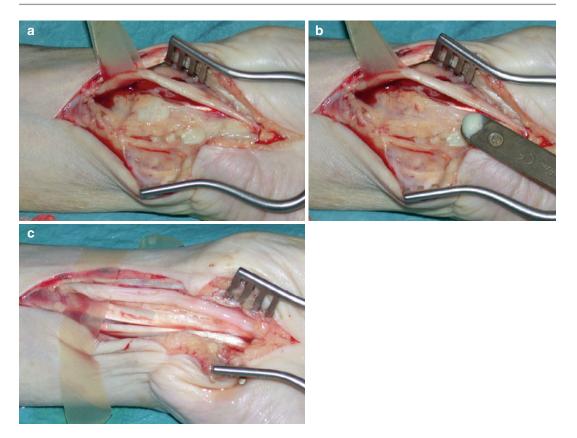


Fig. 3.3 (a) Zone 5 gouty synovitis (b) gouty material (c) post debridement

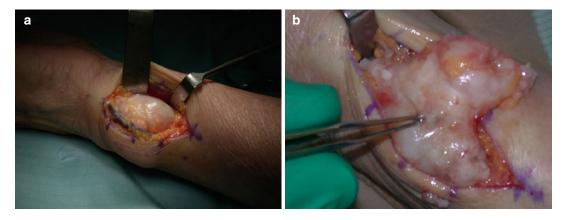


Fig. 3.4 Synovial chondrocalcinosis (a) initial exposure in Zone 5 and (b) calcified material prior to full excision

(Fig. 3.4a, b) or radiologically as a granular or fluffy appearance on Xray. The crucial clinical and pathological differentiation from cartilage tumours is made histologically by the presence of birefringent crystals. Whilst there is little specific in the literature describing surgical treatment for infiltration by CPDD of the flexor sheath [37] basic principles of debulking, syn-ovectomy and/or mass removal also apply to these deposits in the hand.

Acute Calcific Tendonitis (ACT)

Non-specific calcific infiltration of the tendon sheath or tendon is an idiopathic cause of an acute inflammatory synovitis which again must be differentiated from a purulent synovitis. Isolated ACT is rare but can occur in tendons around the wrist, especially FCU around the pisiform, and within the carpal tunnel [43]. It may represent either a primary infiltration or secondary to extension from an underlying joint. Radiology may reveal the diagnosis where heterotopic, fluffy calcification is seen within the soft tissues and again emphasizes the importance of plain radiography even with an apparently "obvious" soft tissue infection. Differentiation from purulent synovitis is made by the absence of systemic inflammatory signs and relevant laboratory tests and may be confirmed by the needle aspiration of non-purulent, calcific material from the sheath [44].

Medical treatment with rest, non-steroidal inflammatory medication, splintage and sometimes steroid injection will almost invariably reverse the acute inflammatory episode and surgery is seldom indicated.

Storage Diseases

Sarcoid

This systemic disease may rarely present to the hand surgeon with either a chronic tenosynovitis, nodular cutaneous involvement, osteolytic bone lesions or a synovial mass [45, 46]. Any hand involvement may unusually be the first presentation and appropriate biopsy and/or synovectomy may lead to the diagnosis and onward referral to Rheumatology.

Amyloid

Amyloidosis may present as a synovial infiltrate in either the primary form or, more commonly, the secondary form in renal dialysis patients where it may cause carpal tunnel syndrome [47]. Thick plaque-like lesions infiltrate the synovium and tendons and may even produce tendon rupture. It is rarely inflammatory in nature but presents with symptoms typical of carpal tunnel syndrome, triggering, chronic swelling and restricted movement. Surgery for Amyloid usually involves either an extended carpal tunnel release and synovectomy and/or synovectomy of the flexor sheaths along a digit(s).

Diabetic Hand

Whilst this is not a storage disease, hand pathology is quite common in diabetics. The precise mechanism of why diabetes causes many of these conditions remains unclear. Included among them, however, are stenosing tenosynovitides, carpal tunnel syndrome, infection, Dupuytren's disease and the "diabetic stiff hand" or diabetic cheiroarthropathy [48–50].

Many of these pathologies are discussed elsewhere. However, the diabetic stiff hand is a great mimicker and it is obviously important to distinguish this from more easily treatable conditions such as trigger finger and Dupuytren's contracture. Synovectomy of the idiopathic diabetic stiff hand, without trigger finger, is fraught with difficulty and often results in a poor outcome.

The condition itself manifests clinically with steadily increasing loss of both flexion and extension with palpable thickening of the soft tissues. The ulnar fingers are often affected first. The precise aetiology is unknown, but typically occurs in long-standing disease, often juvenile onset [49]. Added to that, there is usually evidence of microvascular disease elsewhere [50].

Steroid injection may well improve range of movement in these fingers acutely but can lead to problems with diabetic control and early relapse.

Technique of Flexor Synovectomy

The typical clinical presentation of flexor synovitis, of whatever cause, is of a swollen, often tender digit (or boggy swelling cross the wrist) with reduced active range of movement. Palpation will reveal increased "bogginess" of the flexor

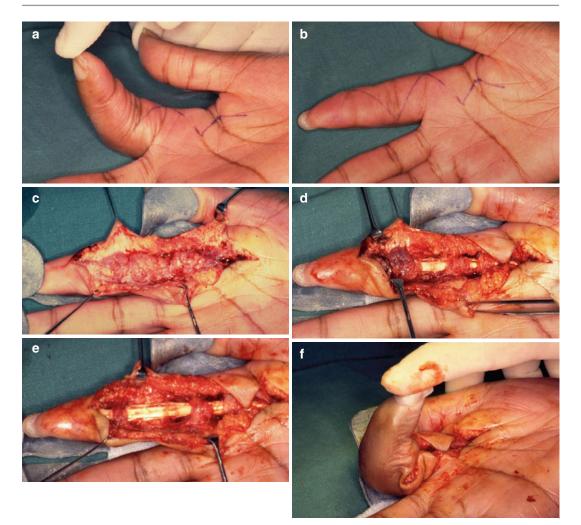


Fig. 3.5 Rheumatoid flexor synovitis (**a**) showing reduced passive range of motion (**b**) incision (**c**) initial exposure with bulging synovium and (**d**) partially cleared

synovium (e) fully cleared synovium with only pulleys left on flexor tendons (f) improved passive range of motion

sheath when palpated across the volar aspect of the proximal phalanx. Passive range of movement is greater than active range but often painful (Fig. 3.5a).

Whilst the underlying diagnosis is often known e.g. rheumatoid, psoriatic, or gouty tendonopathy, supportive ultrasound and/or MRI scans may corroborate the clinical findings and delineate the extent of the synovitis. Plain radiography will also determine bone and/or joint involvement. Not uncommonly idiopathic synovitis of an isolated digit may be secondary to extension of the inflammatory process from an underlying osteoarthritic joint. Medical management is the first line of treatment for most synovitis and most referrals for surgical management will come from one's Rheumatology colleagues.

Surgery

This is usually performed within an ambulatory day-case setting under regional anaesthesia and tourniquet using loupe magnification. The affected digit(s) is approached via a volar zig-zag incision (Fig. 3.5b) with full exposure of the flexor sheath (Fig. 3.5c) from Zone 3–1 (and proximally across the carpal tunnel if need be).

The fleshy, exuberant synovial thickening will then be obvious bulging through the membranous pulleys between the fibrous pulley system (Fig. 3.5c). Synovectomy is then performed by a combination of sharp dissection and traction with bone rongeurs to clear the entire sheath of macroscopic synovium (Fig. 3.5d, e). Once complete, traction on the proximal flexor tendon should demonstrate an improved passive range of movement of the entire digit (Fig. 3.5f). Following haemostasis, suture placement and light dressings early hand therapy is commenced.

Summary

Trigger finger is one of the commoner conditions affecting the hand. It is more common in females, particularly in the 5th and 6th decade of life. In the majority of cases, the exact aetiology remains unknown. However, it can be associated with a number of systemic conditions, for example, diabetes. A diagnosis is made solely on clinical grounds, that is by history and examination. Rarely is further investigation required. Conservative treatment can often be successful. However, most patients are treated by an injection of local anaesthetic and steroid into the flexor sheath. This often results in significant improvement. Surgery is reserved for the persistent or recurrent cases, where there is a locked digit, or more complex cases. The results of surgery are often excellent with a low recurrence rate and few reported complications.

References

- Notta A. Research about a particular affection of the tendons of the hand, characterized by the development of a nodosity on the trajectory of the flexor tendons of the digits that impedes their movements. Archives generales de medecine. 1850;4:142–61.
- Fiorini HJ, Santos JB, Hirakawa CK, Sato ES, Faloppa F, Albertoni WM. Anatomical study of the A1 pulley: length and location by means of cutaneous landmarks on the palmar surface. J Hand Surg Am. 2011;36(3):464–8. Epub 2011 Jan 31.
- Sampson SP, Badalamente MA, Hurst LC, Seidman J. Pathobiology of the human A1 pulley in trigger finger. J Hand Surg Am. 1991;16(4):714–21.

- Sbernardori MC, Bandiera P. Histopathology of the A1 pulley in adult trigger fingers. J Hand Surg Eur Vol. 2007;32(5):556–9. Epub 2007 Aug 7.
- Drossos K, Remmelink M, Nagy N, de Maertelaer V, Pasteels JL, Schuind F. Correlations between clinical presentations of adult trigger digits and histologic aspects of the A1 pulley. J Hand Surg Am. 2009; 34(8):1429–35. Epub 2009 Aug 20.
- Moore JS. Flexor tendon entrapment of the digits (trigger finger and trigger thumb). J Occup Environ Med. 2000;42(5):526–45. Review.
- Wolfe SW. Tenosynovitis, Chapter 60. In: Green DP, Hotchkiss RN, Pederson WC, Wolfe SW, editors. Green's operative hand surgery. Philadelphia: Elsevier/Churchill Livingstone; 2005. p. 2137–58.
- Trezies AJ, Lyons AR, Fielding K, Davis TR. Is occupation an aetiological factor in the development of trigger finger? J Hand Surg Br. 1998;23(4):539–40.
- Schofield CB, Citron ND. The natural history of adult trigger thumb. J Hand Surg Br. 1993;18(2):247–8.
- Patel MR, Bassini L. Trigger fingers and thumb: when to splint, inject, or operate. J Hand Surg Am. 1992; 17(1):110–3.
- Howard Jr LD, Pratt DR, Bunnell S. The use of compound F (hydrocortone) in operative and nonoperative conditions of the hand. J Bone Joint Surg Am. 1953;35-A(4):994–1002.
- Taras JS, Raphael JS, Pan WT, Movagharnia F, Sotereanos DG. Corticosteroid injections for trigger digits: is intrasheath injection necessary? J Hand Surg Am. 1998;23(4):717–22.
- Lambert MA, Morton RJ, Sloan JP. Controlled study of the use of local steroid injection in the treatment of trigger finger and thumb. J Hand Surg Br. 1992; 17(1):69–70.
- Murphy D, Failla JM, Koniuch MP. Steroid versus placebo injection for trigger finger. J Hand Surg Am. 1995;20(4):628–31. Erratum in: J Hand Surg [Am] 1995 Nov;20(6):1075. Comment in: J Hand Surg [Am]. 1996 May;21(3):530.
- Marks MR, Gunther SF. Efficacy of cortisone injection in treatment of trigger fingers and thumbs. J Hand Surg Am. 1989;14(4):722–7.
- Newport ML, Lane LB, Stuchin SA. Treatment of trigger finger by steroid injection. J Hand Surg Am. 1990;15(5):748–50.
- Ring D, Lozano-Calderón S, Shin R, Bastian P, Mudgal C, Jupiter J. A prospective randomized controlled trial of injection of dexamethasone versus triamcinolone for idiopathic trigger finger. J Hand Surg Am. 2008;33(4):516–22; discussion 523–4.
- Peters-Veluthamaningal C, Winters JC, Groenier KH, Jong BM. Corticosteroid injections effective for trigger finger in adults in general practice: a doubleblinded randomised placebo controlled trial. Ann Rheum Dis. 2008;67(9):1262–6. Epub 2008 Jan 7.
- Chammas M, Bousquet P, Renard E, Poirier JL, Jaffiol C, Allieu Y. Dupuytren's disease, carpal tunnel syndrome, trigger finger, and diabetes mellitus. J Hand Surg Am. 1995;20(1):109–14.

- Akhtar S, Burke FD. Study to outline the efficacy and illustrate techniques for steroid injection for trigger finger and thumb. Postgrad Med J. 2006;82(973): 763–6. Review.
- Baumgarten KM, Gerlach D, Boyer MI. Corticosteroid injection in diabetic patients with trigger finger. A prospective, randomized, controlled double-blinded study. J Bone Joint Surg Am. 2007;89(12):2604–11.
- 22. Sato ES, Gomes Dos Santos JB, Belloti JC, Albertoni WM, Faloppa F. Treatment of trigger finger: randomized clinical trial comparing the methods of corticosteroid injection, percutaneous release and open surgery. Rheumatology (Oxford). 2012;51(1): 93–9. Epub 2011 Oct 29.
- Peters-Veluthamaningal C, van der Windt DA, Winters JC, Meyboom-de Jong B. Corticosteroid injection for trigger finger in adults. Cochrane Database Syst Rev. 2009;(1):CD005617.
- Eastwood DM, Gupta KJ, Johnson DP. Percutaneous release of the trigger finger: an office procedure. J Hand Surg Am. 1992;17(1):114–7.
- Pope DF, Wolfe SW. Safety and efficacy of percutaneous trigger finger release. J Hand Surg Am. 1995; 20(2):280–3.
- 26. Fu YC, Huang PJ, Tien YC, Lu YM, Fu HH, Lin GT. Revision of incompletely released trigger fingers by percutaneous release: results and complitions. J Hand Surg Am. 2006;31(8):1288–91.
- 27. Tanaka J. Percutaneous trigger finger release. Tech Hand Up Extrem Surg. 1999;3(1):52–7.
- Zyluk A, Jagielski G. Percutaneous A1 pulley release vs steroid injection for trigger digit: the results of a prospective, randomized trial. J Hand Surg Eur Vol. 2011;36(1):53–6. Epub 2010 Aug 13.
- Ragoowansi R, Acornley A, Khoo CT. Percutaneous trigger finger release: the 'lift-cut' technique. Br J Plast Surg. 2005;58(6):817–21.
- Will R, Lubahn J. Complications of open trigger finger release. J Hand Surg Am. 2010;35(4):594–6. Epub 2010 Feb 26.
- Lange-Riess D, Schuh R, Hönle W, Schuh A. Longterm results of surgical release of trigger finger and trigger thumb in adults. Arch Orthop Trauma Surg. 2009;129(12):1617–9. Epub 2009 Jan 6.
- Ty JM, James MA. Failure of differentiation: part II (arthrogryposis, camptodactyly, clinodactyly, madelung deformity, trigger finger, and trigger thumb). Hand Clin. 2009;25(2):195–213.
- Schaverien MV, Godwin Y. Paediatric trigger finger: literature review and management algorithm. J Plast Reconstr Aesthet Surg. 2011;64(5):623–31. Epub 2010 Oct 20.
- Khazzam M, Patillo D, Gainor BJ. Extensor tendon triggering by impingement on the extensor retinaculum: a report of 5 cases. J Hand Surg Am. 2008;33(8):1397–400.

- Kardashian G, Vara AD, Miller SJ, Miki RA, Jose J. Stenosing synovitis of the extensor pollicis longus tendon. J Hand Surg Am. 2011;36(6):1035–8.
- Cooper HJ, Shevchuk MM, Li X, Yang SS. Proliferative extensor tenosynovitis of the wrist in the absence of rheumatoid arthritis. J Hand Surg Am. 2009;34(10): 1827–31.
- Bullocks JM, Downey CR, Gibler DP, Netscher DT. Crystal deposition disease masquerading as proliferative tenosynovitis and its associated sequelae. Ann Plast Surg. 2009;62(2):128–33.
- Pittman JR, Bross MH. Diagnosis and management of gout. Am Fam Physician. 1999;59(7):1799–806, 1810.
- Moore JR, Weiland AJ. Gouty tenosynovitis in the hand. J Hand Surg Am. 1985;10(2):291–5.
- Abrahamsson SO. Gouty tenosynovitis simulating an infection. A case report. Acta Orthop Scand. 1987; 58(3):282–3.
- 41. Chen CK, Chung CB, Yeh L, Pan HB, Yang CF, Lai PH, Liang HL, Resnick D. Carpal tunnel syndrome caused by tophaceous gout: CT and MR imaging features in 20 patients. AJR Am J Roentgenol. 2000;175(3):655–9.
- Jensen PS. Chondrocalcinosis and other calcifications. Radiol Clin North Am. 1988;26(6):1315–25.
- Moyer RA, Bush DC, Harrington TM. Acute calcific tendinitis of the hand and wrist: a report of 12 cases and a review of the literature. J Rheumatol. 1989; 16(2):198–202.
- McAuliffe JA. Tendon disorders of the hand and wrist. J Hand Surg Am. 2010;35(5):846–53; quiz 853.
- Wang HT, Sunil TM, Kleinert HE. Multiple unusual complications after extensive chronic sarcoid tenosynovitis of the hand: a case report. J Hand Surg Am. 2005;30(3):610–4.
- Fodor L, Bota IO, Fodor M, Ciuce C. Sarcoid flexor tenosynovitis as a single early manifestation of the disease. J Plast Reconstr Aesthet Surg. 2012;65(8): e217–9. Epub 2012 Apr 1.
- Wilson SW, Pollard RE, Lees VC. Management of carpal tunnel syndrome in renal dialysis patients using an extended carpal tunnel release procedure. J Plast Reconstr Aesthet Surg. 2008;61(9):1090–4. Epub 2007 Sep 21.
- Fitzgibbons PG, Weiss AP. Hand manifestations of diabetes mellitus. J Hand Surg Am. 2008;33(5): 771–5.
- Ceruso M, Lauri G, Bufalini C, Bartolozzi G, Bernardini S, Cinti S, Morroni M, Matucci-Cerinic M. Diabetic hand syndrome. J Hand Surg Am. 1988; 13(5):765–70.
- Brown E, Genoway KA. Impact of diabetes on outcomes in hand surgery. J Hand Surg Am. 2011; 36(12):2067–72.

Osteoarthritis of the Wrist and DRUJ

Alberto Lluch

Keywords

Wrist joint • Distal radio-ulnar joint • Scapho-trapezium-trapezoid joint • Degenerative arthritis • SLAC wrist • SNAC wrist • SMAC wrist • Proximal row carpectomy • Scaphoid resection • Midcarpal fusion • Radio-scapholunate arthrodesis • Sauvé-Kapandji procedure

Introduction

In comparison to other joints, such as the spine, hips or knees, primary degenerative arthritis of the wrist is relatively uncommon. Inflammatory arthritis of the wrist is frequently observed in patients suffering from rheumatoid arthritis or crystalline arthropathies. Eventually, crystal deposits in the wrist will lead to degenerative changes. Deposition of urate and calcium pyrophosphate dehydrate (CPPD) material in the synovial tissue are the most common problems affecting the wrist, producing gouty and pseudogout arthritis respectively.

Although primary degenerative arthritis of the wrist is rare, cartilage wear is frequently seen during postmortem examinations of the wrist joints in elderly people. It is not diagnosed radiographically because osteophyte formation is rare in the carpal joints. It is also clinically silent because these patients place less strain on their wrists as they get older, in comparison to weight bearing joints such as the hips and knees.

Degenerative arthritis or arthrosis of the wrist is generally observed around the scaphoid bone. Apart from scapho-trapezium-trapezoid arthritis, as its cause is not yet fully known, the rest are secondary to fractures or subluxations of the scaphoid. The scaphoid bone links the proximal and distal carpal rows, providing stability to the midcarpal joint. When the scapho-lunate ligaments are ruptured or the scaphoid is fractured, joint misalignment will occur, causing wear of the affected joints.

SLAC Wrist

In 1984, Watson and Ballet [1] reviewed more than 4,000 wrist radiographs and found degenerative arthritis in 210 cases (5.25 %), and, in the majority of them, the joints around the scaphoid bone were involved. Most patients presented a pattern of degenerative arthritis from ruptures of

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the scapho-lunate ligaments and they coined the term "SLAC wrist" [2]. This acronym stands for Scapho-Lunate Advanced Collapse. Four years earlier, Watson and Hempston [2] had already proposed to perform a scapho-trapezium-trapezoid ("triscaphoid joint") arthrodesis for the treatment of "rotary subluxation of the scaphoid".

In the lateral radiographic view of a normal wrist, the scaphoid is seen in about 47° of flexion (ranging from 30° to 60°) in relation to the longitudinal axis of the radius. When grasping objects, the forces transmitted through the distal carpal row will add a flexion torque to the scaphoid. Further flexion of the scaphoid is prevented by its distal ligament insertions to the trapezium and trapezoid and the proximal ligament insertions to the lunate. When the scapho-lunate ligaments are ruptured from injury, flexion of the scaphoid increases, causing a dorsal subluxation of its proximal pole. This type of lesion has been named rotary instability, rotatory instability or flexion instability of the scaphoid. The terms rotary and rotator are not very adequate as they refer to rotation around an axis, which is not this case. Flexion instability of the scaphoid describes the deformity, but scapho-lunate dissociation is probably more appropriate as it defines the lesion better.

In normal circumstances, the lunate is collinear with the radius and the capitate. The normal alignment of the lunate is maintained by the length of the scaphoid and the intact scapholunate ligaments, which keeps separated from the radius the distal carpal row. When the scapholunate ligaments are ruptured, the scaphoid subluxes dorsally in relation to the radius, and the radial column of the carpus, formed by the trapezium, the trapezoid and the scaphoid becomes shorter. As a consequence, the compression forces through the central column, formed by the capitate and the lunate, will be increased. When the lunate is compressed between the capitate and the radius it always goes into extension. This can be explained because its dorsal horn is thinner than the volar one, and the joint surface of the distal radius is volarly angulated about 11° (Fig. 4.1). The previously described morphology of the distal radius and the lunate explains why it is easier for the lunate to extend rather than flex. RELATION CALIFICATION CALIFICAT

Fig. 4.1 MRI of the central column of a normal carpus in the sagittal plane. Under axial compression, the lunate will always go into extension, due to the volar inclination of the distal radius and the fact that the dorsal horn of the lunate is thinner than the volar one. This will not occur if the scaphoid maintains its normal length and the scapholunate ligaments are intact

Consequently, wrist extension takes place primarily at the radio-lunate joint, while flexion is greater at the midcarpal joint [3].

Clinical Diagnosis

Some SLAC wrists are asymptomatic for many years, and the same has been observed for some scaphoid non-unions [4, 5]. In these cases, pain frequently starts after minor trauma, and it is usually localized at the dorsal radio-scaphoid joint. Wrist mobility will be moderately diminished, particularly on extension, as it can only do so at the midcarpal joint, because the radio-lunate joint is already in extension. The arc of wrist flexion is preserved, as both the radio-lunate and the midcarpal joint are able to flex.

Radiological Diagnosis

Originally, the radiographic degenerative changes observed in the SLAC wrist have been classified in three stages, although more recent publications have added two more stages [6, 7]. We, therefore propose a classification in five stages, following a topographical sequence of joint degeneration:

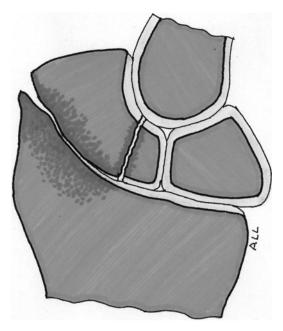


Fig. 4.2 Schematic drawing of a SNAC wrist in stage I. Degenerative arthritis between the radius and the distal fragment of the scaphoid is seen. In cases of SLAC wrist in stage I, the degenerative arthritis will be seen between the radius and the proximal pole of the scaphoid

radio-scaphoid, scapho-capitate, luno-capitate, triqueto-hamate and radio-lunate.

Stage I. The proximal scaphoid is not constraint with the distal radius, has a smaller radius of curvature, and therefore will have a tendency to sublux dorsally when the scapho-lunate ligaments are ruptured. This will cause an increase of its normal flexed position of about 45°. Apart from the increased flexion of the scaphoid, signs of degenerative arthritis will be seen between the radius and the proximal end of the scaphoid, because the area of joint contact will be reduced, causing cartilage wear (Fig. 4.2). In stage I, the radiographic changes at the radio-scaphoid joint will be different in SLAC and SNAC wrists. In SLAC wrists, the proximal scaphoid will be involved, while in SNAC wrist the degenerative arthritis will be most evident at the distal part of the scaphoid, and the proximal scaphoid fragment will be free of degenerative changes. The proximal scaphoid will behave as a lunate, having a similar radius of curvature as that of the radius and following the lunate into extension. The rest of the four stages are quite similar for SLAC, SNAC and SMAC wrists.

- Stage II. Arthrosis will progress to the scaphocapitate joint. The joint between the scaphoid and capitate is somewhat cylindrical, and joint incongruence will occur as the scaphoid increases its normal flexed position (Fig. 4.3a, b). Cartilage wear will not be as important as it bears less compressive forces.
- Stage III. Because the lunate is in extension, the patient has to flex the wrist at the midcarpal joint in order to keep the hand and fingers aligned with the long axis of the forearm. This causes a dorsal subluxation of the capitates and a reduced contact area between the lunate and the capitate. Joint compression forces are also increased because the scaphoid fails to bear the transmission forces through the radial column of the carpus. Both, the reduced contact area and the increase of compression forces, will cause degenerative arthritis at the luno-capitate joint (Fig. 4.4a-c). The lunate and the radius are very congruent, with their joints having similar radii of curvature, thus radio-lunate arthrosis will be very rare.
- Stage IV. This stage is not usually described, but with the passing of time, degenerative changes also occur at the joint between the triquetrum and the hamate bones (Fig. 4.5a–c). Identification of a stage IV SLAC wrist is important for the election of the surgical procedure, as will be discussed later.
- Stage V. Degenerative arthritis of the radiolunate joint was first described by Peterson and Zsabo in 2006 [5]. They named it stage IV SLAC wrist, but we have reserved this stage for the cases with degenerative arthritis of the triquetro-hamate joint, as previously described. Degenerative arthritis is rare at the radio-lunate joint as the articulating bones have a similar radius of curvature and the joint remains congruent even in cases with important extension deformities of the lunate. One possible explanation for the presentation of radio-lunate arthritis is that the lunate goes into extreme extension, most frequently in hyperlax wrists, and gets fixed in this

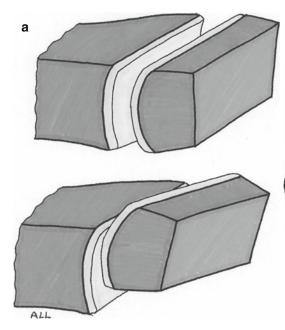
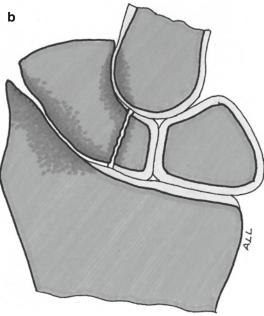
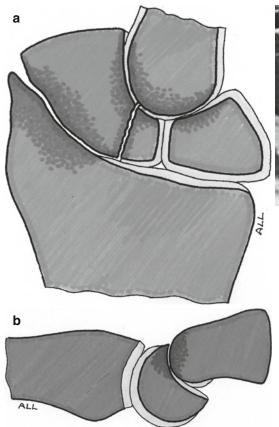


Fig. 4.3 (a) Flexion deformity of the scaphoid causes joint incongruence at both the radio-scaphoid and the scapho-capitate joints. (b) In stage II SLAC and SNAC



wrists, degenerative arthritis between the scaphoid and the capitate is seen





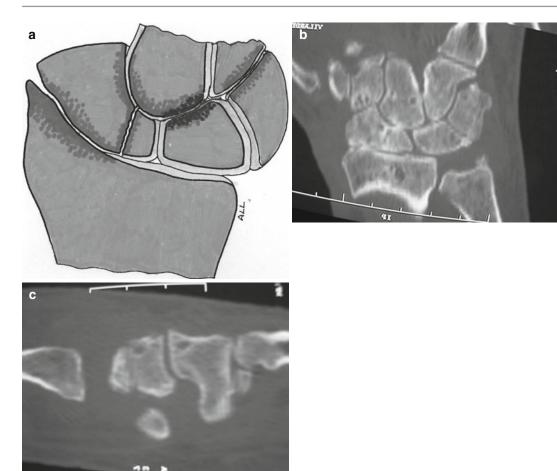


Fig. 4.5 (a) In stage IV SLAC and SNAC wrists, degenerative arthritis is also seen between the hamate and the triquetrum. (b) Degenerative arthritis between the hamate

and triquetrum in the coronal plane. (c) Degenerative arthritis between the hamate and triquetrum in the sagittal plane

position, and also pushed down by the head of the capitate, from the very early stages (Fig. 4.6a–c). Since the patient wants to have his hand and fingers aligned with the long axis of the forearm, he has to flex the wrist at the midcarpal joint, which causes dorsal subluxation of the capitate. In case of discomfort, he will bring the wrist in moderate extension, thus reducing the capitate subluxation and changing the contact areas of the joint.

Most times, it is very difficult to make a diagnosis of a stage V SLAC or SNAC wrists from radiographic or MRI examinations. Careful exploration of both joints during the surgical approach will disclose a more severe radio-lunate rather than midcarpal degenerative

Fig. 4.4 (a) In stage III SLAC and SNAC wrists, degenerative arthritis will be observed between the capitate and the lunate. (b) Extension of the lunate will cause dorsal subluxation of the capitate, causing cartilage wear of the

capito-lunate joint. (c) MRI of the capito-lunate joint showing synovitis, and degenerative changes in the head of the capitate and dorsal horn of the lunate (Note from the author: please rotate this image horizontally, left to right)

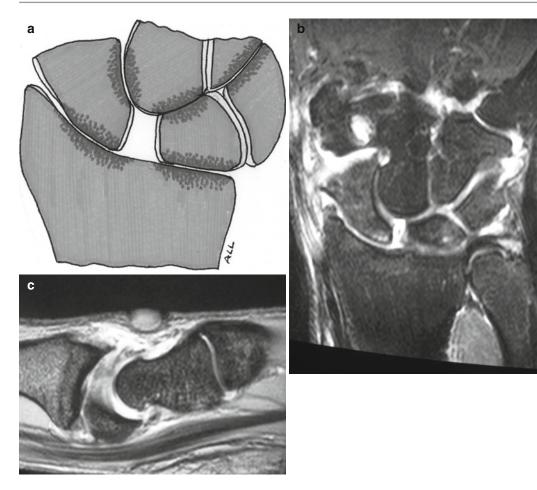


Fig. 4.6 (a) In stage V SLAC and SNAC wrists, degenerative arthritis can be observed between the radius and the lunate. (b) MRI in the frontal plane of a stage V SLAC wrist in which the midcarpal joint is better preserved than the radio-lunate joint. (c) MRI in the sagittal plane

showing an important extension deformity of the lunate with alteration of the radio-lunate joint. The midcarpal joint is better preserved as it will become more congruent when the patient extends the wrist

arthritis (Fig. 4.7a, b). After examining 146 wrists with scapho-lunate dissociation, Lane et al. [7] have observed 9 wrists (6 %) with radio-lunate arthritis and no radio-scaphoid and midcarpoal degenerative changes.

SNAC Wrist

In 1970, Fisk [8] first reported degenerative changes after longstanding scaphoid nonunions. These findings were later confirmed by other studies [9, 10]. The pattern of degenerative arthritis is

similar to the one described for the SLAC wrist, and for this reason this acronym has been used until recently to describe degenerative arthritis secondary to scaphoid non-union [1]. However, the acronym SNAC wrist, standing for Scaphoid Nonunion Advanced Collapse, seems more appropriate, and its use was suggested by Krakauer et al. [11], even though they continued to use the term SLAC wrist in their own publication. The acronym SNAC is now more commonly used [12, 13].

In the presence of a scaphoid nonunion, the distal fragment flexes from the compression forces transmitted by the distal carpal row. The

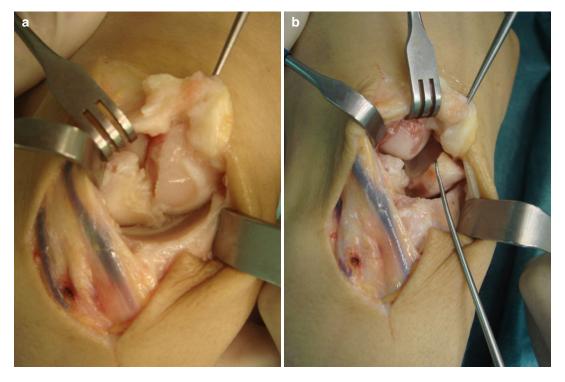


Fig. 4.7 (a) Intraoperative photograph of a SLAC wrist showing severe destruction of the proximal joint surfaces of scaphoid and lunate bones. (b) The midcarpal joint, particularly the head of the capitate is better preserved

scaphoid also loses its normal length from wear of the bone at the fracture ends. Belsole et al. [14] observed an average of 6-15 % of bone loss (average 10 %) in this bone, which has a volume of 2.3–4 cm³ (average 3 cm³). The bone loss is even greater in longstanding cases, as seen in tomographic studies, where the proximal fragment of the scaphoid is barely in contact with the distal radius (Fig. 4.8).

Because the scaphoid is shortened, all compression forces are transmitted by the central column of the carpus, causing extension of the lunate as previously described for the SLAC wrist. The proximal fragment of the scaphoid is brought into extension by the intact scapholunate ligaments [15]. The lunate is very congruent with the radius because their joints have the same radii of curvature. For this reason, perilunate or trans-scapho-perilunate dislocations are more common than pure lunate dislocations. The fact that the lunate is congruent with the radius in all positions of flexion and extension explains



Fig. 4.8 Severe shortening and flexion deformity of the scaphoid after a longstanding pseudoarthrosis. The scaphoid is shortened so much that it barely contacts the radius. The extended lunate brings the proximal fragment of the scaphoid into extension

why degenerative arthritis between the lunate and the radius does not usually occur even in long standing cases of SLAC or SNAC wrists. In the SNAC wrist the proximal fragment of the scaphoid follows the extended position of the lunate and keeps congruency with the distal radius, without causing cartilage wear at this level. The only difference between SLAC and SNAC is in stage I. In the SLAC wrist the degenerative arthritis is seen between the proximal end of the scaphoid and the radius, while in the SNAC wrist, the degenerative arthritis is seen between the distal fragment of the scaphoid and the radial styloid [16].

SMAC Wrist

This acronym proposed by Lluch in 1998 [17] derived from the initial letters of the following words: "Scaphoid Malunion Advanced Collapse". When the scaphoid fracture heals in a deformed position, with shortening, flexion of the distal fragment and extension of the proximal fragment, a pattern of degenerative arthritis similar to the SNAC wrist will also occur [18, 19]. Therefore, the aim of the treatment for scaphoid fractures should not only be to obtain healing, but union of the fracture should be without bone deformity. To prevent a future SMAC wrist pattern of degenerative arthritis, a young patient with a fractured scaphoid which has healed in a very deformed position, should be treated by corrective osteotomy and the addition of an anterior bone graft [20].

The other difference between SLAC, SNAC and SMAC wrists is the time elapsed between the injury and the onset of degenerative arthritis. The SLAC wrist is the one that will progress faster, while the SMAC wrist will take longer to develop degenerative arthritis, and will always have a direct relationship to the degree of scaphoid deformity. Within the SNAC wrists, the worst prognosis will be seen in those cases with a very proximal fracture of the scaphoid, where the distal fragment of scaphoid will very rapidly develop a degenerative arthritis with the radial styloid. When the pseudoarthrosis occurs at the distal end of the scaphoid, the SNAC wrist will take longer to develop.

Clinical Pearls

- SLAC, SNAC and SMAC wrists are patterns of degenerative arthritis secondary to alterations of the scaphoid bone
- SLAC wrist is secondary to scapho-lunate ligament ruptures
- SNAC wrist is secondary to pseudoarthrosis of the scaphoid
- SMAC wrist is secondary to scaphoid malunion
- Radiographic alterations are progressive, and five stages are described
- Radiographic alterations are similar in all of the cases, except for stage I. In SLAC wrists, radio-scaphoid arthrosis is mainly observed at the proximal pole of the scaphoid. In SNAC wrists, the proximal fragment of the scaphoid does not usually develop degenerative arthritis.

Treatment of SLAC, SNAC and SMAC Wrists

When planning for the treatment of SLAC, SNAC or SMAC wrists, standard wrist radiographs will not be sufficient, as it is quite difficult to accurately determine the degree of deformities of the scaphoid and the extended position of the lunate in the lateral projection. Tomographic studies should always be done, which will demonstrate the degree of shortening of the scaphoid, as well as the flexion of the distal fragment and the extension of the proximal fragment [21]. The amount of extension of the lunate can also be measured with precision, as its correction should be the priority of all surgical corrective procedures

Conservative Treatment

Since it is known that some cases of degenerative arthritis are asymptomatic, a trial of conservative treatment is recommended. Apart from the intake of non-steroidal anti-inflammatory medication, the most effective treatment is the immobilization of the wrist with a splint or plaster cast for 3–4 weeks. This proves to be effective in older patients, rather than in younger individuals, and in particular those with a SLAC wrist.

Wrist Denervation

This is a surgical alternative to take into consideration in the older group of patients, because it has the advantages of a shorter postoperative recovery and does not decrease wrist mobility. However, since its effectiveness and duration are limited, it is not a good indication for treating young patients [22, 23].

Radial Styloidectomy

This may be indicated for cases of SNAC or SMAC wrists but not in SLAC wrists. In the latter, a limited dorsal styloidectomy of the radius will alleviate symptoms, but in the long run will cause more instability of the scaphoid, unless the deformity is corrected at the same time. A true radial styloidectomy is a good procedure for the treatment of stages I and II of SNAC wrists, but only when it is associated with the treatment of the nonunion and correction of the deformity by bone grafting of the pseudoarthrosis and internal fixation of the scaphoid. One should be careful not to perform a large styloidectomy, of no more than 6 mm, as there is the danger of causing ulnar translocation of the carpus and radio-carpal incongruence from disruption of the radioscapho-capitate ligaments [24, 25].

Proximal Row Carpectomy

Proximal row carpectomy can be done in patients with stages I and II, with the advantage of it being an easier procedure than a midcarpal fusion, as it does not require reduction of the lunate deformity, fixation and fusion to the capitate, and no need for possible hardware removal. Postoperative recovery is also faster. Good results have also been reported for the treatment of stage III, when there is minimal wear of the cartilage covering the head of the capitate [26]. However, the good initial results will deteriorate with the passing of time, because the head of the capitate and the distal radius have different radii of curvature. Therefore it is not recommended in patients younger than 35 years of age [27].

Scaphoid Resection and Partial Wrist Fusion

Resection of the scaphoid is the most effective treatment, as pain originated at the radio-scaphoid and scapho-capitate joints will disappear. However, scaphoid excision alone should never be done, as it will cause further collapse of the midcarpal joint, with secondary extension of the lunate. To prevent this, some kind of midcarpal fusion should be associated [28]. The most frequently performed fusion has been called the "four corner fusion". We do not use this term because we do not fuse corners, but rather bones. A four bone fusion is a more acceptable term but, to be more precise, we prefer to name the bones to be fused.

Watson and Ryu [29] recommended fusing the capitate and hamate to the lunate and triquetrum. At first, they replaced the scaphoid with a silicone implant, which was later abandoned because of frequent implant dislodgement and occasional foreign body reaction to silicone particles. Internal fixation can be achieved with transosseous K-wires, compression staples, headless compression screws or circular plates.

The skin incision at the dorsum of the wrist can be longitudinal, transverse or zig-zag. A longitudinal incision seems to be favored by most surgeons as it decreases the chances of injury to the venous return and the branches of the sensory radial nerve. However, it will require more retraction of the skin edges and increased soft tissue damage. A dorsal transverse incision will provide adequate visualization of the carpal bones, as the skin is very mobile in this area. This is the recommended incision for women with smaller size wrists, because of its superior aesthetic result. In the case of large hands, usually in men, a zigzag incision is recommended as it provides a better





Fig. 4.9 (a) Scaphoid excision and midcarpal fusion for the treatment of a SNAC wrist stage IV. A *Spider* ® circular plate has been used for internal fixation of capitate, lunate, hamate and triquetrum bones. (b) Same

exposure. The middle part of the zigzag goes in an oblique direction, following the 23° inclination towards the ulnar side of the distal end of the radius. The incision is extended proximally and distally at 90° angles. This will provide the best exposure by gently retracting the proximal and distal flaps to the sides, without any traction to the skin edges. The surgical scar will also be less noticeable than using a longitudinal incision. Next, the capsule is divided from the distal end of the radius, being careful not to divide the dorsal radio-triquetral ligament at the most ulnar side of the radius. This will provide excellent exposure of the radio-carpal and midcarpal joints. After the midcarpal joint is identified, a 2-2.5 mm K-wire is inserted at the dorsal horn of the lunate. The direction of the wire should be from posterior to anterior and from proximal to distal, so the lunate is transfixed perpendicularly from the posterior to the anterior horn. Fluoroscopic examination should be done to confirm the correct position of the K-wire, as it should not go through the midcarpal joint or protrude anteriorly outside the lunate. Using the K-wire as a lever arm, the lunate

Spider [®] plate in the lateral x-ray projection. Because the plate was too superficially placed over the carpal bones, it caused impingement with the radius on wrist extension

is brought into neutral position by pushing the K-wire distally. More or less force will be required depending on the duration and the degree of deformity, which will have caused remodeling of the anterior capsular structures of the midcarpal joint. As mentioned before, a K-wire protruding anteriorly on the lunate will impinge on the anterior border of the distal radius, causing difficulties for a complete reduction. Correction of lunate extension will be easier if the scaphoid is removed first. This can be done with just a bone rongeur, but excision of the distal scaphoid will be difficult, and a scalpel will be needed to divide the scapho-trapezium-trapezoid ligaments. To obviate this, the distal scaphoid does not need to be removed, as it will not cause any impingement with the distal radius during radial inclination of the wrist, as it is located in a plane anterior to the distal radius (Fig. 4.9a, b).

Due to its convexity, excision of the subchondral bone of the head of the capitate can be done easily with a rongeur. However, excision of the subchondral bone of the lunate, because of its concavity, will be more difficult with a rongeur,



Fig. 4.10 Capito-lunate fusion for the treatment of a SLAC wrist. Internal fixation was achieved with two parallel headless compression screws. The distal scaphoid does not need to be removed, as it is located anterior to the radius and will not cause impingement during radial inclination of the wrist

particularly in those cases with sclerotic subchondral bone. The intermittent use of a small bone curette will facilitate this task. After the subchondral bone of the capitate and lunate are removed, correction of the extension of the lunate should be done with the K-wire used as a lever arm. If a complete midcarpal fusion is intended, the subchondral bone of the hamate and triquetrum should also be removed. This should always be done in stages IV of degenerative arthritis. In the other cases, we prefer to fuse only the capito-lunate joint (Fig. 4.10). Fusion of the hamate and triquetrum may cause ulno-carpal impingement in those patients with an ulna which is of equal length or longer than the radius, as the fused triquetrum will not be able to move distally during ulnar inclination of the wrist. To prevent this, some surgeons also recommend excising the triquetrum.

Internal fixation of the midcarpal joint, either partial (luno-capitate) or total (luno-capitate and triquetrum-hamate) can be done with K-wires, although this can be quite difficult, as they must be inserted from distal to proximal and follow the longitudinal axis of the carpus. To prevent impingement of the wires with the radius, another possibility is to place the wires transversely from the ulnar side of the wrist. Internal fixation can also be accomplished with a circular plate and screws: Spider plate ® (Kineticos Medical, Inc.) (Fig. 4.9a, b). A Diamond Carpal fusion plate ® (Small Bone Innovations, Inc.) with 4 screws has also been designed for internal fixation of a midcarpal fusion. The Spider plate has the advantage of facilitating the technique of denuding the subchondral bone with a specially designed Spider rasp and the placing of 6-8 screws, depending on which size plate is being used for the arthrodesis. A possible complication is impingement of the plate against the distal end of the radius, during extension of the wrist, when it is placed too superficial over the carpal bones to be fused (Fig. 4.9b). Care should be taken that the screw for the triquetrum is not too long, because it will transfix of the piso-triquetral joint. Another inconvenience, not really related to the design of the plate, is that this technique makes the surgery too easy, and the less experienced surgeon may not really perform and adequate removal of all the subchondral bone of the capitate, lunate, triquetrum and hamate, causing non-union of some of the joints. Vance et al. [30] reported more complications with the use of a circular plate versus traditional techniques.

Total midcarpal fusion, including capitate, hamate, lunate and triquetrum, will prevent the normal distal displacement of the triquetrum during ulnar inclination of the wrist. As a consequence, ulno-carpal impaction may occur in cases where the ulna is of equal length or longer than the radius. To prevent this, some authors recommend to perform only a capito-lunate arthrodesis, with [31, 32] or without excision of the triquetrum [33].

We obtained the best results by only fusing the capito-lunate joint, after excision of the scaphoid and correction of lunate extension. Internal fixation is accomplished with two parallel cannulated headless screws of 2 mm diameter introduced from the proximal joint of the lunate into the capitate (Fig. 4.10). Dimitrios et al. [34] obtained a 100 % union rate of capito-lunate fusions, but 3 out 8 patients still had persistent pain. The pain, most probably originated at the triquetral-hamate joint in cases of stage IV degenerative wrist. To prevent this possible complication we recom-

mend a total midcarpal fusion only in cases of

stage IV degenerative wrist.

Another subject that needs to be discussed is what to do with the proximal pole of the scaphoid in cases of SNAC or SMAC wrist. In such cases, the proximal pole of the scaphoid behaves as the lunate, placing itself in extension without causing degenerative arthritis with the radius. For this reason, the proximal pole of the scaphoid should be left in place, for the purpose of increasing the surface of joint contact between the carpal bones and the radius, particularly when the lunate is not fully covered by the radius. The proximal pole of the scaphoid can be left intact, after its correction together with the lunate, or can be fused to the capitate as proposed by Viegas [35].

Radio-scapho-lunate Arthrodesis

This would be the preferred technique for cases of stage V arthritis (Fig. 4.11), as mobility at the midcarpal joint will be preserved. The so called "dart throwers motion" (DTM), which follows a plane that varies from 37° to 59° from the sagittal plane according to the different investigators [36–38] takes place at the midcarpal joint. This is the plane followed by the wrist extensors, extensor carpi radialis longus and brevis, and the main wrist flexor, the flexor carpi ulnaris, and used for the majority of hand activities. Patients prefer and adapt quicker to a radio-scapho-lunate arthrodesis than a midcarpal fusion. Excision of the distal scaphoid increases mobility after radio-scapho-lunate arthrodesis [39].

Total Wrist Fusion

Since scaphoid excision and partial or total midcarpal arthrodesis provide good results, the indications for a total wrist fusion are limited. This is due to the fact that the radio-lunate joint maintains

Fig. 4.11 A stage V SLAC wrist has been treated by means of a radio-scapho-lunate fusion

its integrity, even in longstanding cases, except for the rare stage V degenerative arthritis.

Total Wrist Arthroplasty

A flexible silicone wrist implant was designed by Swanson in 1973 [40]. It was mainly intended for use in rheumatoid patients. Early results were generally gratifying, with good pain relief and an acceptable range of motion. However, longer follow-up revealed subsidence within the distal carpal row and rupture of the implant in the majority of cases [41, 42]. Foreign body reaction to silicone particles had a lower incidence as compared to scaphoid or lunate implants.

The first designs of rigid two component implants also presented an unacceptable complication rate, and consequently most are no longer in use, except for low demand rheumatoid patients. However, in the past several years substantial design modifications have been introduced. The distal bone anchorage is not into the metacarpals but into the distal carpal row by means of an elliptical metallic plate and screws [43]. The "Universal Total Wrist" ® implant was



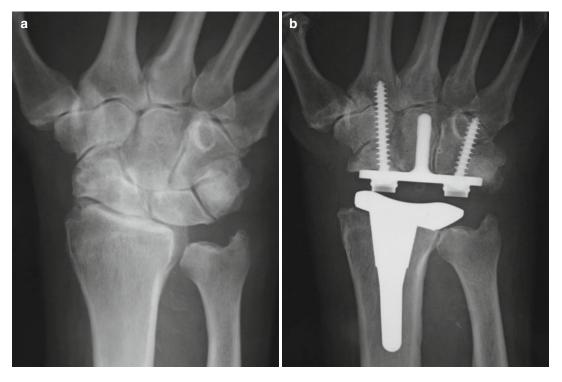


Fig. 4.12 (a) SMAC wrist stage IV with degenerative changes between the radius and the scaphoid, and the entire midcarpal joint. A volar approach to the scaphoid and a radial styloidectomy had been undertaken many

manufactured by *Kineticos Medical Inc. San Diego CAL. USA*. Since the anchorage should be in the distal carpal row, these bones should be well preserved, or fused in cases of rheumatoid arthritis [34]. These total wrist implants are semiconstrained, with the polyethylene bearing between both components having a condylar shape, which has recently changed from toroid to ellipsoid to improve stability and decrease wear [44, 45]. After some modifications from the original design, this new implant is known as "Universal Total Wrist II ® (Integra LifeSciences, Plainsboro, NJ, USA).

Although initially used for the treatment of rheumatoid arthritis, these new implants, also called third generation, have also been used for the treatment of degenerative arthritis with short term satisfactory results. We have offered this alternative to patients who require painless wrist mobility for certain activities of daily living or, even more important, for work requirements. The patient should be willing to accept possible future

years ago, causing a moderate ulnar translocation of the carpus. (b) As there was little contact between the radius and the lunate, a wrist replacement using a *Universal Total Wrist II* ® was performed, with very satisfactory results

complications, such as implant loosening or wear. In our experience the midterm clinical and radiographical results have been very satisfactory [46] (Fig. 4.12a, b).

Other third generation total wrist implants are the *Re-motion* [®] (*Small Bone Innovations, Morrisville, PA, USA*) [47] and the *Maestro* [®] (*Biomet Orthopedics, Warsaw, IN. USA*) [48].

Clinical Pearls

- The most important surgical procedure consists of removing the scaphoid
- The midcarpal joint should be fused after correcting the extension deformity of the lunate
- A luno-capitate arthrodesis will provide equal results to a four-bone fusion
- Proximal row carpectomy is another surgical alternative when the head of the capitate is not involved.

Scapho-trapezio-trapezoid (STT) Arthritis

Incidence

The first description of S-T-T arthritis was made by Carstam et al. in 1968 [49]. Since then, the incidence of degenerative arthritis of the S-T-T joint, based on radiographic examinations, has been reported from 7 to 55 % [50–55]. As in all degenerative arthritis in the wrist, the incidence is much higher if the diagnosis is done by anatomical dissection of the wrist of elderly people. Bathia et al. [55] examined 73 cadaveric wrists, 25 male and 48 female, with an average age of 84 years, and found an incidence of 83.3 %. Degeneration of the scapho-trapezoid articular surface was found to be more common and severe than that of the scapho-trapezium joint.

Etiopathogenesis

Some authors think that S-T-T arthritis and S-L dissociation may be related [56]. Weinzeig and Watson [57] observed a SLAC pattern of degenerative changes in 57 % of the cases of periescaphoid arthritis, 27 % of arthritis only at the S-T-T joint, and a combination of both in 15 % of the cases. However, careful observation of the lateral radiographs of SLAC and SNAC wrists demonstrate opposite deformities of the scaphoid. In cases of S-L dissociation, the scaphoid is flexed and the trapezium and trapezoid are frequently found to rest on the dorsal nonarticular portion of the distal scaphoid. On the contrary, in the presence of S-T-T arthritis, the scaphoid is usually extended. Simultaneous observation of S-L dissociation and S-T-T arthritis (15 %) in the same patient could only be considered a coincidence.

The progression of scapho-trapezial ligament injuries to S-T-T osteoarthritis was first suggested by Taleisnik [58]. Linscheid et al. [59] reported that 16 of their 38 patients with isolated S-T-T osteoarthritis had previously sustained injuries to the thumb column. Sicre et al. [60] also observed osteoarthritis of the S-T-T joint after scaphotrapezial ligament injuries, and we also have seen the same finding. Taleisnik [58] believes that the development of osteoarthritis of the S-T-T joint may, in many patients, be the result of an unstable S-T-T articulation. This concept should be taken into consideration, as most S-T-T arthroses are seen in women, who also have a higher predisposition than men to develop both T-MC and interphalangeal arthritis secondary to joint instability.

Pinto et al. [61] reported a 48 year old man with S-T-T osteoarthritis secondary to a well documented nontraumatic multidirectional instability of the S-T-T joint. However, not all authors are clear as to which came first: joint instability or joint arthritis [62].

MClean et al. [63] found an association between S-T-T arthritis and lunate morphology. They evaluated 48 patients with S-T-T arthritis, and found that 83 % of them had a lunate type II, while in the control group this was only present in 64 % of the wrists. Lunate type II is defined as that which has two distinct distal facet joints, one for the capitate and the other for the hamate [64]. They postulate that lunate morphology is associated with variations in scaphoid motion, and that these differences may contribute to the development of S-T-T osteoarthritis. We reviewed 27 cases of S-T-T osteoarthritis and found that the majority of patients had a lunate type I. As the authors conclude in their manuscript, further research is required to investigate the relationship between midcarpal morphology and carpal kinematics.

We have observed that chondrocalcinosis or pseudogout, caused by deposition of crystals of Calcium Pyrophosphate Dihydrate (CPDD), is another cause of S-T-T arthritis, all of them confirmed by pathological examination with polarizing light.

Clinical Diagnosis

The onset of symptoms is usually insidious and slowly progressive, although it can be precipitated by an injury such as a fall on the outstretched hand. Radiographic examination done at the time of consultation will already show degenerative arthritis, proving that this may have remained asymptomatic for a long period of time. Such is the case that arthritis can be a radiographic finding during an examination done for other purposes.

The patient complaints of pain and weakness during forceful use of the thumb. Decreased wrist mobility is also a common finding, because mobility at the midcarpal joint will be restricted, although the patient may not have been aware of this previously. Differential diagnosis should be done between trapezio-metacarpal arthritis and De Quervain tenosynovitis. This should not be very difficult, as the patient will have pain on direct pressure over the S-T-T joint, volarly, radially and dorsally. Dorsal swelling from joint synovitis can be seen on the dorsal and radial aspect of the wrist.

Some patients may have more pain at the radiovolar aspect of the wrist, from synovitis of the flexor carpi radialis (FCR) tendon or ganglia originating from the scapho-trapezial capsule and migrating proximally along the tendon sheath [65]. The FCR tendon courses over the volar surfaces of the scaphoid and trapezium in a separate fibroosseous tunnel. MRI studies in patients with S-T-T arthritis demonstrated synovitis of the FCR, partial and full thickness tears and ganglion formation.

An intra-articular infiltration with a local anesthetic will cause immediate temporary relief of pain, confirming the diagnosis.

Radiographic Diagnosis

Due to bony overlap, it is difficult to view the STT joints using standard radiographs. The best way to see the S-T-T joint is by placing the wrist in 30° of ulnar inclination so that the thumb is fully extended and in a straight line with the forearm. The thumb pulp is facing and touching the cassette while the hand and forearm are lifted from the cassette forming an angle of about 30°. The x-ray beam is centered at the S-T-T joint, with the x-rays crossing perpendicular to the joint line [66].

Joint space narrowing, subchondral bone sclerosis, and peripheral hyperthrophic spurring will be observed. Subchondral bone cysts are rare. The most interesting finding is an extension deformity of the scaphoid, best seen on lateral



Fig. 4.13 S-T-T degenerative arthritis with shortening of the scaphoid secondary to cartilage and bone wear. The scaphoid and the lunate are in an extended position

radiographs. In the lateral x-ray projection, the normal radio-scaphoid angle, varies between 45° and 60°, and in advanced cases of S-T-T arthritis we can see angles of 20° or even less. We believe that the extension deformity of the scaphoid is due to shortening from wear of the cartilage and the bone ends, as seen in other joints, such as the T-MC, hip or knee joints (Fig. 4.13). Furthermore, the extension of the scaphoid appears to be progressive with increasing joint space narrowing. Another factor is the presence of a moderate dorsal subluxation of the distal scaphoid, although, this could be secondary to the latter. Since the radial column of the carpus is shortened, the scaphoid will not be able to maintain the length of the central column, which will progressively collapse [67]. The wedge shaped lunate, with a dorsal horn thinner than the anterior horn, interposed between the capitate and the radius, will be pushed into extension. The normal 11° volar inclination of the distal radius will facilitate the extension of the lunate (Fig. 4.1).

Clinical Pearls

- S-T-T ostheoarthritis is the most frequent primary arthritis of the wrist
- The proximal carpal row becomes progressively extended
- Extension of the lunate will cause dorsal subluxation of the capitate.

Treatment

Conservative

As many cases of S-T-T arthritis remain asymptomatic for several years, a trial of conservative treatment is recommended. Apart from the intake of non-steroidal anti-inflammatory medication, the most effective treatment is the immobilization of the wrist with a splint or plaster cast for 3–4 weeks. Intra-articular steroid injections will enhance the results of the conservative treatment. This can be effective in older patients, but not so much in younger individuals.

Flexor Carpi Radialis Tendon Release and Joint Debridement

In some patients, the discomfort caused by flexor carpi radialis tenosynovitis is more important than that caused by the arthrosis. In these cases, through a volar zig-zag surgical approach, the FCR tendon is released and synovectomy performed. Prominent anterior osteophytes, originating at the scapho-trapezoid joint, should also be removed, as these will cause tendon synovitis, tendon attrition and even rupture in some cases.

Arthroscopic debridement has also been described [68]. The authors performed a washout of the joint with arthroscopic debridement of the hyperthrophic synovial tissue, chondral flaps and osteophytes, reporting satisfactory results on an average of 36 months of follow-up.

Resection of the Distal Scaphoid

As in many other joints of the hand a resection arthroplasty will provide excellent results. The distal pole of the scaphoid can be removed through a dorso-radial zigzag incision or with a volar approach in those cases presenting with FCR tenosynovitis. Crosby et al. [69] recommended to interpose a slip of the FCR in a coiled fashion.

Although excision of the distal scaphoid will provide satisfactory results, it will cause further midcarpal instability in some cases [70]. It may be well tolerated in some of them, but others will require a capito-lunate arthrodesis after correcting the extension of the lunate. To prevent midcarpal subluxation after distal scaphoid excision, Garcia-Elias and Lluch [71] proposed a dorsal capsulodesis of the scapho-trapezoid joint with a distally based capsular flap tightly reattached on to the dorsum of the scaphoid. It is recommended that before proposing a resection of the distal scaphoid a dorsal midcarpal instability should be ruled out under fluoroscopic control. If a "posterior drawer sign" of the midcarpal joint is clearly demonstrated, then an S-T-T arthrodesis would probably be a better treatment option [72].

Trapezium Silicone Implant Arthroplasty

Removal of the trapezium and replacement with a silicone implant will provide satisfactory results when the main arthrosis is present at the T-MC joint [73]. Implant arthroplasty will alleviate pain from the trapezio-metacarpal and the scaphotrapezium arthritis, as both joints are treated by replacement of the trapezium with the implant. Pain from the scapho-trapezoid joint is also relieved from the joint distraction effect provided by the implant (Fig. 4.14a, b). The implant will prevent further erosion of the trapezium and scaphoid, with secondary shortening and extension deformity of the latter. A subluxation of the implant is a major concern, but it can be prevented by using a careful surgical technique.

Pyrocarbon Implant Interposition

After excision of the distal scaphoid, Pequinot [74] proposed to interpose a disc made of pyrocarbon between the scaphoid and the trapezium called "*STPI implant*" ®, standing for Scaphoid Trapezium Interposition Implant, *BIOProfile, Grenoble, France*. This disc provides



Fig. 4.14 (a) Degenerative arthritis of the T-MC and S-T-T joints in an elderly woman who had previously suffered a fracture of the distal radial metaphysis. In spite of the deformity, the distal radio-ulnar joint was painless and she had full pronation and supination of the forearm.

(b) The arthritis of the T-MC and S-T-T joints was treated with a trapezium silicone implant. The implant restored the length of the radial column of the carpus, preventing collapse of the central column and alleviating pain at the scapho-trapezoid joint

a satisfactory relief of symptoms and an increase in grip strength, but postoperative dislodgement of the implant is a possible complication: 20 % in the first reported series of cases.

Arthrodesis of the Scapho-trapeziotrapezoid Joints

Arthrodesis of the S-T-T joints was first done by Watson in 1980 [2]. In 2003 they reported the results of a follow-up study on 800 S-T-T fusions, mostly done for the treatment of scapho-lunate dislocations. The results were uniformly good except for the risk of developing radio-scaphoid degenerative arthritis with the passing of time [75]. S-T-T fusion restricts scaphoid flexion, mainly during radial inclination of the wrist causing radio-scaphoid impingement. For this reason it is contraindicated in cases with preexisting degenerative changes of the radio-scaphoid joint. Radio-scaphoid arthrosis after S-T-T fusion has also been reported by other authors [76]. To prevent this, they recommended to arthrodese the distal scaphoid at 58° of flexion. Minamikawa et al. [77] recommended performing the S-T-T fusion with a radio-scaphoid angle from 41° to 60°. The purpose is to fuse the scaphoid in a slightly more flexed position than normal, in order to prevent radio-scaphoid impaction during wrist mobility.

The arthrodesis can be done trough a dorsoradial transverse or zigzag incision. The S-T-T joint is quite large, and careful removal of all the cartilage and subchondral bone should be performed so as to avoid nonunions. Internal fixation can be done with Kirschner wires, although headless cannulated screws will provide compression and better stability of the bones to be fused (Fig. 4.15a, b).

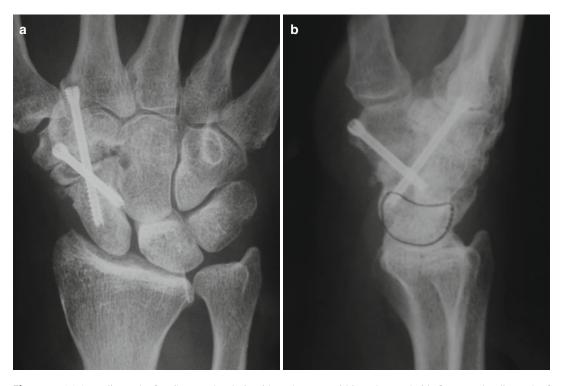


Fig. 4.15 (a) AP radiograph of an S-T-T arthrodesis with the interposition of a bone graft to correct the extension deformity of the scaphoid and regain length of the radial column of the carpus. The arthrodesis was stabilized with two headless compression screws, from the trapezium and

the trapezoid into the scaphoid. (b) Lateral radiograph of the S-T-T arthrodesis demonstrating the correction of the extension deformity of the lunate. The pisiform was used as a bone graft as the patient also had important severe pain from a piso-triquetral degenerative arthritis

The most difficult deformity to be treated is the extension deformity of the scaphoid causing dorsal subluxation of the midcarpal joint. If the scaphoid position is not corrected, the patient will continue to have pain at the luno-capitate joint, and luno-capitate fusion may be needed in the future. Since our aim is to reduce the extension of the lunate, after denuding the S-T-T joint, we correct the lunate extension with a K-wire, using it as a lever arm, the same technique that is used for the SLAC and SNAC wrists. Next, the gap at the S-T-T joint is filled with a corticocancellous bone graft obtained from the distal radial metaphysis.

Radio-lunate Arthrodesis

Excision of the distal scaphoid will cause dorsal subluxation of the capitate, mainly in patients with hyperlax wrists, causing degenerative arthritis and pain at the luno-capitate joint. S-T-T arthrodesis after correction of the extension deformity of the scaphoid will prevent midcarpal joint collapse, but the surgical technique is quite demanding as it is difficult to regain scaphoid length without the use of a large bone graft.

Recently we have treated S-T-T arthritis with a radio-lunate arthrodesis after correcting the extension deformity of the lunate. With this procedure the midcarpal collapse is corrected and future osteoarthritis at this level is prevented. Simultaneous excision of the distal scaphoid will alleviate symptoms at the S-T-T joint. In other cases, we have performed a FCR tendon release and a circumferential capsulotomy between the scaphoid and the trapezium and trapezoid bones through an anterior approach with equally satisfactory results.

Clinical Pearls

- Several treatment options have been proposed: conservative, FCR tendon release, resection of the distal scaphoid, trapezium silicone implant arthroplasty and arthrodesis
- Resection of the distal scaphoid should not be done in patients with laxity of the midcarpal joint, as dorsal subluxation of the capitate will increase
- Radio-lunate arthrodesis is a newly proposed technique.

Distal Radio-Ulnar Joint (DRUJ) Degenerative Arthritis

Primary degenerative arthritis is rare, but secondary arthritis will occur from joint cartilage destruction after fractures of the distal radius or ulna. Other causes are joint in congruencies secondary to joint instability or length discrepancies between the radius and ulna, usually occurring after malunited fractures of the distal radius.

The anatomy of the radio-carpal joint varies very little among the different individuals, with the joint facet of the distal radius presenting an almost constant ulnar inclination of 23° and a volar inclination of 11°. However, the filogenesis of the human DRUJ explains the wide variety of morphological changes observed among different individuals. We may see different sizes of the head and styloid process of the ulna, as well as a joint with a variety of sizes and inclinations. For this reason, the length of the ulna in relation to the radius is not constant, with some individuals having a shorter ulna with a joint with an inclination following a plane from proximal-radial to distal-ulnar. The only anatomical feature constant in everyone is the radius of curvature of the sigmoid notch of the radius being larger than that of the head of the ulna, which does not make the DRUJ a fully constrained joint. The head of the ulna has a slight proximal displacement on pronation of the forearm, and a larger antero-posterior displacement during forearm rotation. The different radii of curvature allows for the ulnar head to displace anteriorly during forearm supination, and dorsally, in reference to the radius, on full pronation of the forearm [78].

Diagnosis

Patients with degenerative arthritis of the DRUJ complain of pain on the ulnar side of the wrist on pronation and supination of the forearm, mainly during load bearing. Pain is usually more severe during forearm supination, due to the anterior displacement of the head of the ulna which causes friction against the sigmoid notch of the radius.

Radiographic diagnosis of DRUJ arthrosis is usually not conclusive, except in very advanced cases. At surgery we see severe joint cartilage destruction, mainly on the head of the ulna, but on standard radiographs we will not see joint narrowing, osteophytes and subchondral bone cysts, as seen in other weight bearing joints (Fig. 4.16a, b). MRI examinations will be helpful in showing synovitis, which will be best seen on fluid-sensitive sequences, such as a T2-weighted fast spin echo with chemical fat suppression or short time inversion recovery [79]. The most helpful diagnostic test is scintigraphy. A bone scan has high sensitivity and specificity for active bone turnover, but is nonspecific to identify the underlying cause. Differential diagnosis should be made on a clinical basis. An intra-articular infiltration with a local anesthetic will cause immediate temporary relief of pain, confirming the diagnosis.

Clinical Pearls

- Primary degenerative arthritis of the DRUJ is unusual
- Painful DRUJ is usually secondary to fractures or ligament injuries
- Pathology of the DRUJ is seldom diagnosed only on radiographic examination.

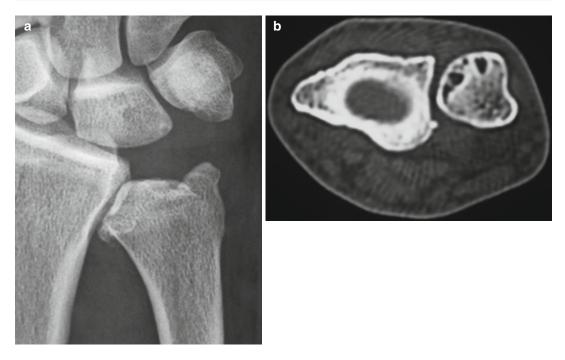


Figure. 4.16 (a) Antero-posterior radiograph of the DRUJ showing joint irregularity and small proximal osteophytes. (b) CAT scan demonstrating subchondral cysts on the head of the ulna

Treatment

Many procedures have been described for the management of the altered distal radio-ulnar joint (DRUJ). As the arthrosis is due to a broad spectrum of pathology there is no single procedure superior to another, and different surgical techniques must be considered for each lesion.

Resection of the Head of the Ulna

Resection of the ulna head was first described by Joseph François Malgaine in 1855 [80], by Moore in 1880 [81], and popularized by William Darrach in 1913 [82]. It is an easy procedure to perform with early good functional results. Darrach reported one case in which the patient regained full forearm rotation 5 weeks after surgery. Resection of the distal end of the ulna was very popular many years ago for the treatment of secuelae of fractures of the distal end of the radius, which often healed with shortening and angulation. As a consequence, the ulna was longer, causing ulno-carpal impingement and restriction of forearm rotation.

It was not until 1982 that Ekenstan et al. [83] first reported complications related to the resection of the head of the ulna. Other publications commented that this procedure was not free of complications, particularly if an excessive amount of ulna was excised [84]. These problems consisted mainly of instability of the proximal ulna and loss of grip strength. Bell et al. [85] introduced the term "ulnar impingement syndrome" to describe painful clicking of the ulna against the radius on forearm rotation. Radiographs showed scalloping of the distal radius at the site of impingement of the distal ulna stump.

Tulipan et al. [86] performed a resection of the head of the ulna at the level of the proximal end of the sigmoid notch of the radius and sloping proximally on the opposite side to prevent a bony prominence under the skin. Through a dorsal 3 cm incision, Di Benedetto et al. [87] performed an excision of the ulna head leaving it just 3–4 mm shorter than the radius. Both reported better results after minimal excision of the head of the ulna, therefore defending the Darrach procedure after modification of the original technique. What they really accomplished was not to disturb the interosseous membrane and the pronator quadratus muscle, the latter being the main stabilizer of the distal ulna stump.

It has subsequently been recognized that the head of the ulna is an important load bearing mechanism for wrist function [88, 89].

Partial Resection of the Head of the Ulna

To avoid the above mentioned possible complications, techniques of partial resection of the ulna head, while preserving the ulna styloid and the ligaments inserting at its base, have been described. Bowers [90] proposed a resection of the cartilage of the head of the ulna with interposition of a tendon slip to prevent the tendency of approximation of the distal ulna to the radius causing pain from bone impingement. This technique is not indicated in cases where arthrosis is secondary to instability of the distal ulna, or when the ulna is longer than the radius. In these cases, the styloid process of the ulna will cause impaction with the triquetrum, particularly during ulnar inclination of the wrist.

Watson et al. [91] proposed a slightly longer excision of the end of the ulna to match the shape of the distal radius, with the aim of preventing recurrence of pain from joint impingement. The distal ulna was reshaped for about 5–6 cm in length, obviating for soft tissue interposition. However, close to 10 % of the patients had painful radio-ulnar impingement, requiring a second operation [92].

Silicone Implant Arthroplasty

Swanson [93] proposed to excise the head of the ulna and cover the bone stump with a silicone cap: *Silastic* ® (*Dow Corning. Midland, MI. USA*). The implant had a stem which was fitted into the medullary canal of the ulna. This procedure has been abandoned for the treatment of

degenerative arthritis due to an unacceptable rate of complications, such as bone resorbtion underneath the cap, causing tilting of the implant, and also ruptures of the stem.

Hemiarthroplasties

To overcome the complications related to the excision of the head of the ulna, the use of a variety of types of partial or total joint implants has been proposed.

In 1998, Scoonhoven, Herbert and Krimmer [94] designed a rigid implant to substitute the head of the ulna. This was called the *UHP Herbert ulnar head prosthesis* (*KLS Martin Group. Tuttlingen. Germany*) and had two components: a conical titanium stem for press fit and later bone in-growth, with three collar designs, and a ceramic head of three different sizes. Although some type of capsular reconstruction can be done at the time of the surgical procedure, the use of this implant is not recommended in cases of major instabilities of the DRUJ.

A similar modular ulnar head replacement was designed at the Mayo Clinic [95]. This was called the *uHead* (*Avanta Orthoopaedics Inc. San Diego, CA. USA*), and later manufactured by *Small Bone Innovations. Morrisville, PA. USA*. It has an intramedullary stem made of chromecobalt, with a titanium sprayed finish, and has two stem-neck designs. The head of the implant is semispheric and made of cobalt-chrome alloy. The ulnar head component has two holes that can be used for fixation of soft tissues to provide stabilization.

Masaoka et al. [96] performed a biomechanical analysis of the two previously described implants, in fresh frozen cadaver wrists, and both maintained near-normal biomechanics of the DRUJ as compared to a resection of the head of the ulna. However, on radiological examination in living patients, bone resorbtion of the distal ulna, related to stress shielding, was observed in the majority of cases in the two previously described implants. Another radiological observation was a radial sigmoid notch erosion opposite the implant.

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A radial sigmoid notch hemiarthroplasty has been designed to add stability to the *uHead* TM implant at the time of surgery or as a secondary procedure: *Stability*TM *Sigmoid Notch* (*Small Bone Innovations. Morrisville, PA. USA*). This is a modular implant with two components: a cobalt chromium radial plate, plasma sprayed with CPTi, and a UHMW polyethylene insert fixed to the plate. The radial plate is fixed to the distal radius by means of a distal peg and a proximal cobalt chrome screw. This can also be used in cases with painful wear of the sigmoid notch of the radius as seen after some ulna head implants.

Kopylov and Tagil [97] designed a one piece metallic ulna head implant in which the ulna head has the shape of an asymmetrical hemi-cylinder, called *First choice DRUJ system* (*Ascension Orthopedics, Inc. Austin, TX. USA*). Its design allowed for some preservation of the ligament insertions into the styloid process of the ulna.

Garcia-Elias [98] designed an ulna head implant called Eclypse ® (Bioprofile. Grenoble, France), to substitute the damaged portion of the ulna head, for the treatment of DRUJ arthrosis. It has two components: a titanium stem implanted inside the medullary canal of the ulna and a hemispheric ulna head made of pyrocarbon. The ulnar stem has a distal peg into which the head is stabilized. It provides a loose fitting of the ulna head component which allows for some proximo-distal translation and slight axial rotation to adjust its position in relation to the sigmoid notch concavity of the radius on pronation and supination of the forearm. The advantage of this design is that it can be inserted without detaching the foveal insertion of the triangular fibro-cartilage and preserves the extensor carpi ulnaris sheath intact. The preliminary results are very encouraging, as it has the best design for the treatment of DRUJ degenerative arthritis without causing instability of the ulna head.

Total DRUJ Implant Arthroplasty

Scheker [99, 100] designed a total implant arthroplasty with a semiconstrained ball and socket design: *APTIS* (*B*) (*APTIS Medical. Louisville*,

KY. USA). The ulnar stem was originally manufactured with 316 L stainless-steel and later changed to cobalt chromium to increase strength. The stem has a highly polished distal peg into which the ball is fitted, allowing proximal-distal migration of the ball during pronation and supination of the forearm. This ball is made of UHMW polyethylene and comes in three sizes. The radial component is a plate which is fixed to the radius with a peg at the level of the sigmoid notch and five proximal cortical screws. At the distal end of the plate there is a socket to accommodate the sphere.

Laurentin-Pérez et al. [101] reviewed the results of 31 implanted prosthesis at an average follow-up of 5.9 years (from 4 to 9 years), only reporting two fractures of the stem inside the ulna as the main complication.

The preliminary results of many of the available prostheses are encouraging, although some of them have an unacceptable complication rate, and their cost-benefit has not yet been established.

Arthrodesis of the DRUJ with Proximal Pseudoarthrosis of the Ulna (The Sauvé-Kapandji Procedure)

In 1921, Baldwin [102] reported restoration of pronation and supination of the forearm, after malunited distal radial fractures. A pseudoarthrosis of the ulna was created after excision of a 2 cm segment of bone proximal to the DRUJ.

In 1936, Louis Sauvé de Gonzagues and Mehmed Ibrahim Kapandji [103] described a similar technique, with the variant that a DRUJ arthrodesis was added to the pseudoarthrosis of the ulna. This technique was attributed to Lauenstein by Arthur Steindler [104] in his book on "The traumatic deformities and disabilities of the upper extremity", and for many years he was referred as the author of this technique in the English medical literature [105]. What Carl Lauenstein, from Hamburg, had actually described was a resection of the head of the ulna [106]. However, the so called Sauvé-Kapandji technique had already been published by Berry [107, 108], from New Zealand, in 1931. This procedure had been done the year before, the only difference being that instead of screws or Kirschner wires a bone peg was used to stabilize the DRUJ arthrodesis.

Arthrodesis is the most reliable and durable surgical procedure for the treatment of a joint disorder, despite the main disadvantage of loss of motion of the fused joint. However, the distal radio-ulnar joint can be arthrodesed, while forearm pronation and supination are maintained or even improved by creating a pseudoarthrosis of the ulna just proximal to the arthrodesis. This is known as the Sauvé-Kapandji (S-K) procedure.

The S-K differs from the Darrach procedure in that it preserves ulnar support of the wrist, as the distal radio-ulnar ligaments and ulno-carpal ligaments are maintained. Aesthetic appearance is also superior after the S-K procedure, as the normal prominence of the ulna head, most noticeable when the forearm is in pronation, is not lost. It also allows for unlimited shortening of the ulna head, which cannot always be done with resection arthroplasties that preserve the length of the styloid process of the ulna.

Just as in any surgical procedure there can be complications, but by using this technique only three are directly related to the procedure: nonunion or delayed union of the arthrodesis, fibrous or osseous union at the pseudoarthrosis and painful instability of the proximal ulna stump. The first two are not of much concern as they can be easily addressed. However, a painful instability of the proximal ulna stump can cause a serious disability, which in most cases can be very difficult to correct. Such complications can be prevented if one follows a careful surgical technique [109–112]. Another advantage of the S-K technique is that the postoperative immobilization period is shorter, which is an added benefit for the patient.

Nonunion of the arthrodesis will not occur if a malleolar or other compression lag screw is used for internal fixation [113], as it provides excellent stability as well as compression of the bone

surfaces (Fig. 4.17a-c). Some authors recommend using a segment of the ulna that has been resected as a bone graft [114–116]. We do not recommend interposing a bone graft between the radius and ulna, as this creates an unnecessary barrier of devascularized tissue, particularly if cortical bone is used. There are no strains or transmission forces through the head of the ulna and therefore it is rare to observe a pseudoarthrosis [117]. Following Kapandji's recommendation [118, 119], most surgeons favour the use of 2 screws, or 1 screw and a Kirschner wire, for the internal fixation. It is true that the head of the ulna can rotate around the axis of the screw during insertion, which should be controlled by the surgeon, but this will never occur after the joint surfaces are engaged under compression. Removing large amounts of ulna, as well as interposing a flap of the pronator quadratus (PQ) muscle, with the aim of decreasing the probability of fibrous union at the pseudoarthrosis will have the deleterious effect of increasing instability of the stump of the ulna [120-128].

The pseudoarthrosis should be created just proximal to the ulnar head, leaving a bone defect no larger than 5 mm. There are three reasons for making the most distal pseudoarthrosis possible. One is to create the pseudoarthrosis as close as possible to the axis of rotation of the forearm, which runs obliquely from the centre of the radial head proximally to the centre of the ulna head distally. An osteotomy done at a more proximal level will cause a divergence of movements between the osteotomized ends of the ulna. Another reason is that the proximal stump of the ulna will contact with the radius in a relatively flat triangular shaped surface, proximal to the sigmoid articular facet of the radius, measuring on average 20.5 ± 1.3 mm in length, which serves for insertion of the deep head of the PQ muscle [129] (Fig. 4.18). Finally, is not to disturb the static and dynamic structures which provide stability to the proximal ulna: the pronatus quadratus muscle [130-133] (Fig. 4.19), the ECU muscle and tendon [134, 135], the FCU muscle and the interosseous membrane insertions [136–138].



Figure. 4.17 (a) Degenerative arthritis of the DRUJ.(b) Early postoperative radiograph of a Sauvé-Kapandji procedure creating a very distal pseudoarthrosis of the

ulna with minimal bone resection. (c) A few weeks after the procedure, the ends of the osteotomized ulna show some shortening and remodeling



Figure. 4.18 Photograph showing the sigmoid notch of the radius and the flat trapezoidal shaped area, proximal to it, where the proximal stump of the ulna should contact

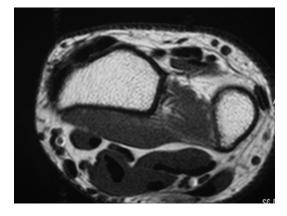


Figure. 4.19 MRI of the DRUJ showing the two heads of the pronator quadrates muscle. The deep head is the main stabilizer of the head of the ulna and should not be disturbed when performing surgery to the distal end of the ulna

Clinical Pearls

- Many surgical procedures have been proposed for the treatment of DRUJ arthrosis
- The choice of surgical treatment should be based on the primary cause of the arthrosis
- Resection of the head of the ulna is the easiest and most effective procedure for pain relief, but it may cause instability of the proximal ulna stump
- The Sauvé-Kapandji procedure can be used for the treatment of all causes of a painful DRUJ. The main complication of this procedure, if not properly done, is instability of the proximal ulna stump, which is again quite difficult to treat.

References

- Watson HK, Ballet FL. The SLAC wrist: scapholunate advanced collapse pattern of degenerative arthritis. J Hand Surg Am. 1984;9A:358–65.
- Watson HK, Hempston RF. Limited wrist arthrodesis. Part I: the triscaphoid joint. J Hand Surg Am. 1980;5:320–7.
- Sarrafian SK, Melamed JL, Goshgarian GM. Study of wrist motion in flexion and extension. Clin Orthop. 1977;126:153–9.
- Lindström G, Nyström A. Natural history scaphoid non-union, with special reference to "asymptomatic" cases. J Hand Surg Br. 1982;17B:697–700.
- Kerluke L, McCabe SJ. Nonunion of the scaphoid: a critical analysis of recent natural history studies. J Hand Surg Am. 1983;18A:1–3.
- Peterson B, Szabo RM. Carpal osteoarthrosis. Hand Clin. 2006;22:517–28.
- Lane LB, Daher RJ, Leo AJ. Scapholunate dissociation with radiolunate arthritis without radioscaphoid srthritis. J Hand Surg Am. 2010;35A:1075–81.
- 8. Fisk GR. Carpal instability and the fractured scaphoid. Ann R Coll Surg Engl. 1970;46:63–76.
- Mack GR, Bosse MJ, Gelberman RH, Yu E. The natural history of scaphoid non-union. J Bone Joint Surg. 1984;66A:504–9.
- Ruby LK, Leslie BM. Wrist arthritis associated with scaphoid non-union. J Bone Joint Surg. 1985;67A: 428–32.
- Krakauer JD, Bishop AT, Cooney WP. Surgical treatment of scapholunate advanced collapse. J Hand Surg Am. 1994;19A:751–9.
- Krimmer H, Krapohl B, Sauerbier M, Hanh P. Postraumatic carpal collapse (SLAC and SNACwrist). Stage classification and therapeutic possibilities. Handchir Mikrochir Plast Chir. 1997;29:228–33.
- Cooney WP, DeBartolo T, Wood MB. Post-Traumatic arthritis of the wrist. In: Cooney WP, Linscheid RL, Dobyns JH, editors. The Wrist. Diagnosis and operative treatment. St. Louis: Mosby; 1998. p. 588–629.
- Belsole RJ, Hilbelink R, Llewllyn JA, Dale M, Greene TL, Rayhack JM. Computet analyses of the pathomechanics of scaphoid waist nonunions. J Hand Surg Am. 1991;16A:899–906.
- Smith DK, Cooney WP, Linscheid RL, Chao EYS. The effects of simulated unstable scaphoid fractures on carpal motion. J Hand Surg Am. 1989;14A:283–91.
- Viegas SF, Patterson RM, Hillman GR, Peterson PD, Crossley M, Foster R. Simulated scaphoid proximal, pole fracture. J Hand Surg Am. 1991;6(16A):495–500.
- Lluch A. Concepto de muñeca SNAC: Scaphoid Non-union Advanced Collapse. Rev Ortop Traumatol. 1998;42 suppl 1:33–8.
- Amadio PC, Berquist TH, Smith DK, Ilstrup DM, Cooney WP, Linscheid RL. Scaphoid malunion. J Hand Surg Am. 1989;14A:679–87.

- Lindstrom G, Nystrom A. Incidence of posttraumatic arthrosis after primary healing of scaphoid fractures: a clinical and radiological study. J Hand Surg Br. 1990;15B:11–3.
- Birchard D, Pichora D. Experimental corrective scaphoid osteotomy for scaphoid malunion with abnormal wrist mechanics. J Hand Surg Am. 1990; 15A:863–8.
- Sanders WE. Evaluation of the humpback scaphoid by computed tomography in the longitudinal axial plane of the scaphoid. J Hand Surg Am. 1988;13A: 182–7.
- 22. Buck-Gramcko D. Denervation of the wrist joint. J hand Surg Am. 1977;2A:54–61.
- Rothe M, Rudolf KD, Partecke BD. Long term results following denervation of the wrist in patients with stages II and III SLAC-SNAC wrist. Handchir Mikrochir Plast Chir. 2006;38:261–6.
- Siegel DB, Gelberman RH. Radial styloidectomy; an anatomical study with special reference to radiocarpal intracapsular ligamentous morphology. J Hand Surg Am. 1991;16A:40–4.
- Nakamura T, Cooney WP, Lui W-H, Hangsvedt J-R, Zhao KD, Berlund L, Ann K-N. Radial styloidectomy: a biomechanical study on stability of the wrist joint. J Hand Surg Am. 2001;26A:85–93.
- Coen MS, Kozin SH. Degenerative arthritis of the wrist: proximal row carpectomy versus scaphoid excision and four-corner arthrodesis. J Hand Surg Am. 2001;26A:94–104.
- DiDonna ML, Kiefhaber TR, Stern PJ. Proximal row carpectomy: study with a minimum of ten years of follow up. J Bone Joint Surg. 2004;86A:2359–65.
- Strauch RJ. Scapholunate advanced collapse and scaphoid non non-union advanced collapse arthritis – update on evaluation and treatment. J Hand Surg. 2011;36A:729–35.
- Watson HK, Ryu J. Degenerative disorders of the carpus. Orthop Clin North Am. 1984;15:337–53.
- Vance MC, Hernandez JD, DiDonna ML, Stern PJ. Complications and outcome of four-corner arthrodesis: circular plate fixation versus traditional techniques. J Hand Surg Am. 2005;30A:1122–7.
- 31. Gaston RG, Greenberg JA, Baltera RM, Mih A, Hastings H. Clinical outcomes of scaphoid and triquetral excision with capitolunate arthrodesis versus scaphoid excision and four-corner arthrodesis. J Hand Surg Am. 2009;34A:1407–12.
- Calandruccio JH, Gelberman RH, Duncan SF, Goldfarb CA, Pae R, Gramig W. Capitolunate arthrodesis with scaphoid and triquetrum excision. J Hand Surg Am. 2000;25A:824–32.
- Kirschenbaum D, Schneider LH, Kirkpatrick WH, Adams DC, Cody RP. Scaphoid excision and capitolunate arthrodesis for radioscaphoid arthritis. J Hand Surg Am. 1993;18A:780–5.
- Dimitrios G, Athanasios K, Ageliki K, Spiridon S. Capitolunate arthrodesis maintaining carpal height for the treatment of SNAC wrist. J Hand Surg Eur Vol. 2010;35B:198–201.

- Viegas SF. Limited arthrodesis for scaphoid nonunion. J Hand Surg Am. 1994;19A:127–33.
- Werner FW, Short WH, Fortino MD, Palmer AK. The relative contribution of selected carpal bones to global wrist motion during simulated planar and outof-plane wrist motion. J Hand Surg Am. 1997;22A: 708–13.
- Crisco JJ, Coburn JC, Moore DC, Akleman E, Weiss A-P C, Wolfe SW. In vivo radiocarpal kinematics and the dart thrower's motion. J Bone Joint Surg. 2005;87A:2729–40.
- Moritomo H, Murase T, Goto A, Oka K, Sugamoto K, Yoshikawa H. In vivo three-dimensional kinematics of the midcarpal joint of the wrist. J Bone Joint Surg. 2006;88A:611–21.
- Garcia-Elias M, Lluch A, Fereres A, et al. Treatment of radiocarpal degenerative osteoarthritis by radioscapholunate arthrodesis and distal scaphoidectomy. J Hand Surg Am. 2005;30A:8–15.
- 40. Swanson AB. Flexible implant arthroplasty for arthritis disabilities of the radiocarpal joint. A silicone rubber intramedullary stemmed flexible hinge implant for the wrist joint. Orthop Clin North Am. 1973;4:383–94.
- Stanley JK, Tolat AR. Long-term results of Swanson Silastic arthroplasty in the rheumatoid wrist. J Hand Surg Br. 1993;18B:381–8.
- Lluch A, Proubasta I. Les implants radio-carpiens de Swanson. Résultats à long terme. La Main. 1998; 3:176–84.
- Menon J. Universal total wrist implant: experience with a carpal component fixed with three screws. J Arthroplasty. 1998;13:515–23.
- Anderson MC, Adams BD. Total wrist arthroplasty. Hand Clin. 2005;21:621–30.
- Kamal R, Weiss A-P. Total wrist arthroplasty for the patient with non-rheumatoid arthritis. J Hand Surg Am. 2011;36A:1071–4.
- Ferreres A, Lluch A, del Valle M. Universal total wrist arthroplasty: midterm follow-up study. J Hand Surg Am. 2011;36A:967–73.
- Gupta A. Total wrist arthroplasty. Am J Orthop. 2008;37:12–6.
- Packer G. The Maestro total wrist replacement. In: Røkkum M, editors. Wrist and hand joint replacements. FESSH Instructional Course: Oslo; 2011. p. 78–83.
- Carstam N, Eiken O, Andren L. Osteoarthritis of the trapezio-scaphoid joint. Acta Orthop Scand. 1968; 39:354–8.
- Kessler I. Silicone arthroplasty of the trapeziometacarpal joint. J Bone Joint Surg. 1973;55B: 285–91.
- Swanson AB. Disabling arthritis at the base of the thumb: treatment by resection of the trapezium and flexible (silicone) implant arthroplasty. J Bone Joint Surg. 1972;54A:456–71.
- Stark H, Moore J. Fusion of the first metacarpotrapezial joint for degenerative arthritis. J Bone Joint Surg. 1990;59A:22–6.

- Oberlin C, Dunois O, Oberlin F. L'arthrose scaphotrapézo-trapézoïdienne. Son retentissement sur le carpe. Ann Chir Main. 1990;3:163–7.
- Wilhelm K, Rolle A, Hild A. The scaphoidtrapezium-trapezoid arthrosis. A clinical study 1982–1985. Unfallchirurg. 1989;92:59–63.
- Bhatia A, Pisoh T, Touam C, Oberlin C. Incidence and distribution of scaphotrapezotrapezoidal arthritis in 73 fresh cadaveric wrists. Ann Chir Main. 1996;15:220–5.
- Wadhwani A, Carey J, Propek T, Hentzen P, Eustace S. Isolated scaphotrapeziotrapezoid osteoarthritis: a possible radiographic marker of chronic scapholunate ligament disruption. Clin Radiol. 1998;53:376–8.
- Weinzweig J, Watson HK. Limited wrist arthrodesis. In: Watson HK, Weinzeig J, editors. The wrist. Philadelphia: Lippincott Williams & Wilkins; 2001. p. 521–43.
- Taleisnik J. The wrist. New York: Churchill Livingstone; 1985. p. 235.
- Linscheid RL, Lirette R, Dobyns JH. L'arthose dégénerative trapézienne. In: Saffar P, editor. La rhizarthrose. Paris: Exp Scientifique Française; 1990. p. 185–93.
- SicreG,LaulanJ,RouleauB.Scaphotrapeziotrapezoid osteoarthritis after scaphotrapezial ligament injury. J Hand Surg Br. 1997;22B:189–90.
- Pinto CL, Obermann WR, Deijkers RL. Nontraumatic multidirectional instability of the scaphotrapeziotrapezoid joint: a cause of scaphotrapeziotrapezoid osteoarthritis and static intercalated segment instability. J Hand Surg Am. 2003;28A:744–50.
- Cooney WP. Comentary: multidirectional instability of the scaphotrapeziotrapezoidal joint. J Hand Surg Am. 2003;28A:751–2.
- Mclean JM, Turner PC, Bain GI, Rezaian N, Field J, Fogg Q. An association between lunate morphology and scaphoid-trapezium-trapezoid arthritis. J Hand Surg Eur Vol. 2009;34E:778–82.
- Viegas SF. The lunatohamate articulation of the midcarpal joint. Arthroscopy. 1990;6:5–10.
- 65. Parellada AJ, Morrison WB, Reiter SB, Carrino JA, Kloss LA, Glickman PL, McLean M, Culp RW. Flexor carpi radialis tendinopathy: spectrum of imaging findings and association with triscaphe artritis. Skeletal Radiol. 2006;35:572–8.
- Wollstein R, Wandzy N, Mastella DJ, Carlson L, Watson HK. A radiographic view of the scaphotrapezium-trapezoid joint. J Hand Surg Am. 2005;30A:1161–3.
- Ferris BD, Dunnett W, Lavelle JR. An association between scapho-trapezo-trapezoid osteoarthritis and static dorsal intercalated segment instability. J Hand Surg Br. 1994;19B:338–9.
- Ashwood N, Bain GI, Fogg Q. Results of arthroscopic debridement for isolated STT arthritis. J Hand Surg Am. 2003;28A:729–32.
- Crosby EB, Linscheid RL, Dobyns JH Scaphotrapoezial trapezoidal arthrosis. J Hand Surg Am. 1978;3:223–34.

- Corbin C, Warwick D. Midcarpal instability after excision arthroplasty for scapho-trapezial-trapezoid (STT) arthritis. J Hand Surg Eul Vol. 2009;34B: 537–8.
- Garcia-Elias M, Lluch AL. Partial excision of the scaphoid: is it ever indicated? Hand Clin. 2001;17: 687–95.
- Garcia-Elias M. Letter. Midcarpal instability after excision arthroplasty for scapho-trapezial-trapezoid (STT) arthritis. J Hand Surg Eur Vol. 2010;35E:82.
- Lluch A. Silicone spacers. In: Simmen BR, Allieu Y, Lluch A, Stanley JK, editors. Hand arthroplasties. London: Martin Dunitz; 2000. p. 233–42.
- Pequinot JP, D'Ansnieres de Veigy L, Allieu Y. Traitement de l'arthrose STT par un implant en pyrocarbone. Premiere resultats. Chir Main. 2005;24: 117–206.
- Wollstein R, Watson HK. Scaphotrapezoid arthrodesis for arthritis. Hand Clin. 2005;21:539–43.
- Fortin PT, Louis DS. Long-term follow-up of scaphoid-trapezium-trapezoid arthrodesis. J Hand Surg Am. 1993;18A:675–81.
- Minamikawa Y, Peimer CA, Yamaguchi T, Medige J, Sherwin FS. Ideal scaphoid angle for intercarpal arthrodesis. J Hand Surg Am. 1992;17A:370–5.
- Hagert CG. The distal radioulnar joint in relation to the whole forearm. Clin Ortop. 1992;275:56–64.
- Amrami KK, Moran SL, Berger RA, Ehman EC, Felmee JP. Imaging the distal radioulnar joint. Hand Clin. 2010;26:467–75.
- Malgaine JF. Traité des fractures et des luxations, vol. 2. Paris: JB Brailliére; 1855.
- Moore EM. Three cases illustrating luxation of the ulna in connection with Colles's fracture. Med Rec. 1880;17:305–8.
- Darrach W. Partial excision of lower shaft of the ulna for deformity following Colles's fracture. Ann Surg. 1913;57:764–5.
- af Ekenstam F, Engkvist O, Wadin K. Results from resection of the distal end of the ulna after fractures of the lower end of radius. Scand J Plast Reconstr Surg. 1982;16:177–81.
- Field J, Majkowski RJ, Leslie IJ. Poor results of Darrach's procedure after wrist injuries. J Bone Joint Surg. 1993;75B:53–7.
- Bell MJ, Hill RJ, McMurtry RY. Ulnar impingement syndrome. J Bone Joint Surg. 1985;67-B:126–9.
- Tulipan DJ, Eaton RG, Eberhart RE. The Darrach procedure defended: technique and long-term follow-up. J Hand Surg Am. 1991;16-A:438–44.
- DiBenedetto MR, Lubbers LM, Coleman CR. Longterm results of the minimal resection Darrach procedure. J Hand Surg Am. 1991;16-A:445–50.
- Shaaban H, Giakas G, Bolton M, et al. The distal radioulnar joint as a load-bearing mechanism – a biomechanical study. J Hand Surg Am. 2004;29A: 85–95.
- Hagert E, Hagert CG. Understanding stability of the distal radioulnar joint through an understanding of its anatomy. Hand Clin. 2010;26:459–66.

- Bowers WH. Distal radioulnar joint arthroplasty. The hemiresection interposition technique. J Hand Surg Am. 1985;10A:169–78.
- Watson HK, Ryu J, Burgess R. Matched distal ulnar resection. J Hand Surg Am. 1986;11A:812–7.
- Watson HK, Gabuzda GM. Match distal ulnar resection for post-traumatic disorders of the distal radioulnar joint. J Hand Surg Am. 1992;17A: 724–30.
- Swanson AB. Inplant arthroplasty for disabilities of the distal radioulnar joint. Use of a silicone rubber capping implant following resection of ulna head. Orthop Clin North Am. 1973;4:373–82.
- Van Schoonhoven J, Herbert TH, Krimmer H. Neue Konzepte der Endoprothetik des distalen Radioulnargelenkes. Handchir Mikrochir Plast Chir. 1998; 30:387–92.
- Berger RA, Cooney III WP. Use of an ulnar head endoprosthesis for treatment of unstable distal ulnar resection: review of mechanics, indications, and surgical technique. Hand Clin. 2005;21:603–20.
- Masaoka S, Longsworth SH, Werner FW, Short WH, Green JK. Biomechanical analysis of two ulnar head prostheses. J Hand Surg Am. 2002;27A:845–53.
- Kopylov P, Tagil M. Distal radio ulnar joint replacement. Tech Hand Up Extream Surg. 2007;11: 115–20.
- Garcia-Elias M. Eclypse: partial ulnar head replacement for the isolated distal radio-ulnar arthrosis. Tech Hand Up Extrem Surg. 2007;11:1–9.
- Scheker LR. Distal radio-ulnar joint prostheses to rescue the so-called salvage procedures. In: Simmen BR, Allieu Y, Lluch A, editors. Hand arthroplasties. London: Martin Dunitz; 2000. p. 151–8.
- Shecker LR. Implant arthroplasty for the distal radioulnar joint. J Hand Surg Am. 2008;33A: 1639–44.
- 101. Laurentin-Pérez LA, Goodwin AN, Babb BA, Shecker LR. A study of functional outcomes following implantation of a total radioulnar joint prosthesis. J Hand Surg Eur Vol. 2008;33E:18–28.
- 102. Baldwin WI. Orthopaedic surgery of the hand and wrist. In: Jones R, editor. Orthopaedic surgery of injuries. London: Henry Frowde and Hodder & Stoughton; 1921. p. 241–82.
- 103. Sauvé K. Nouvelle technique de traitement chirurgical des luxations récidivantes isolées de l'extrémité inférieure du cubitus. J Chir. 1936;47:589–94.
- Steindler A. The traumatic deformities and disabilities of the upper extremity. Springfield: Charles C. Thomas; 1946. p. 333–6.
- 105. Buck-Gramcko D. On the priorities of publication of some operative procedures on the distal end of the ulna. J Hand Surg Br. 1990;15B:416–20.
- 106. Lauenstein C. Zur Behandlung der nach karpaler Vorderarmfraktur zurückbleibenden Störung der Pro-und Supinations-bewegung. Centralblatt für Chir. 1887;23:433–5.
- Berry JA. Chronic subluxation of the distal radioulnar articulation. Br J Surg. 1931;18:526–7.

- Arandes JM, Ferreres A. Letter. J Hand Surg Br. 1999;24B:755.
- Lluch AL, Garcia-Elias M. The Sauvé-Kapandji procedure: technical considerations. Orthop Surg Tech. 1995;9:67–70.
- 110. Lluch A, Garcia-Elias M. Arthrodesis of the distal radio-ulnar joint with pseudoarthrosis of the distal ulna: the Sauvé-Kapandji procedure. Paris: Elsevier; 2004; 55-290-A-20. p. 1–7.
- Lluch A, Garcia-Elias M. The Sauvé-Kapandji procedure. In: Slutsky DR, editor. Principles and practice of wrist surgery. Philadelphia: Elsevier; 2010. p. 335–44.
- Lluch A. The Sauvé-Kapandji procedure: indications and tips for surgical success. Hand Clin. 2010; 26:559–72.
- 113. Proubasta IR, De Frutos AG, Salo GB, et al. Sauvékapandji procedure using the herbert canulated bone screw. Tech Hand Up Extrem Surg. 2000;4:120–6.
- Kapandji IA. The Kapandji-Sauvé operation: its techniques and indications in non-rheumatoid arthritis. Ann Chir Main. 1986;5(3):181–93.
- 115. Nakamura R, Tsunoda K, Watanabe K, Horii E, Miura T. The Sauvé-Kapandji procedure for chronic dislocation of the distal radio-ulnar joint with destruction of the articular surface. J Hand Surg Br. 1992;17B:127–32.
- 116. Minami A, Suzuki K, Suenaga N, et al. The Sauvé-Kapandji Procedure for osteoarthritis of the distal radioulnar joint. J Hand Surg Am. 1995;20A:602–8.
- 117. Rothwell A, O'Neill L, Cragg K. Sauvé-kapandji procedure for disorders of the distal radioulnar joint: a simplified technique. J Hand Surg Am. 1996;21A:771–7.
- 118. Kapandji AI. Technique and indications of the Kapandji-Sauvé procedure in non-rheumatoid diseases of the wrist. In: Nakamura R, Linscheid RL, Miura T, editors. Wrist disorders. Current concepts and challenges. Tokyo: Springer; 1992. p. 275–84.
- Kapandji AI. Amélioration technique de l'óperation Kapandji-Sauvé, dite "Technique III". Ann Chir Main. 1998;17:78–86.
- Carter PB, Stuart PR. The Sauve-Kapandji procedure for post-traumatic disorders of the distal radioulnar Joint. J Bone Joint Surg. 2000;82B:1013–8.
- 121. Slater RR. The Sauvé-Kapandji procedure. J Hand Surg Am. 2008;33A:1632–8.
- 122. Ruby LK, Ferenz CC, Dell PC. The pronator quadratus interposition transfer: an adjunct to resection arthroplasty of the distal radioulnar joint. J Hand Surg Am. 1996;21A:60–5.
- 123. Taleisnik J. The wrist. New York: Churchill Livingstone; 1985. p. 430–2.
- 124. Sanders RA, Frederick HA, Hontas RB. The Sauvé-Kapandji procedure: a salvage operation for the distal radioulnar joint. J Hand Surg Am. 1991;16A: 1125–9.
- 125. Gordon L, Levinsohn DG, Moore SV, et al. The Sauvé-Kapandji procedure for the treatment of posttraumatic distal radioulnar problems. Hand Clin. 1991;7:397–403.

- Schneider LH, Imbriglia JE. Radio-ulnar joint fusion for distal radio-ulnar joint instability. Hand Clin. 1991;7:391–5.
- Condamine JL, Lebreton L, Aubriot JH. L'intervention de Sauvé-Kapadji. Ann Chir Main. 1992;11:27–39.
- Millroy P, Coleman S, Ivers R. The Sauvé-Kapandji Operation. Technique and results. J Hand Surg Br. 1992;17B:411–4.
- Garcia-Elias M. Soft tissue anatomy and relationship about the distal ulna. Hand Clin. 1998;14:165–76.
- Stuart PR. Pronator quadratus revisited. J Hand Surg Br. 1996;21B(6):714–22.
- 131. Gordon KD, Pardo RD, Johnson JA, et al. Electromyographic activity and strength during maximum isometric pronation and supination efforts in healthy adults. J Orthop Res. 2004;22:208–13.
- 132. Johnson RK, Shewsbury MM. The pronator quadratus in motions and stabilization of the radius and ulna at the distal radioulnar joint. J Hand Surg Am. 1976;1:205–9.
- McConkey MO, Schwab TD, Travlos A, Oxland T, Goetz T. Quantification of pronator quadratus

contribution to isometric pronation torque of the forearm. J Hand Surg Am. 2009;34A:1612–7.

- Spinner M, Kaplan EB. Extensor carpi ulnaris. Its relationship to the stability of the distal radio-ulnar joint. Clin Orthop. 1970;68:124–9.
- 135. Garcia-Elias M. Radioulnar instability. Curr Orthop. 1999;13:283–9.
- 136. Gabl M, Zimmermann R, Angermann P, Sekora P, Maurer H, Steinlechner M, Pechlaner S. The interosseous membrane and its influence on the distal radioulnar joint. J Hand Surg Br. 1998;23B:179–82.
- 137. Ward LD, Ambrose CG, Masson MV, Levaro F. The role of the distal radioulnar ligaments, interosseous membrane, and joint capsule in distal radioulnar joint capsule in distal radioulnar joint stability. J Hand Surg Am. 2000;25A:341–51.
- 138. Daecke W, Martini AK, Schneider S, et al. Amount of ulnar resection is a predictive factor for ulnar instability problems after the Sauve-Kapandji procedure: a retrospective study of 44 patients followed for 1–3 years. Acta Orthop. 2006;77:290–7.

Osteoarthritis of the Thumb

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Keywords

Osteoarthritis • Carpometacarpal • Trapeziometacarpal • Scaphotrapezial • Thumb • Interphalangeal joint • Metacarpophalangeal joint • Fusion • Arthrodesis • Arthroplasty • Arthroscopic • Outcomes • Complications

Introduction

The hand is a common site of peripheral joint involvement in osteoarthritis (OA) and, although often underestimated as a cause of disability, the effect on the quality of life, from limitations in performing activities of daily living such as dressing and feeding, may be considerable. One longitudinal radiographic study of 751 patients over a 24 year period, showed that in those without OA at baseline, women had more incident disease than men in almost all hand joints, but the joints most frequently affected were the same in both sexes: the distal interphalangeal (DIP), followed by the base of the thumb, proximal interphalangeal (PIP) and metacarpophalangeal (MP) joints [1]. In another radiographic study of 3,327 men and women between the ages of 40-80+, the DIP joint demonstrated the highest OA prevalence, while the PIP joint showed the lowest. Jointspecific hand OA prevalence rates for the second DIP, third PIP and trapeziometacarpal (TM) joint were 35, 18 and 21 %, respectively. Women demonstrated higher hand OA prevalence rates for the three sites examined [2]. The age adjusted prevalence of CMC arthritis based on radiographic evidence has been reported to be 15 % for the female population and 7 % for the male population [3]. The prevalence increases to 33 % for the postmenopausal female population. This predilection for the thumb axis makes it one of the most expensive and debilitating diseases in terms of cost of diagnosis, treatment and lost productivity.

Osteoarthritis is not merely a wear and tear or age related phenomenon, but it is a common disease of articular cartilage that becomes more prevalent with advancing age. The biomechanical loads are of importance. In a biomechanical study, Cooney and Chao demonstrated that a pinch force of 1 kg at the thumb tip was amplified to 3.68 kg at the IP joint, 6.61 kg at the MCP joint and up to 13.42 kg at the TM joint. The typical joint compression forces averaged three kilograms of force at the interphalangeal joint, 5.4 kg at the metacarpophalangeal joint and 12.0 kg at the TM joint during simple pinch. Compression

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forces of as much as 120 kg may occur at the TM joint during strong grasp [4]. The alterations in the contact forces that may occur after injury or surgery to the TM joint due to ligamentous insufficiency, can lead to even higher forces, which can accentuate the wear on the articular cartilage. In this chapter the discussion will focus on OA affecting the thumb interphalangeal (IP) joint, the MP and the trapeziometacarpal (TM) joints.

History and Examination

IP and MP Joints

Similar to other joints, underlying OA of the thumb joints may be asymptomatic. The symptomatic patient, however, will typically present with complaints of joint stiffness, possible instability and pain. With IP involvement, one sees joint enlargement (Heberdens node) and possibly joint subluxation with flexion/extension or varus/ valgus deformities due to collateral ligament incompetence. A palpable joint effusion is not common as compared to an inflammatory arthritis. There may be crepitus with motion and/or loading, but this is not always painful. A mucoid cyst, with or without a nail groove, is a frequent association and may require separate treatment. With MP joint involvement, one will also see joint enlargement (Bouchards node), loss of motion and possibly instability. An MP joint hyperextension deformity is often associated with the lateral subluxation of the thumb metacarpal that occurs with TM OA and must be addressed separately. A flexion deformity seems to be more common in primary MP OA. Stress testing of the radial and ulnar collateral ligaments in extension and flexion may reveal a chronic ulnar or radial collateral ligament tear, which can be a primary cause of isolated post traumatic OA.

The radiographic evaluation of the IP and MP joint of the thumb includes posteroanterior, lateral and oblique views. Radiographically, the features of OA include joint space narrowing due to the erosion of the articular cartilage, the development of periarticular osteophytes and subchondral cysts.

Trapeziometacarpal Joint

The patient who presents with basal joint arthritis may complain of palmar-sided pain, which is frequently localized to the thenar eminence and may radiate up the radial wrist. Complaints of thumb weakness and clumsiness with fine manipulation tasks are common. On inspection, one may see a prominent TM joint, due to lateral subluxation of the thumb metacarpal base with or without marginal osteophytes and synovitis. There is often a loss of joint motion, especially thumb retropulsion and there may be a contracted first web space which interferes with grasping large objects. MP joint hyperextension may occur as an adaptive response to increase the first web span. There may be thenar muscle weakness and atrophy due to misuse. A concomitant carpal tunnel syndrome, however, should be sought by history of a sensory loss in the median nerve distribution, as well as through physical findings, which include a tinels sign over the carpal tunnel and a positive Phalen's test or median nerve compression test. On palpation, the patient will often have tenderness localized to the TM joint and the scaphoid tuberosity as well as a positive scaphoid shift test, but this may also occur with scapholunate instability or scaphotrapeziotrapezoidal (STT) OA, which should be ruled out. Flexor carpi radialis tendinitis can also present with tenderness over the scaphoid tuberosity. The trapeziometacarpal grind test will be positive in the face of TM OA and can help to distinguish these entities. The test is performed by applying an axial load to the thumb metacarpal, combined with manipulation of the metacarpal in a dorsal and volar direction. A positive test will produce variable degrees of crepitus and pain, depending on the stage of the arthritis. Alterations in grip and pinch strengths are documented in order to gauge the effects of treatment, but are nonspecific findings.

The radiographic evaluation of the thumb carpometacarpal joint includes a true anteroposterior view, which is performed by placing the forearm in maximum pronation, with the dorsal aspect of the thumb resting on the x-ray table and a true lateral view. A radial stress view of the thumb can

 Table 5.1
 Eaton classification [5]

Stage I	Normal articular surfaces, $\leq 1/3$ subluxation
Stage II	Mild joint space narrowing, >1/3 subluxation, normal STT.
Stage III	Significant joint space narrowing, peripheral osteophytes>2 mm in diameter, normal STT joint.
Stage IV	Pantrapezial OA.

be performed by asking the patient to push the radial borders of their thumbs together. This can demonstrate the degree of joint laxity by the amount of lateral subluxation of the metacarpal base. Littler and Eaton described a radiographic staging classification of TM OA, which is in wide use (Table 5.1) [5]. Stage I comprises normal articular surfaces without joint space narrowing or sclerosis. There is less than 1/3 subluxation of the metacarpal base. Stage II reveals mild joint space narrowing, mild sclerosis or osteophytes<2 mm in diameter. Instability is evident on stress views with > 1/3 subluxation. The STT joint is normal. In stage III there is significant joint space narrowing, subchondral sclerosis and peripheral osteophytes>2 mm in diameter, but a normal STT joint. In stage IV there is pantrapezial OA with narrowing, sclerosis and osteophytes involving both the TM and STT joints. Burton modified this classification by incorporating the clinical findings [6]. Stage I includes ligamentous laxity and pain with forceful and/or repetitive pinching. The joint is hypermobile, which can be seen on stress views, but the x-rays are normal. In stage II crepitus and instability can be demonstrated clinically, whereas x-rays reveal a loss of the joint space. Stage III and IV are similar to Eaton's classification.

Etiology

MP and IP Joints

Although mechanical stress has been postulated to play a role in the development of thumb IP and MP joint OA, handness has not been shown to have a clear association with either [7]. Post traumatic OA, following an intra-articular fracture, can occur at either joint, in addition to MP joint OA joint can occur as a sequelae of long standing radial and ulnar collateral ligament instability.

Trapeziometacarpal Joint

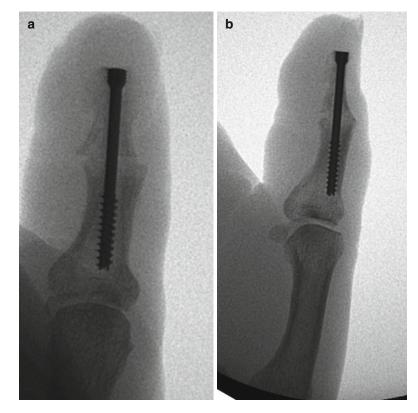
Primary thumb osteoarthritis typically presents at the TM joint. Women are affected more frequently than men, which may be the result of anatomic variations between the sexes [8] or repetitive activities, but an occupational causation has been hard to prove. The deep anterior oblique ligament, or volar beak ligament and the dorsal radial ligament have been shown to be the principal checkreins to dorsal subluxation during physiologic motion of the trapeziometacarpal joint. During key pinch, the incongruity of the articular surfaces causes apex loading on the volar articular surface of the trapezium, which transmits loads that are as high as 13 times the joint reactive force [9]. Because of the repeated eccentric loading, osteoarthritic changes begin volarly. Any laxity or incompetence to the anterior oblique ligament allows this fulcrum to move dorsally and adds to the eccentric force concentration. Post traumatic OA can also be seen after mal-reduced intra-articular fractures or sepsis.

Treatment

IP Joint

Save for historical reports [10], implant arthroplasty for the IP joint is not common. The mainstay of treatment consists of activity modification with the use of assistive devices and removable static IP joint splints. NSAIDs and selected cortisone injections are often beneficial, provided medical monitoring is instituted with chronic use. With unrelieved pain or disabling joint instability an IP joint fusion is the procedure of choice. The techniques are straightforward. Most consist of a dorsal capsulotomy, followed by removal of the joint surfaces and immobilization of the joint with percutaneous k-wires or a

Fig. 5.1 IP joint fusion. (a) AP view following an IP joint fusion using a headless screw. (b) Lateral view revealing early fusion with the joint in neutral (Published with the permission of David J Slutsky MD)



headless compression screw. Cobb has described an arthroscopic assisted procedure using a headless screw [11].

IPJ Arthrodesis

A longitudinal or S-shaped incision is made, centered over the IP joint. The extensor mechanism and dorsal capsule are divided in the midline, keeping the tissue planes separate for soft tissue closure over any exposed hardware. The collateral ligaments are released by sliding a scalpel blade underneath the insertion of the ligament on the neck of the proximal phalanx and cutting upwards to prevent injury to the neurovascular bundles. It is my preference to use a cup and cone arthrodesis, which allows one to adjust the amount of rotation and varus/valgus without increasing the risk of joint incongruity. The joint can be fused in neutral or 15-25° of flexion (Fig. 5.1a, b). A rongeur is used to gently remove any cartilage remnants on the head of the proximal phalanx, while preserving the normal convexity of the condyles since the metaphyseal bone is often quite soft, which can

lead to excessive bone removal. The cartilage and subchondral bone is removed from the base of the distal phalanx matching the curve of the head of the proximal phalanx and preserving a circumferential rim of cortical bone. Two convergent double ended 0.035 (0.9 mm) or 0.045 in. (1.1 mm) K-wires are advanced in a retrograde fashion through the base of the distal phalanx and out through the thumb tip. Ideally the k-wires should be placed immediately underneath the nail plate. The joint surfaces are then manually compressed in the desired degree of rotation and in neutral angulation or mild flexion, while the k-wires are advanced down the intramedullary canal of the proximal phalanx, until they abut the cortex or subchondral bone plate of the phalangeal base. The k-wire position is checked fluoroscopically and adjusted as necessary. Any defects at the fusion site can be packed with local bone graft or bone substitute. The capsule and extensor mechanism are closed in layers, followed by tourniquet release and skin closure.

When a headless screw is used, the joint is fused in neutral to prevent screw cut out and to

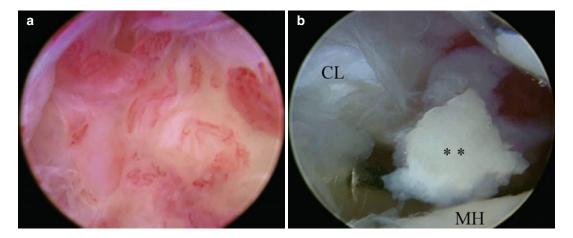


Fig. 5.2 MP joint arthroscopy. (a) MP joint arthroscopy demonstrating a diffuse synovitis. (b) Demonstration of a loose body (*asterisks*). *MH* metacarpal head, *CL* collateral ligament (Published with the permission of David J Slutsky MD)

permit adequate insertion down the intramedullary canal of the proximal phalanx. One of the k-wires is used as a guide wire to allow screw insertion in a retrograde fashion through the distal pulp. Due to the small canal size, caution is needed to prevent screw cut out in the distal phalanx or interference with the nail matrix. The screw head should be buried in the distal tuft to prevent tenderness with tip pinch, while insuring there are adequate numbers of screw threads (at least 4) crossing the fusion site. Post operatively, the IP joint is immobilized for 6–8 weeks until there are radiographic signs of union at which time a home program of gradual strengthening is instituted.

Outcomes

Most series are concerned mostly with the results of DIP arthrodesis and include but a few cases of thumb IPJ arthrodesis. Sieber and Segmuller, however, reported a series of 32 IP joint fusions using a figure-of-eight tension band wire and two crossed K-wires. They adapted the angle of fusion based on the patient needs: for instrument grip, the IP joint was fused in 5-10° of flexion, while for precision grip a larger angle of $20-30^{\circ}$ was preferred. They believed tactile gnosis was best preserved when pronation of 5-10° and slight ulnar deviation of 5° of the distal phalanx, in relation to the proximal phalanx, was achieved. They also switched from an H-shaped incision to a fork shaped incision to better preserve distal sensation [12]. Stern and Fulton reviewed 131 patients (61 with OA) who underwent a distal joint fusion, 37 of whom had an IP joint fusion. Techniques included crossed K-wires with and without cerclage wires or Herbert screws, but the breakdown by joint was not listed. There were 4/37 IPJ non-unions as compared to 17/144 DIP non-unions. Time to union was not specified by joint, but averaged 9 weeks for k-wires, 7 ½ weeks with cerclage wires and 10 weeks with screws. There were two thumb infections vs. four finger infections [13]. Clearly the data is lacking to recommend one type of fixation method over another with IP joint OA, hence surgeon preference largely dictates the technique.

MP Joint

The MP joint can be adequately splinted using a custom palmar based thumb spica splint. NSAIDs and activity modification, with avoidance of repetitive pinching exercises, will provide adequate pain control in many cases. One or two selected cortisone injections can provide temporary relief, but should not be used as a chronic form of treatment. Thumb metacarpophalangeal joint arthroscopy can be of use for staging the degree of articular involvement, synovectomy and removal of loose bodies in early OA (Fig. 5.2a, b). Thumb MP arthroplasty for OA is not commonly performed. Historically, a few investigators have attempted this with mixed results. Brannon and Klein reported the first

prosthetic MP joint in 1959. This early prosthesis was a hinged replica of the MP joint and was constructed of stainless steel initially and then titanium [14]. Moreover, these implants were shown to corrode after some time and the bone was unable to tolerate the metal.

Swanson popularized the use of Silastic arthroplasty using a silicone dioxide elastomer, which was used as an elastic spacer for the treatment of rheumatoid arthritis in the finger MP joints. Swanson and Herndon reported on their experience, with its use for rheumatoid thumb deformities (mostly Boutonniere) in 44 rheumatoid thumb MP joint, with good to excellent results at a 2–6 years follow up [15]. In a longer term study of 38 patients with 43 implants, good pain relief and function was noted with only one revision at a mean follow up of 6 1/2 years (3–13 years) [16].

In 1981, Beckenbaugh and Steffee reported their experience with a two-part metal and polyethylene prosthesis that were snap locked together. Their series included 8/42 MP joint replacements in patients with MP OA. Due to the limited arc of motion (16°) and equal functional and aesthetic outcomes, as compared to an MP fusion, this prosthesis was largely abandoned [17]. Pyrocarbon implants are currently in vogue for treatment of MP OA in the finger joints, but studies on their use in the thumb MP joint are lacking.

MP Joint Arthroscopy

The procedure is often done under tourniquet control with the 10 lbs. of thumb distraction using a wrist tower and a 1.9 mm, 30° angle arthroscope. A 2.0-mm shaver is useful, but a thermal probe can be used sparingly and with caution due to the risk of thermal injury to the articular cartilage and/or tendon in a small joint. The two portals are localized with a 22 gauge needle on either side of the extensor hood, followed by a shallow skin incision and insertion of the cannula and scope. The volar aspect of the joint is poorly seen, but the volar plate can be elevated with percutaneous insertion of a freer elevator. A synovectomy and removal of loose bodies can provide symptomatic comfort. Small chondral defects can be drilled to stimulate fibrocartilage formation. Following portal closure

a bulky dressing is used for the first week, followed by thumb mobilization.

MP Fusion

The most popular form of treatment for painful MP joint OA or instability is an arthrodesis. There are a number of options including K-wire fixation, with and without adjuvant tension band fixation, screw and plate fixation. According to Inglis et al. the MP joint of the thumb should be fused in approximately 15° of flexion, along with mild pronation and abduction of the proximal phalanx to facilitate tip pinch with the fingers [18].

Most procedures begin with a straight or S-shaped incision centered over the MP joint, followed by a midline incision through the extensor hood and capsule. A cup and cone technique is my preference, similar to the IP joint, with the thumb flexed approximately 15°. With severe deformity or bone loss, matching bone cuts can be made, but it is more difficult to achieve congruity at the fusion site if the MP joint is placed in flexion. The articular cartilage and subchondral bone is removed, preserving the matching convexity/concavity of the joint surfaces. The joint is held in mild flexion and two convergent 0.035 or 0.045 in. Kirschner wires are inserted through the dorsal neck of the thumb metacarpal neck and advanced distally down the intramedullary canal of the proximal phalanx to the subchondral bone at the IP joint, or alternatively engaging the palmar cortex at the midportion of the proximal phalanx. A transverse bone tunnel is drilled dorsal to the neutral axis at least 5 mm from the fusion site, with a 0.045 in. k-wire to prevent cut out. A 22- gauge wire strand is passed through the bone tunnel, looped in a figure of eight fashion over the longitudinal pins and simultaneously twisted on the radial and ulnar sides just until the point of deformation of the twisted loop is seen. The K-wires are then withdrawn 5 mm, bent 90° using a needle holder and suction tip and then tamped back in to place capturing the tension band loop with the hooked ends, which are buried in bone. Bone graft substitute is used to fill any gaps at the fusion site. All sharp metal ends are trimmed and the capsule is closed over the exposed hardware (Fig. 5.3a, b). The extensor mechanism and skin are repaired,

5 Osteoarthritis of the Thumb



Fig. 5.3 MP joint fusion. (a) Thumb x-ray demonstrating MP and IPJ OA. (b) AP view following a tension band arthrodesis of the MP joint and IP fusion with a headless

followed by a thumb spica splint for 6–8 weeks, until there is radiographic evidence of fusion.

One large diameter or two smaller diameter headless screws can be used in lieu of k-wire

screw. (c) Lateral view (Published with the permission of David J Slutsky MD)

fixation, provided there is adequate bone stock. A dorsally applied 1.5–2.0 mm nonlocking/locking plate can be used in cases with bone loss or for revision surgery. OA of the IP and MP joints can be treated with fusion.

Outcomes

Stanley et al. reviewed 42 cases of MP arthrodesis using a chevron arthrodesis. Most of the cases were for inflammatory arthritis with an 83 % patient satisfaction rate. Seven patients had continued pain or instability [19]. Schmidt et al. used a cup and cone technique, using a 3.0 mm cannulated AO screw and threaded washer in 26 patients. The indications included instability (12), osteoarthritis (6), inflammatory arthritis (7) and paralytic boutonniere (1). Twenty-five of 26 joints went on to fusion at an average time of 10 weeks. There were no infections and no need for hardware removal [20]. Bicknell, MacDermid and Roth performed a retrospective review of an interesting method of MP fusion used by Roth. This consisted of bone excision creating two flat surfaces, followed by the use of a single 0.062 in. k-wire inserted in a retrograde manner across the IP and MP joints with the MP in 10° of flexion and neutral deviation. The average age of the 28 patients was 49±17 years. The indications included instability (9), inflammatory arthritis (18), OA (5), trauma (5) and skeletal dysplasia (1). The patients were casted for 4 weeks and splinted until there was radiographic union, followed by pin removal at an average of 78±32 days. Older patients took longer to unite, 209 days in one case! Although the mean DASH score was 28 ± 24 , dexterity, hand function and pinch was not significantly different from the opposite side and patient satisfaction was high. There were three complications consisting of pin loosening (1) and superficial infection (2). Twenty-five patients (89 %) were able to return to their previous occupations. There were no radiographic malunions or nonunions [21].

Treatment: Trapeziometacarpal Joint

Non-operative

Similar to other joints, the radiographic severity of osteoarthritic changes at the trapeziometacarpal joint does not correlate with the severity of clinical symptoms. The main thrust of treatment is pain management. A trial of activity modification and splinting should in general be undertaken in any patient prior to any surgical consideration. This involves avoidance of any repetitive pinching or grasping activities and the use of assistive devices as needed. Therapy may be useful for retaining range motion and augmenting thumb stability, whereas strengthening exercises are generally avoided while the patient has pain. NSAIDs are commonly used in addition to a limited number of selected cortisone injections in the trapeziometacarpal joint for flare-ups or persistent pain that is unresponsive to conservative measures. Hyaluronic acid injections are still investigational, but do not appear to be superior to steroid injections. The use of splints can provide pain relief and help enforce activity modification. In general a forearm based thumb spica splint, with the thumb held in palmar abduction, can be used on a full-time basis until the pain has been controlled and then intermittently as needed. Whether the IP joint is immobilized is largely dependent upon patient and surgeon preference. A palmar based thumb spica splint which immobilizes the trapeziometacarpal joint by abducting the thumb, can provide pain relief but may be more functional.

Surgical

There are a myriad of techniques for the open treatment of trapeziometacarpal osteoarthritis. There has been a step wise evolution in these procedures over time, which has created a bewildering number of surgical choices. It is therefore instructive to discuss these options in light of the stage of the disease. In the early stages, the treatment options have included a volar ligament reconstruction without bony resection or an extension osteotomy of the thumb metacarpal. A variety of less invasive arthroscopic resection techniques have recently emerged. In more advanced stages, various authors have advocated for a partial or complete trapezium excision with or without ligament reconstruction and with or without either tendon or an artificial interposition substance. Joint resurfacing arthroplasty or total joint replacement will not be specifically discussed, due to the paucity of long-term clinical data.

Stage I – II Disease

Volar Ligament Reconstruction

This procedure is mostly indicated in patients with painful symptomatic laxity of the trapeziometacarpal joint, or stage I disease. The joint surfaces are typically normal, but there may be trapeziometacarpal synovitis. An incision is made along the radial border of the thumb metacarpal, curving proximally to the radial border of the FCR tendon preserving any sensory nerve branches. The thenar muscles are elevated from the metacarpal, along with an exposure of the TM and STT joints. A volar to dorsal drill hole of progressively larger diameters is created using a cannulated 3.0 mm drill guide across the metacarpal base, parallel to the TM joint and perpendicular to the metacarpal long axis. The TM joint is reduced and temporarily pinned in abduction with a 0.045-in. K wire. The FCR tendon is identified in the proximal extent of the incision. A number of small transverse incisions are made proximally along the course of the tendon up to its musculotendinous junction. The tendon is then split in to a radial and ulnar half at the wrist incision. The tendon is split into two halves at the wrist and a 26 gauge wire loop is used to split the tendon proximally by grasping the loop with a needle holder and then pulling the wire proximally through each transverse incision. Since the FCR tendon rotates 180° as it courses distally, the ulnar half of the FCR is released at the musculotendinous junction since it lies radially at the wrist incision. The volar portion of the transverse carpal ligament is split and the superficial communicating branch of the radial artery is either retracted or ligated and divided. If there is an associated carpal tunnel syndrome, a carpal canal can be decompressed by dividing the dorsal leaf of the transverse carpal ligament which is also the floor of the FCR sheath. The FCR graft is then pulled in to the TM incision, while preserving its attachment onto the base of the index metacarpal. A suture retriever is used to pass the free end of the FCR graft through the drill hole from volar to dorsal, where it is sutured to the periosteum with a 3-0 non-absorbable suture. The remaining end of the graft is passed through the abductor pollicis longus tendon insertion and across the volar capsule and sutured at both

places. The tendon graft is then looped around the remaining FCR tendon and sutured to itself. The thenar muscles are repaired and the K-wire is removed. The thumb is immobilized in a spica splint, or cast for 3 weeks followed by mobilization and strengthening.

Stage I TM OA can be treated with a volar ligament reconstruction or a metacarpal extension osteotomy.

Outcomes

Eaton et al. had one of the largest long-term series, reviewing 50 patients with an average follow-up of 7 years. Intractable pain was the primary indication for surgery. Ninety-five percent of the patients with stage I or II disease achieved good or excellent pain relief. All stage I cases and 82 % of stage II cases showed no x-ray evidence of osteoarthritis up to 13 years postoperatively. Only 74 % of patients with stage III and IV disease achieved good or excellent results [22]. Lane and Henley included some of these patients in their review of 37 operations in 35 patients with an average follow-up of 5.2 years (range 1-17 years). Sixty-seven percent had excellent results and 30 % had good results, with almost universal partial or complete pain relief. All patients had a stable trapeziometacarpal joint and an improvement in pinch strength. There was no clinical or x-ray evidence of osteoarthritis in any patient at the final follow-up [23].

Thumb Metacarpal Extension Osteotomy

Pellegrini et al. demonstrated in a biomechanical study that the volar contact area of the trapeziometacarpal joint was unloaded and shifted more dorsally, as long as the location of the OA did not extend more dorsal than the midpoint of the trapezium, which provides the rationale for this procedure [24]. It is indicated in symptomatic Eaton stage I and II disease, for both pain relief and to restore thumb stability. A 3 cm dorsal incision is made from the base of the thumb metacarpal, moving distally. Sensory nerve branches are protected and retracted. A subperiosteal dissection is performed and a dorsally based 30° wedge of bone is resected 1 cm distal to the metacarpal base. The wedge of bone is removed and the distal metacarpal is extended to close the wedge, and then the osteotomy is held in place with kwires, staples or a plate.

Outcomes

There are no large series on this procedure. Tomaino prospectively evaluated 12 patients who were treated with an extension osteotomy, with an average follow-up of 2.1 years (range: 6–46 months). Eleven patients were satisfied with the outcome and the grip and pinch strength increased by an average of 8.5 and 3 kg, respectively. Parker reviewed Amadio's experience with 8 patients who were treated with this procedure, with stage I (3 patients), stage II (3 patients) and stage III (2 patients) TM osteoarthritis at a mean follow-up of 9 years (range, 6–13 years). The average lateral pinch strength was 5 kg or 129 %, oppositional pinch strength 3 kg or 103 % and grip strength 19 kg or 108 % of the opposite side. There was no radiographic progression of the arthritis in 5 of the 8 patients. Gwynne–Jones and colleagues reviewed the results of thumb extension osteotomies without internal fixation in 28 patients with a mean age 54 years (range, 30–69 years). At a mean follow-up of 34 months (range, 12-73 months), 21 patients with 22 procedures had good or excellent results, 2 had fair and one had a poor result. Three patients required further surgery for persistent pain. Key pinch, pulp pinch and tripod pinch were 22-32 % lower, but there was no decrease in grip strength. The Michigan Hand Outcomes Questionnaire scores increased by an average of 28 points (range, 1-56) points, with a significant improvement in pain (44 points), activities of daily living (41 points) and satisfaction (35 points) [25].

Stage II – IV Disease

Ligament Reconstruction Interposition Arthroplasty (LRTI)

This procedure, described by Burton and Pellegrini in 1986, is typically used for more advanced stage II – IV disease (Fig. 5.4a–h) [26].

An 8 cm dorsoradial incision is made along the radial border of the thumb metacarpal, curving down to the FCR tendon at the wrist. Branches of the superficial radial nerve and radial artery are identified and protected throughout the procedure. A longitudinal incision is made through the TM joint capsule. An oscillating saw is used to remove the base of the thumb metacarpal and the medullary canal is enlarged with a curette. A trapeziectomy is then performed by inserting a 0.62 in. K-wire to act as a joy stick, by sequentially elevating soft tissue restraints until the trapezium can be removed in one piece. Alternatively, the trapezium can be osteotomized into four sections and removed piecemeal, while taking care to avoid injury to the FCR tendon which is in the volar depths of the wound. Caution is needed to prevent injury to the radial artery in the snuffbox, which is just proximal to the STT joint. A 6 mm bony window is made with a power burr along the dorsal aspect of the thumb metacarpal, 1 cm distal to the base.

One half of the FCR tendon is harvested and pulled into the thumb incision as described above. Some authors prefer to use the entire FCR, without any apparent deleterious effects. The free end of the tendon slip is passed into the medullary canal of the thumb metacarpal and out the dorsal cortical window, where it is sutured to the periosteum. It is then looped around the metacarpal base and sutured to itself, while longitudinal traction is applied until the metacarpal base is even with the index CMC joint. Proper tensioning of the FCR graft helps prevent proximal migration and radial subluxation of the thumb. A percutaneous 0.045 in. k-wire can be inserted through the metacarpal base and into the distal scaphoid with the thumb in abduction to maintain the arthroplasty space. Two 2-0 non-absorbable sutures are then placed in the deep volar capsular. One of the sutures is left attached to the deep capsule and threaded through the distal end of the FCR tendon slip to create an anchovy interposition, which is then tied down to the capsule. The remaining deep suture is used to repair the capsule over the anchovy, in a vest over pants fashion. In the original surgical description, the extensor pollicis brevis is released from its insertion and tenodesed to

the metacarpal shaft proximal to the metacarpophalangeal joint, to eliminate the hyperextension moment acting on the proximal phalanx. The subcutaneous tissue and skin are closed in layers and a thumb spica splint is applied. Postoperatively, a thumb spica splint is maintained until 4 weeks at which time the k-wire is removed and thumb abduction and extension exercises are instituted. Opposition is started at 6 weeks, followed by progressive strengthening exercises. Variants of the LRTI have included the following: tendon interposition alone, ligament reconstruction alone, a partial or complete trapeziectomy with tendon interposition but no ligament reconstruction, a limited trapeziectomy with tendon interposition and volar ligament reconstruction, a trapezial resection suspensionplasty using the abductor pollicis longus tendon and a partial trapeziectomy with implantation of a synthetic biological implant. A double interposition



Fig. 5.4 LRTI. (a) Clinical appearance of advanced TM OA with contracted 1st web space. (b) AP x-ray view revealing obliteration of the joint space at the TM and STT joints (Eaton stage IV). (c) Harvesting of FCR tendon which is left attached to the index metacarpal base. (d) Creation of

a tendon anchovie after suspensionplasty with the FCR strip. (e) immediate postoperative AP x-ray. (f) Maintenance of arthroplasty space at the 1 year follow. (g) Improved thumb abduction at I year. (h) preserved thumb opposition at 1 year (Published with the permission of David J Slutsky MD)



Fig. 5.4 (continued)

resurfacing arthroplasty was described for stage IV disease and involves a TM joint and STT joint resection and interposition [27].

Stage II – IV TM OA can be treated with a variety of methods including implant arthroplasty, excisional arthroplasty with or without ligament reconstruction and with or without tendon interposition

Trapeziectomy

This procedure is used in stage III and IV disease. A simple trapeziectomy has been complicated by metacarpal subsidence, which can theoretically weaken the pinch strength and, in extreme cases, lead to metacarpal scaphoid impingement (Fig. 5.5). This procedure has largely been supplanted by a haematoma distraction arthroplasty, which consists of a trapeziectomy and temporary distraction pinning of the thumb metacarpal. This procedure relies on the development of a stable pseudarthrosis, that develops from the ingrowth of fibrous tissue which replaces the initial hematoma. The trapeziectomy is performed as described above. The thumb is then grasped and held in a position of wide palmar abduction, slight opposition, distraction and one or two 0.062 in. percutaneous K-wires are inserted from



Fig.5.5 Marked metacarpal subsidence at 6 years following a trapeziectomy (done elsewhere) with metacarpal-scaphoid impingement (Published with the permission of David J Slutsky MD)

the base of the thumb metacarpal in a transverse orientation into the base of the index metacarpal or the trapezoid. Pinning the thumb metacarpal base to the index for 5–6 weeks is integral to the procedure, but tendon interposition or reconstruction of the TM joint capsule is not.

Outcomes

Froimson described the use of a rolled up tendon anchovy for an interposition arthroplasty in 1970 [28]. Although he reported pain relief in 80 procedures and 72 patients, the long-term follow-up showed a 30 % reduction in pinch strength, in addition to a 50 % decrease in the arthroplasty space. Eaton and Littler introduced the concept of a beak ligament reconstruction in 1973 [5]. Although they removed any osteophytes, no trapeziectomy was performed. They reported that all the patients with stage II disease and 63 % with stage III disease had excellent results at 1 year. They subsequently modified their procedure by adding a resection of the second metacarpal base and distal trapezium with 92 % good or excellent results reported in 1985 [29]. In 1986 Burton and Pellegrini published their technique of a ligament reconstruction interposition arthroplasty (LRTI) [26]. They used 1/2 of the FCR tendon to reconstruct the volar oblique ligament combined with a partial or complete trapeziectomy and interposition of the remaining tendon as an anchovy. In a retrospective series of 25 thumbs, treated with an LRTI and hemitrapeziectomy (stage II and III), or complete trapeziectomy (stage IV, pantrapezial disease) at an average follow-up of 2 years, 92 % of the patients had excellent pain relief and an average increase in pinch and grip strength of 19 %.

De Smet et al. compared the outcomes of 22 patients who underwent a trapeziectomy and 34 patients were treated with an LRTI at a follow-up of 26–34 months. They found that the trapezial arthroplasty space was better maintained in the LRTI group with a loss of height of 32 % (compared to the preoperative level) as compared to a 57.5 % decrease in height in the trapeziectomy group. Despite this finding there were no statistically significant differences for pain relief, patient satisfaction, DASH score or grip strengths, but the key-pinch strength was correlated with the amount of metacarpal subsidence.

Mahoney and Meals believed that the poor results following a simple trapeziectomy reported by Gervis in 1949 [30] could be improved by adding k-wire immobilization of the thumb in a distracted position for 5-6 weeks, to allow the post-operative hematoma to organize and the surrounding capsular remnants to consolidate to sufficiently anchor the metacarpal base. They reported their results with this procedure in 26 patients, with a minimum 2 year follow-up. Ninety-two percent of the patients were pain free and the majority recovered normal thumb motion. They noted a 47 % improvement in grip and a 33 % increase in key pinch and 23 % improvement in tip pinch over the preoperative values. They did note, however, that there was more metacarpal subsidence as compared to a more formal ligament reconstruction, but this did not correlate with outcomes.

Davis et al. performed a prospective, randomized, controlled study on 183 patients with trapeziometacarpal osteoarthritis. They were randomized to either a simple trapeziectomy, trapeziectomy with palmaris longus interposition, or trapeziectomy with ligament reconstruction and tendon interposition using one half of the FCR tendon. K-wire fixation was used for 4 weeks in all patients [31]. At the 1-year follow-up, 82 % achieved good pain relief and good range of motion. This result was independent of the type of procedure that was performed. They concluded that the outcomes of these three variations of trapeziectomy were very similar at 1 year and that there appeared to be no benefit to tendon interposition or ligament reconstruction in the short term.

Wajon et al. performed a meta-analysis of the Cochrane database of seven studies, with 384 patients having Stage II or III disease who had undergone a trapeziectomy, trapeziectomy with interpositional arthroplasty, trapeziectomy with ligament reconstruction, trapeziectomy with ligament reconstruction and tendon interposition (LRTI) and joint replacement. No procedure demonstrated any superiority over another in terms of pain, function, range of motion or strength. However, participants who underwent trapeziectomy had 16 % fewer adverse effects (p=0<.001) [32].

Associated MP Joint Hyperextension

MP joint hyperextension often accompanies advanced trapeziometacarpal osteoarthritis, which produces a dorsal subluxation stress at the TM joint arthroplasty site and may lead to recurrent subluxation. Correction of this MP hyperextension reverses the metacarpal collapse and improves the force transmission along the thumb ray. If there is MP joint OA, an MP joint fusion is indicated. In cases where there is a mild MCP joint hyperextension (20-30°), Eaton has recommended transferring the EPB to the base of the thumb metacarpal, which eliminates the extension force on the MP joint and augments abduction of the thumb metacarpal [33]. The joint is pinned or casted in flexion for 4 weeks postoperatively. For MP joint hyperextension of 30° or more, an MP joint volar capsulodesis or a sesamoidesis is

added. In their series of MP capsulodesis of 13 patients with an average follow-up of 39 months, 10 patients had complete correction of the MP hyperextension deformity, with 9 excellent results, 3 good results, and 1 fair result.

MP joint hyperextension $> 30^{\circ}$ with TM OA should be treated with a capsulodesis or fusion.

Trapeziometacarpal Fusion

Trapeziometacarpal fusion has been advocated for patients with stage II and III disease with strong functional demands, such as manual laborers and patients younger than 40 years with posttraumatic osteoarthritis, since it provides a stable thumb ray with no risk of instability. The TM joint is exposed and the joint surfaces are denuded of cartilage and subchondral bone to create two parallel surfaces. The thumb is positioned in 35-45° of palmar abduction and 20-30° of radial abduction and pronation, so that the thumb pulp rests on the radial aspect of the index finger middle phalanx in mild opposition. K-wire, staple or plate fixation can be used. In cases of revision or if there is bone loss with a small trapezial remnant, a minicondylar blade plate is ideal. A short arm thumb spica cast is used for 6 weeks, followed by a splint, until there are signs of fusion.

Outcomes

Rizzo and co-workers examined the Mayo clinic experience of TM fusion for OA in 114 patients/126 thumbs at an average follow up of 11.2 years (range 3–28 years). The average patient age was 57 years (range 32–77). Supplemental bone graft was used in 90 thumbs. There were 17 nonunions, 9 of them requiring re-operation. The appositional pinch increased to 5.9 kg vs. 3.0 kg preoperatively, oppositional pinch 5.4 kg vs. 2.7 kg and grip strengths 23 kg vs. 14 kg. The average pain score decreased to 0.4 vs. 6.6 preoperatively (p < .01). Thirty-nine patients developed radiographic signs of STT OA, but only 8 patients were symptomatic. MP joint OA occurred in 16 patients, but no additional treatment was needed [34].

There are many published case series that examined the results of these disparate procedures. Taylor et al. performed a retrospective study of 83 patients with trapeziometacarpal joint osteoarthritis who were treated with a TM fusion (36), trapeziectomy with and without ligament reconstruction (25) or a Silastic replacement (22) [35]. The follow-up ranged from 1 to 5 years. There was a higher rate of complications and reoperation in the fusion group, but otherwise no significant differences in terms of patient satisfaction, range of movement, tip and key pinch.

Raven et al. reviewed 54 patients (74 thumbs) with TM OA who were treated with a trapeziectomy (18 thumbs), trapeziectomy with tendon interposition (17 thumbs) or trapeziometacarpal arthrodesis (28 thumbs). The average follow-up was 13 years for the resection group, 8 years for the trapeziectomy group and 9 years for the arthrodesis group. Patients in the trapeziectomy group had significantly less pain and better radial abduction as compared to the arthrodesis group. There was no difference among the three groups in grip and tip pinch strength. None of the patients in the trapeziectomy group needed a re-operation, one patient in the resection arthroplasty group had a re-operation and 22 patients in the arthrodesis group had one or more re-operations for hardware removal or because of a complication [36].

Arthroscopic Treatment

The good results that have been obtained with an open trapeziectomy provided the impetus for the development of arthroscopic techniques. Arthroscopic techniques for evaluating and treating trapeziometacarpal disease surfaced in 1994 [37, 38]. As a general rule, any patient who is an appropriate candidate for a hemiresection arthroplasty of the TM joint is also suitable for an arthroscopic hemi-trapeziectomy. This would typically include patients in Eaton stage II and stage III who have unremitting pain, despite appropriate conservative measures. This form of treatment does not preclude an open trapeziectomy and/or ligament reconstruction at a later date as a salvage procedure for failed arthroscopic surgery. The presence of Eaton Stage IV disease is a relative contraindication, although an arthroscopic hemitrapeziectomy, combined with an arthroscopic debridement or limited resection of the distal scaphoid, is an option. Badia proposed an arthroscopic classification of TM OA that is based upon the arthroscopic changes [39]. Stage I included intact articular cartilage, Stage II included eburnation on the ulnar 1/3 of the metacarpal base and central trapezium and Stage III comprised widespread full thickness cartilage loss on both surfaces. Based upon the intraoperative findings he recommended debridement for stage I, with thermal capsulorraphy in the presence of dorsal subluxation, extension/abduction osteotomy of the metacarpal base ± thermal shrinkage for stage II and an arthroscopic interposition arthroplasty for Stage III. He recommended an open arthroplasty in the presence of associated severe STT joint OA.

A 1.9 mm 30° angled scope, along with a camera attachment is used. A larger 2.7 mm scope may be substituted after the space has been partially decompressed. A 3 mm hook probe is needed for palpation of intracarpal structures. The use of an overhead traction tower greatly facilitates instrumentation. A motorized shaver is needed for debridement. Some type of diathermy unit is required if a capsular shrinkage is contemplated. Intraoperative fluoroscopy is employed to assess the adequacy of bone resection and for locating the portals as needed. The patient is positioned supine on the operating table with the arm extended on a hand table. The thumb is suspended by Chinese finger traps with 5 lb of counter traction, which forces the wrist into ulnar deviation. The relevant landmarks are outlined, including the proximal and dorsal edge of the thumb metacarpal base, the tendons of the abductor pollicus longus (APL) and the extensor pollicus longus (EPL) and the radial artery in the snuff box. The procedure is performed with a tourniquet elevated to 250 mmHg. Saline inflow irrigation is provided through the arthroscope and a small joint pump or pressure bag. To establish the 1-R portal, the thumb metacarpal base is palpated and the joint is identified with a 22 gauge needle just radial to the APL, followed by injection of

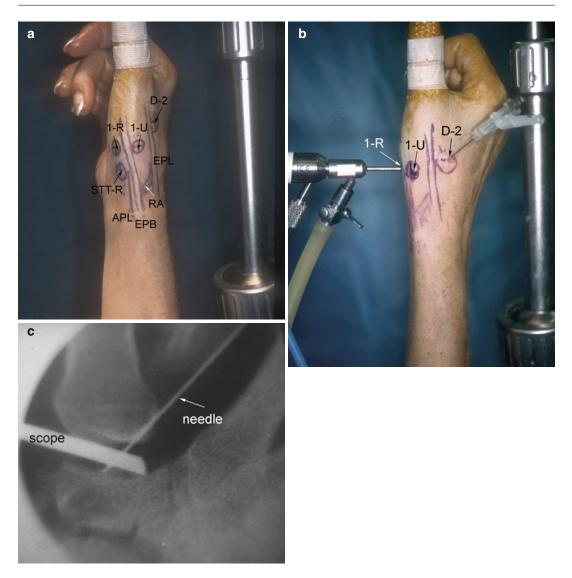


Fig. 5.6 (a) Surface landmarks for TM and STT portals. *APL* abductor pollicus longus, *EPB* extensor pollicus brevis, *EPL* extensor pollicus longus, *RA* radial artery

1–2 cc of saline. A small skin incision is made, followed by wound spread technique with tenotomy scissors. The capsule is pierced and a cannula and blunt trocar are inserted, followed by insertion of the arthroscope. An identical procedure is used to establish the 1-U portal, just ulnar to the EPB tendon, followed by insertion of a 3 mm hook probe. The portals are used interchangeably to systematically inspect the joint, which is facilitated by expedient use of a 2.0 mm synovial resector.

(**b**) Direction scope in 1-R portal with needle placement for the D-2 portal (**c**) Fluoroscopic view of scope and needle (Published with the permission of David J Slutsky MD)

Access to the medial trapezial osteophytes may sometimes be difficult, hence I have found the use of a distal – dorsal (D-2) accessory portal to be of some value [40]. Its main utility is that it allows one to look down on the trapezium rather than across it, which facilitates resection of any medial osteophytes (Fig. 5.6a–c). This accessory portal allows views of the dorsal capsule with rotation of the scope and facilitates triangulation of the instrumentation. It is situated in the dorsal aspect of the 1st web space. An anatomical study of 5 cadaver hands revealed that the D-2 portal surface landmark is ulnar to the EPL tendon and 1 cm distal to V-shaped cleft at the juncture of the index and thumb metacarpal bases. The portal lies just distal to the dorsal intermetacarpal ligament (DIML). To establish the D-2 portal, the intersection of the base of the index and thumb metacarpal are identified just distal and ulnar to the extensor pollicus longus (EPL) tendon. The course of the radial artery can be outlined by palpation or doppler prior elevation of the tourniquet. A 22 gauge needle is inserted 1 cm distal to this juncture and angled in a proximal, radial and palmar direction, hugging the thumb metacarpal. A small skin incision is made and tenotomy scissors are used to spread the soft tissue and pierce the joint capsule. This is followed by insertion of a blunt trocar and cannula and then the arthroscope, or alternatively a hook probe, motorized shaver or 2.9 mm burr.

Arthroscopic hemi-trapeziectomy is an option in Stage II and III TM OA

Arthroscopic Debridement and Capsular Shrinkage Interposition

The essence of arthroscopic capsular shrinkage is akin to that of an anterior oblique ligament reconstruction. It relies on thermal heating of the collagenous fibers in the surrounding ligaments and capsule, followed by a period of joint immobilization in a reduced position. A motorized shaver is used to debride any synovitis and to expose the capsular ligaments. A diathermy probe is then employed to paint the anterior oblique ligament and surrounding capsule, taking care to leave bands of tissue in between. The probe is kept away from the joint surfaces to prevent cartilage necrosis. In light of the meager joint volume, the outflow fluid temperature is frequently monitored to prevent overheating. Use of an 18 gauge needle as an accessory portal enhances fluid circulation, which minimizes this risk.

Arthroscopic Partial or Complete Trapeziectomy Without Tendon Interposition

The 1-R and 1-U portals are established as described. The anterior oblique ligament (AOL) is identified and preserved. After joint debridement a 2.9 mm burr is applied in a to and fro manner to resect the distal trapezium (Fig. 5.7a-c). The diameter of the burr, along with fluoroscopy, provide a gauge as to the amount of bony resection. A larger burr may be substituted, as the space between the metacarpal base and distal trapezium enlarges. It is crucial to remove any medial osteophytes, which will lead to impingement and possibly persistent pain. The D-2 portal is useful for this step since it allows one to debride the medial trapezium from above rather than from across the joint. Culp has recommended resecting at least 1/2 of the distal trapezium [41], although I have found that excising 3-4 mm is sufficient, provided that all of the medial osteophytes are removed. After the bony resection is complete, the thumb is k-wired in a pronated and abducted position (Fig. 5.8a–e). If there is lateral subluxation of the metacarpal base, thermal shrinkage of the anterior oblique ligament can be performed at this time. The thumb is immobilized in abduction by cast or splint for 4 weeks, at which time the k-wire is removed. In more recent years, I have abandoned k-wire immobilization and thermal shrinkage and start protected home motion exercises as pain permits at 1-2 weeks, without any deterioration in the outcomes. Thumb abduction and extension exercises are instituted, followed by adduction and opposition after 6 weeks. Strengthening ensues once motion has been restored. The rehabilitation protocol is modified as necessary if concomitant surgery on the MP joint has been performed.

Arthroscopic Partial or Complete Trapeziectomy with Interposition

After a partial or complete resection of the trapezium, autogenous tendon graft such as the palmaris longus, 1/2 of the flexor carpi radialis or a slip of the APL is harvested through multiple transverse incisions. Alternatively, some form of synthetic interposition material can be substituted. An absorbable suture is placed in the leading end of the

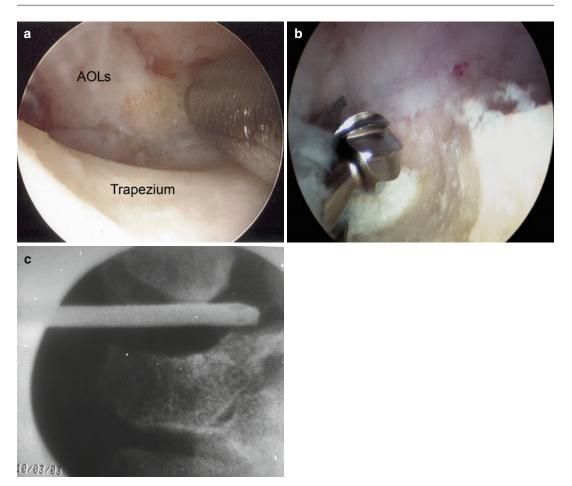


Fig. 5.7 (a) View of the distal trapezium from the 1-R portal. Probe is in the 1-U portal. *AOLs* superficial anterior oblique ligament. (b) 2.9 mm burr in the 1-U portal as

tendon graft and swedged onto a large curved needle, which is used to pass the graft through the joint. The needle is passed through the 1-U portal and brought out through the volar capsule and bulk of the thenar eminence. Traction on the suture pulls the graft into the joint. The remaining graft is packed in with forceps and the portals are closed. The thumb is k-wired in abduction for 4 weeks.

Outcomes

Menon reported his results on performing a partial arthroscopic resection of the trapezium and an interposition arthroplasty in 31 patients (33 hands) [37]. The mean age was 59 years (48–81 years) with an average follow up of 37.6 months (24–48 months). Gortex was used

seen from the 1-R portal. (c) Scope placement to check resection of medial osteophytes (Published with the permission of David J Slutsky MD)

in 19 patients and autogenous tendon or allograft in 14. Complete pain relief was obtained in 25 patients/hands (75.7 %). Three patients had mild pain (4 hands) and 4 patients had persistent pain that required conversion to an open trapeziectomy and ligament reconstruction. All patients maintained their pre-operative motion. Pinch strength improved from 6 p.s.i. preoperatively to 11.1 p.s.i. postoperatively. Because of osteolysis in 3 patients/4 hands, the use of Gortex as an interpositional substance was not recommended.

Hofmeister et al. reviewed the long term results in 18 patients following an arthroscopic hemitrapeziectomy, thermal capsular shrinkage and temporary K-wire fixation. At an average follow-up of 7.6years they noted a subjective improvement in pain,



Fig. 5.8 (a) Fifty-five years of male with left TM OA. Note the large medial osteophyte arising from the trapezium. (b) Arthroscopic TM arthroplasty. Arthroscope is in the D-2 portal, burr is in the 1-R portal. (c) X-ray after

pinch activities, strength and range of motion in all patients. No patient required further surgery on their thumb. No patient had a first carpal-metacarpal grind or laxity by exam. The total thumb range of motion decreased by 20 %, but all patients could oppose to the fifth finger. Grip strength remained unchanged, key pinch improved from 8 to 11 lbs., and tip pinch improved from 4 to 5 lbs. Radiographs showed a metacarpal subsidence of 1.8 mm (0–4 mm). Four complications were noted: two cases of dorsal radial nerve neuritis, one rupture of the flexor pollicis longus and one prolonged hematoma [42].

Edwards and Ramsey reported similar findings. They prospectively evaluated 23 patients with stage

partial trapeziectomy. (d) X-ray at 2 ½ years postoperative. (e) Clinical appearance (Published with the permission of David J Slutsky MD)

III OA at a minimum follow-up of 4 years, who were treated with pan arthroscopic hemitrapeziectomy and thermal capsular shrinkage, without interposition, plus k-wire fixation for 3–4 weeks. At 3 months postoperatively the average DASH score improved from 61 to 10 and pain scores decreased from 8.3 to 1.5. Grip and key pinch strength improved 6.8 and 1.9 kg respectively and the wrist and finger motion were unchanged. Proximal migration of the first metacarpal averaged 3 mm and translation decreased from 30 to 10 %. These findings remained unchanged at 4 years or more [43].

Pegoli et al. performed an arthroscopic hemitrapeziectomy and tendon interposition using the palmaris longus tendon in 16 patients with Stage I and II disease. At 12 months there were 6 excellent, 6 good, 3 fair and 1 poor result using the modified Mayo score [44]. Adams and Steinmann treated 17 patients with an arthroscopic debridement and interposition arthroplasty for stage II and III disease, using a folded acellular dermal matrix allograft. The average age was 61.7 (range 47-86) and the follow-up averaged 17 (range, 6-39) months. Eighty-eight per cent of the patients reported no pain, or only occasional pain, on activities with an average pain score of 1.125 out of 10. The average grip strength was 18.3 vs. 22.6 kg preoperatively and the average pinch strength was 4.0 kg vs. 4.8 kg. Only two patients had limited range of motion, as assessed by the palm flat test and the ability to oppose the thumb to the 5th metacarpal head. None of the patients required revision surgery and there were no instances of graft reaction [45].

Complications

The incidence and nature of the complications that may occur following TM joint surgery vary, depending upon whether the procedure is open or arthroscopic, whether temporary k-wire fixation is used and whether or not there is an interposition substance. Possible complications that are common to all of the procedures include injury to branches of the superficial radial nerve, infection metacarpal subsidence and subluxation of the thumb metacarpal. The use of autogenous tendon carries the risk of neurovascular injury during harvest of the graft. The use of a synthetic implant carries the risk of a foreign body reaction which was seen with silastic trapezial implants, has also been reported following the use of Gortex, polyurethane urea and acellular dermal grafts. Wound healing is less of a problem with arthroscopic techniques vs. open procedures, but residual trapeziometacarpal impingement or inadequate bone resection is more of a risk.

Conclusion

From the above discussion it is apparent that osteoarthritis of the IP and MP joints are commonly treated with fusion rather than arthroplasty, with generally favorable outcomes. The profusion of procedures for the treatment of trapeziometacarpal OA is a testament to the fact that most procedures provide satisfactory pain relief and function, whether performed arthroscopically or open. The type of procedure is largely dependent upon the surgeon's preference and the patient's needs. Similar to the hunt for the perfect mousetrap, innovative surgeons will no doubt continue to search for the ideal procedure, even though the simplest methods often suffice.

References

- Chaisson CE, Zhang Y, McAlindon TE, Hannan MT, Aliabadi P, Naimark A, Levy D, Felson DT. Radiographic hand osteoarthritis: incidence, patterns, and influence of pre-existing disease in a population based sample. J Rheumatol. 1997;24:1337–43.
- Wilder FV, Barrett JP, Farina EJ. Joint-specific prevalence of osteoarthritis of the hand. Osteoarthritis Cartilage. 2006;14:953–7.
- Xu L, Strauch RJ, Ateshian GA, Pawluk RJ, Mow VC, Rosenwasser MP. Topography of the osteoarthritic thumb carpometacarpal joint and its variations with regard to gender, age, site, and osteoarthritic stage. J Hand Surg Am. 1998;23:454–64.
- Cooney 3rd WP, Chao EY. Biomechanical analysis of static forces in the thumb during hand function. J Bone Joint Surg Am. 1977;59:27–36.
- Eaton RG, Littler JW. Ligament reconstruction for the painful thumb carpometacarpal joint. J Bone Joint Surg Am. 1973;55:1655–66.
- 6. Burton RI. Basal joint arthrosis of the thumb. Orthop Clin North Am. 1973;4:347–8.
- Fontana L, Neel S, Claise JM, Ughetto S, Catilina P. Osteoarthritis of the thumb carpometacarpal joint in women and occupational risk factors: a case-control study. J Hand Surg Am. 2007;32:459–65.
- Barron OA, Glickel SZ, Eaton RG. Basal joint arthritis of the thumb. J Am Acad Orthop Surg. 2000;8: 314–23.
- Bettinger PC, Linscheid RL, Berger RA, Cooney 3rd WP, An KN. An anatomic study of the stabilizing ligaments of the trapezium and trapeziometacarpal joint. J Hand Surg Am. 1999;24:786–98.
- Wilgis EF. Distal interphalangeal joint silicone interpositional arthroplasty of the hand. Clin Orthop Relat Res. 1997;(342):38–41.
- Cobb TK. Arthroscopic distal interphalangeal joint arthrodesis. Tech Hand Up Extrem Surg. 2008;12:266–9.
- Sieber HP, Segmuller G. Arthrodesis of the interphalangeal joint of the thumb: indication, technic, results. Handchir Mikrochir Plast Chir. 1983;15:11–6.

- Stern PJ, Fulton DB. Distal interphalangeal joint arthrodesis: an analysis of complications. J Hand Surg Am. 1992;17:1139–45.
- Brannon EW, Klein G. Experiences with a finger-joint prosthesis. J Bone Joint Surg Am. 1959;41-A: 87–102.
- Swanson AB, Herndon JH. Flexible (silicone) implant arthroplasty of the metacarpophalangeal joint of the thumb. J Bone Joint Surg Am. 1977;59:362–8.
- Figgie MP, Inglis AE, Sobel M, Bohn WW, Fisher DA. Metacarpal-phalangeal joint arthroplasty of the rheumatoid thumb. J Hand Surg Am. 1990;15:210–6.
- Beckenbaugh RBSA. Total joint arthroplasty for the metacarpophalangeal joint of the thumb -a preliminary report. J Bone Joint Surg Am. 1981;4:295–7.
- Inglis AE, Hamlin C, Sengelmann RP, Straub LR. Reconstruction of the metacarpophalangeal joint of the thumb in rheumatoid arthritis. J Bone Joint Surg Am. 1972;54:704–12.
- Stanley JK, Smith EJ, Muirhead AG. Arthrodesis of the metacarpo-phalangeal joint of the thumb: a review of 42 cases. J Hand Surg Br. 1989;14:291–3.
- Schmidt CC, Zimmer SM, Boles SD. Arthrodesis of the thumb metacarpophalangeal joint using a cannulated screw and threaded washer. J Hand Surg Am. 2004;29:1044–50.
- Bicknell RT, MacDermid J, Roth JH. Assessment of thumb metacarpophalangeal joint arthrodesis using a single longitudinal K-wire. J Hand Surg Am. 2007;32: 677–84.
- Eaton RG, Lane LB, Littler JW, Keyser JJ. Ligament reconstruction for the painful thumb carpometacarpal joint: a long-term assessment. J Hand Surg Am. 1984; 9:692–9.
- Lane LB, Henley DH. Ligament reconstruction of the painful, unstable, nonarthritic thumb carpometacarpal joint. J Hand Surg Am. 2001;26:686–91.
- Pellegrini Jr VD, Parentis M, Judkins A, Olmstead J, Olcott C. Extension metacarpal osteotomy in the treatment of trapeziometacarpal osteoarthritis: a biomechanical study. J Hand Surg Am. 1996;21:16–23.
- Gwynne-Jones DP, Penny ID, Sewell SA, Hughes TH. Basal thumb metacarpal osteotomy for trapeziometacarpal osteoarthritis. J Orthop Surg (Hong Kong). 2006;14:58–63.
- Burton RI, Pellegrini Jr VD. Surgical management of basal joint arthritis of the thumb. Part II. Ligament reconstruction with tendon interposition arthroplasty. J Hand Surg Am. 1986;11:324–32.
- Barron OA, Eaton RG. Save the trapezium: double interposition arthroplasty for the treatment of stage IV disease of the basal joint. J Hand Surg Am. 1998;23: 196–204.
- Froimson AI. Tendon arthroplasty of the trapeziometacarpal joint. Clin Orthop Relat Res. 1970;70: 191–9.
- Eaton RG, Glickel SZ, Littler JW. Tendon interposition arthroplasty for degenerative arthritis of the trapeziometacarpal joint of the thumb. J Hand Surg Am. 1985;10:645–54.

- Gervis WH. Excision of the trapezium for osteoarthritis of the trapezio-metacarpal joint. J Bone Joint Surg Br. 1949;31B:537–9, illust.
- Davis TR, Brady O, Dias JJ. Excision of the trapezium for osteoarthritis of the trapeziometacarpal joint: a study of the benefit of ligament reconstruction or tendon interposition. J Hand Surg Am. 2004;29:1069–77.
- Wajon A, Ada L, Edmunds I. Surgery for thumb (trapeziometacarpal joint) osteoarthritis. Cochrane Database Syst Rev. 2005;(4):CD004631.
- Eaton RG, Floyd 3rd WE. Thumb metacarpophalangeal capsulodesis: an adjunct procedure to basal joint arthroplasty for collapse deformity of the first ray. J Hand Surg Am. 1988;13:449–53.
- Rizzo M, Moran SL, Shin AY. Long-term outcomes of trapeziometacarpal arthrodesis in the management of trapeziometacarpal arthritis. J Hand Surg Am. 2009; 34:20–6.
- Taylor EJ, Desari K, D'Arcy JC, Bonnici AV. A comparison of fusion, trapeziectomy and silastic replacement for the treatment of osteoarthritis of the trapeziometacarpal joint. J Hand Surg Br. 2005;30: 45–9.
- 36. Raven EE, Kerkhoffs GM, Rutten S, Marsman AJ, Marti RK, Albers GH. Long term results of surgical intervention for osteoarthritis of the trapeziometacarpal joint: comparison of resection arthroplasty, trapeziectomy with tendon interposition and trapeziometacarpal arthrodesis. Int Orthop. 2007;31: 547–54.
- Menon J. Arthroscopic management of trapeziometacarpal joint arthritis of the thumb. Arthroscopy. 1996;12:581–7.
- Menon J. Arthroscopic evaluation of the first carpometacarpal joint. J Hand Surg Am. 1998;23:757.
- Badia A. Trapeziometacarpal arthroscopy: a classification and treatment algorithm. Hand Clin. 2006;22:153–63.
- Slutsky DJ. The use of a dorsal-distal portal in trapeziometacarpal arthroscopy. Arthroscopy. 2007;23:1244 e1241–1244.
- Culp RW, Rekant MS. The role of arthroscopy in evaluating and treating trapeziometacarpal disease. Hand Clin. 2001;17:315–9, x–xi.
- Hofmeister EP, Leak RS, Culp RW, Osterman AL. Arthroscopic hemitrapeziectomy for first carpometacarpal arthritis: results at 7-year follow-up. Hand (N Y). 2009;4:24–8.
- 43. Edwards SG, Ramsey PN. Prospective outcomes of stage III thumb carpometacarpal arthritis treated with arthroscopic hemitrapeziectomy and thermal capsular modification without interposition. J Hand Surg Am. 2010;35:566–71.
- 44. Pegoli L, Parolo C, Ogawa T, Toh S, Pajardi G. Arthroscopic evaluation and treatment by tendon interpositional arthroplasty of first carpometacarpal joint arthritis. Hand Surg. 2007;12:35–9.
- Adams JE, Merten SM, Steinmann SP. Arthroscopic interposition arthroplasty of the first carpometacarpal joint. J Hand Surg Eur Vol. 2007;32:268–74.

Osteoarthritis of the Fingers

6

Daniel Herren

Keywords

Arthroplasty • Arthrodesis • Osteoarthritis • PIP joint • DIP joint • Conservative treatment

Introduction

Osteoarthritis is the most common form of joint disease and constitutes a significant economic burden for western healthcare systems. Although treatment modalities have evolved, only a few studies offer evidence-based data on the effectiveness and long-term results of the different treatment options. Of all the different forms of osteoarthritis, the degenerative or idiopathic type is the most common and this has a high prevalence of finger joint involvement. Some studies suggest that the finger joints are the most common site of osteoarthritis in the entire musculoskeletal system [1].

The degree of functional impairment in OA of the fingers depends on which joints are affected, the degree of limitation of active motion, and the sector in which the deficit lies. If the MCP joints (which are rarely affected in patients with OA) are intact, an extension deficit of the PIP joint is functionally better tolerated than a lack of flexion.

Zurich, Switzerland e-mail: daniel.herren@kws.ch Stability of the interphalangeal joints is an important issue, especially in the radial digits, since it is needed for a strong pinch with the thumb. Patients with an erosive and inflammatory type of OA in these joints may have significant instability and deformity, which must be addressed when evaluating surgical treatment options. The deformity may not only be a functional problem but also an aesthetic one, especially in the DIP joints, where marked osteophytes may be observed.

The increasing number of patients affected by this disease, together with the increasing therapeutic possibilities, make this probably the fastest growing patient population in hand surgery.

Background/Aetiology

Degeneration as a process of aging has long been the simple explanation of osteoarthritic diseases in different joints. Newer studies provide increasing evidence that an individual's genetic background has an important role in the development of OA, and new genes that are important in the pathophysiology of joint destruction have been detected [2]. Goekoop et al. [3] found that the absence of OA in 90-yearolds was associated with male sex, a normal BMI,

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absence of familial predisposition and (surprisingly) heavy physical work. These findings were irrespective of the site of the OA. Dietary influences on the development of OA are strongly disputed. Data from Williams et al. [4] suggest that a diet rich in fruit and vegetables has a beneficial effect on the development of OA.

It has been shown that the cumulative incidence of finger joint osteoarthritis is generally higher in women but the distribution over the different finger joints is the same in both sexes. The distal interphalangeal joint (DIP) is most frequently involved, followed by the thumb saddle joint (CMC I) and the proximal interphalangeal joint (PIP), while the metacarpophalangeal joint (MCP) is rarely affected. Handedness seems to play no part in the development of OA in the finger joints [1].

If the MCP joint shows clinical symptoms and corresponding changes, especially in the second and third fingers, this may signal underlying disease. Both hemochromatosis and chondrocalcinosis have to be actively ruled out [5]. Hemochromatosis typically shows similar degenerative changes to primary OA, with subchondral cyst formation, sclerosis, and thinning of the cartilage. On the other hand, chondrocalcinosis, involving both fibrous and hyaline cartilage, often affects the scaphotrapeziotrapezoid (STT) and the CMC I joints and shows calcifications in the triangular fibrocartilage complex (TFCC) [6].

Overall, the prevalence of OA in the fingers is two to four times higher in women than in men [7]. The duration of the woman's fertile period as well as the age at menopause showed a positive relationship to DIP joint OA, suggesting a strong hormonal dependence [8].

Pearls: Aetiology

- Genetic predisposition seems to be a major factor in disease development.
- The prevalence of OA in the fingers is two to four times higher in women than in men, and late menopause is a negative predictive factor.
- The DIP joint is the most frequently affected joint in primary osteoarthritis of the hand, followed by the thumb saddle joint and the PIP joint. MCP joints are rarely affected by this disease.



Fig. 6.1 Typical pattern of a osteoarthritis of the small finger joints combined with a peritrapezoidal ostearthritis

Presentation, Investigation and Treatment Options

The presentation of OA in the fingers is quite uniform; the diagnosis is based mainly on the clinical picture and confirmed by conventional radiographic examination (Fig. 6.1). The American College of Rheumatology defined the following criteria for the classification of OA of the hand in comparison with rheumatoid arthritis and other inflammatory joint diseases: hard tissue enlargement involving at least two of the ten selected joints (second and third DIP and PIP joints, and CMC I on both hands); swelling of fewer than three MCP joints; and hard tissue enlargement of at least two DIP joints. This classification method has a sensitivity of 92 % and specificity of 98 % [9].

Patients classically present with swollen, tender DIP or PIP joints. Mucoid cysts, with or without nail deformity, are often seen at the level of the DIP joint. The PIP joint has a more diffuse, swollen appearance with a fusiform joint contour. Joint stiffness is almost always present and often correlates with the degree of swelling. Most authors still use the Kellgren and Lawrence scale [10] for the radiographic classification:

- Grade 1: doubtful narrowing of joint space and possible osteophytic lipping
- Grade 2: definite osteophytes, definite narrowing of joint space
- Grade 3: moderate multiple osteophytes, definite narrowing of joint space, some sclerosis and possible deformation of bone contour
- Grade 4: large osteophytes, marked narrowing of joint space, severe sclerosis and definite deformation of bone contour

The initial phase of disease sees the onset of an inflammatory process that comes to a halt at a later stage [11]. This explains the fact that many patients have fewer symptoms in the end stage of the disease than at the beginning. The DIP joint, in particular, can become asymptomatic with time and not need any further treatment. The PIP joint often has residual limited but painful motion.

Ultrasound examination findings do not correlate with the clinical disease or the severity of damage seen on X-ray [12].

MRI studies of finger OA reveal that primary osteoarthritis shows more erosive joint changes than previously thought or indicated on conventional radiographs [13]. However, MRI does not belong in the routine investigation of OA of the fingers and no validated scoring system is yet available [14].

Scintigraphy is used as a screening tool only in unclear cases with polyarticular symptoms and no conventional radiographic changes in the joints.

CT scans are rarely, if ever, indicated in OA to examine symptomatic joints in the fingers.

Pearls: Investigations

- The diagnosis of osteoarthritis in the fingers is based on the symptoms and the clinical picture, confirmed by conventional radiographs. Further investigations are rarely needed.
- The initial phase of the disease often shows an inflammatory process, which diminishes with time.
- DIP joints may become asymptomatic as the disease progresses, despite marked destruction and deformity.

Treatment Options: Conservative Treatment

Osteoarthritis is an incurable disease and all attempts to treat this condition do no more than modify the symptoms or repair the damage. There is little evidence that any sort of prevention might be effective in stopping unaffected joints becoming part of the disease process. In the pathophysiology of the disease, catabolic cytokines and anabolic growth factors play key roles in the destruction of the cartilage. TNFalpha-blocking agents, used mainly in patients suffering from rheumatoid arthritis, are good candidates for suppressing the destructive inflammatory process in OA as well. Beside the classic systemic application of this drug, an intra-articular treatment with injection showed in a pilot study a good symptomatic effect with a possible disease modifying action of intraarticular Infliximab in erosive osteoarthritis of the hands [15].

Conventional treatment includes analgesics and non-steroidal anti-inflammatory drugs. Intra-articular viscosupplementation with hyaluronic acid has been shown to be effective in terms of pain relief and improved disability. In comparison with intra-articular corticosteroids, it seems to have a longer benefit [16], especially in the knee joint. However, personal experience does not support this observation for the finger joints.

Glucosamine and chondroitin are important components of the normal cartilage. Like viscosupplementation, the efficiency of glucosamine and chondroitin in the treatment of OA has been documented best in the knee joint [17]. They seem to reduce the need for anti-inflammatory drugs and improve functionality [18]. Since these substances are of natural origin (fish and other animal cartilage) few side effects have been reported. Most authors recommend a combination of the two, at a dosage of 1,500 mg glucosamine and 1,200 mg chondroitin daily. Since the onset of the effects is slow and takes at least 4 weeks, most authors recommend either 3 months' therapy twice a year or continuous treatment [18].

In the fingers, the PIP joint reacts well to intra-articular corticosteroid injections, while injection of the DIP joint is often painful and has limited effect. The side effect that is most common and difficult to control is atrophy of the skin and subcutaneous tissue, which is more of an aesthetic than a functional problem. There seems to be no correlation between the radiographic appearance of the joint and the effectiveness of intra-articular steroid administration. The infiltration seems to lose its efficiency with time and, together with the side effects, this therapy is selflimiting. There are different techniques for PIP infiltration: we prefer to inject into the dorsal recess of the joint.

Splints for painful inflamed joints might be effective but their regular use limits the functionality of the hand and patient satisfaction is low [19]. Modification of activity may be beneficial in preventing articular inflammation. Joint protection devices may relieve the joints and help to prevent further irritation of the joints affected.

The effects of ultrasound, laser and electrotherapy in the treatment of OA in the fingers are not well documented. Experience has shown limited and short-term effects with an often inappropriate cost-efficiency ratio.

Pearls and Personal Recommendation: Conservative Treatment

- OA of the fingers is an incurable disease and all attempts to treat the condition are limited to modifying the symptoms and, at best, to slowing down its progression
- Anti-inflammatory therapy is the mainstay of conservative treatment in OA. Intraarticular corticosteroid infiltration is the most efficient, especially in the PIP joint. Possible side effects should be discussed with the patient.
- Glucosamine and chondroitin may act as anti-inflammatory agents with a certain chondroprotective potential. Three months' trial therapy with 1,500 mg glucosamine and 1,200 mg chondroitin daily is recommended.

Treatment Options: Surgical Treatment

Surgical treatment options include synovectomy, joint replacement, and joint fusion. There is no literature on synovectomy of the PIP joint for patients suffering from OA. Synovectomy may be considered in the early stages of the osteoarthritic process when there is marked inflammation and the cartilage is still preserved. There is speculation that the physical removal of the synovial mass, together with a denervation effect, might relieve the symptoms. Since no data on the mid- and long-term effects of that procedure are available, we can only report our personal experience of this intervention. Overall, the results of this procedure are mixed at best. Persistent, if not even exacerbated, pain and postoperative joint stiffness are possible complications. We find that the best candidates for this procedure are patients who had a good response to intra-articular steroid injections and have more than 80 % cartilage preserved in the affected joints. But even in this selected patient group, there is only a 50-50 chance of a good result.

In the DIP joints, mucoid cysts are quite often the first sign of a degenerative process. The typical clinical presentation is a swelling distal to the DIP joint, which may involve the subcutaneous tissue and even the skin (Fig. 6.2). Nail deformities are often seen if the cyst presses on the germinal nail matrix. Treatment options include aspiration, injection with corticosteroid, and cyst excision with or without the skin involved. If joint destruction is already advanced, a definitive surgical solution such as joint fusion is indicated.

Aspiration alone has an extremely high recurrence rate of more than 90 % but its



Fig. 6.2 Mucoidcyst of the DIP-joint with pressure on the germinative nail matrix and subsequent nail deformity

combination with an infiltration of locally acting corticosteroid lowers the recurrence rate to 40-50 % [20]. Surgical removal of the cyst carries by far the lowest risk of recurrence – reducing it to less than 5 %. Whether the overlying skin has to be removed and a rotational flap applied is a matter of debate. If the skin is extremely thin or spontaneous drainage has been observed, excision of the involved skin makes sense. Very occasionally, the skin defect is of a size to require soft tissue coverage other than a local rotational flap. The preoperative nail deformity, mainly nail ridging, often resolves within one or two generations of nail after the cyst has been removed.

If finger joint destruction is advanced, a definitive treatment solution has to be found. Joint replacement is the only functionally acceptable option for the MCP joint, although this is rarely affected by primary OA. The rules for joint replacement discussed in the chapter on rheumatoid arthritis should be followed.

The ideal goal for reconstruction of a disabled PIP joint is a pain-free restoration with functional mobility and adequate stability. The index and middle fingers are the pinching partners of the thumb, while the ulnar fingers need mobility in order to grasp larger objects. When considering joint replacement, the degree of instability and deformity has to be taken in account. Experience shows that pre-existing deformity and instability in the PIP joint is difficult to correct, even with formal collateral ligament reconstruction and prolonged splinting during rehabilitation (Fig. 6.3). Arthrodesis should therefore be considered carefully, especially in the radial digits, if the lateral deformation of the PIP joint exceeds 30°. PIP joint fusion in a functionally good position provides adequate function, although fine motor skills in particular are affected. Woodworth et al. [21] evaluated the impact of simulated PIP joint fusion on all four fingers with the PIP joint fixed in 40° of flexion. Low-demand activities of daily living suffered significantly when compared with unrestricted motion in all finger joints, while precision handling was perceived to be more difficult and required more compensation by the MP joints.

Simultaneous fusions of the PIP and DIP joints in the same finger ray are possible, although precision handling will suffer. The combination of PIP arthroplasty and DIP fusion is functionally much better tolerated even if the range of motion in the PIP joint is limited.

PIP joint replacement is a widely accepted procedure in joints with OA destruction. The choice of implant and the approach used are the two most frequently discussed issues. A variety of implants is available, but only a few series with adequate long-term follow-up have been published. Silicone implants, introduced by Swanson in the late 1960s, are still the gold standard for newer generations of implants with respect to functional performance, revision rate, and long-term outcome. Silicone joint spacers carry a risk of implant breakage and silicone synovitis. Newer implant designs of the resurfacing type may, however, show dislocation and implant loosening. Overall, the silicone spacer produces fairly consistent results with good pain relief and reasonable function, with a range of motion between 40° and 60° active flexion/extension. Only a few cases with relevant silicone synovitis have been reported and, although implant failure is seen, it does not necessarily lead to revision [22-25]. There is a newer generation of silicone implants such as the NewFlex[™] and the Sutter prosthesis. These devices have a more rectangular shape, which should provide better stability of the joint. Since the anatomical shape of the subcapital bone of the proximal phalanx is more elliptic, the rectangular shape of these hinges may interfere to some extent with the extensor mechanism, so it is essential that the implant is placed correctly. No randomised controlled trials with series of different silicone implants in the PIP joint are available, and analysis of the different case series suggests similar results for most of the Silicone implant designs.

The newest generation of PIP joint prostheses follows the principles of surface replacement with a two-component concept. The proximal component replaces the bicondylar head of the proximal phalanx and the distal component has some sort of a cup, which articulates with

Fig. 6.3 Recurrence of a pre-existing deformity of a PIP joint suffering form OA and subsequent treatment with a silicone PIP arthroplasty. (a) Preoperative status with the ulnar deviation (b) Postoperative appearance after 6 weeks with good alignment (c) Recurrence of the deformity at the PIP joint 12 months after the intervention



the head. Taking a closer look at the implants, they do not represent a real resurfacing concept, since a significant amount of bone has to be resected and long stems for both components are needed to provide adequate fixation. Several material combinations are available, from the classic chrome cobalt/polyethylene to ceramic/ ceramic and pyrocarbon/pyrocarbon. Most of these implants can be used without cement, although some of them require cementing for primary fixation in the bone. The majority of surgeons prefer non-cemented implants, since revision is easier and removal of the implant causes less damage and bone loss. Overall, the newer generation of PIP implants based on the resurfacing concept seemed a logical development in PIP arthroplasty, but most of them have not yet stood the test of time and real-life longterm follow-up series are still lacking for most implant designs.

The concept of resection-interposition arthroplasty, with a volar plate for example, is reported only for traumatic or post-traumatic conditions. No formal series has been published for joints with osteoarthritic destruction.

For the vast majority of hand surgeons, arthrodesis is still the standard procedure for treating a deformed and painful DIP joint. It gives consistent, sustained results and allows the correction of pre-existing deformity. This treatment concept is the result of the observation that DIP joint fusion can occur spontaneously in patients suffering from OA and is functionally well tolerated. Most patients experience slow degradation of DIP joint motion in the course of the disease and adapt to that functional impairment quite well. Although DIP joint movements become more important when there is also limited PIP joint mobility, fine motor skills in particular suffer from additional DIP joint restriction. The option of DIP arthroplasty has been advocated for these cases. Although it is logical as an alternative to joint fusion, DIP arthroplasty has so far made no real breakthrough in most surgeons' treatment concepts. The few publications suggest this procedure as a good alternative to DIP fusion [26, 27].

Pearls and Personal Recommendation: Surgical Treatment Options

- Removal of mucoid cysts in the DIP joint is most successful when there is only mild destruction of the joint. Mucoid cysts in combination with advanced destruction of the joints are best treated with DIP fusion.
- PIP synovectomy in OA of the fingers produces mixed results and the indication must be considered carefully.
- The standard treatment for painful destruction of the PIP joints is arthroplasty, or arthrodesis in selected cases, and joint fusion in the DIP joint.

Surgical Techniques and Rehabilitation

PIP and DIP arthroplasty and arthrodesis are the most common surgical procedures in patients with joint destruction from primary OA in the fingers. The following section focuses on these treatment options.

PIP Joint

PIP arthroplasty has a shorter history of use than MCP joint replacement. For decades, joint fusion was the standard procedure for painful PIP joint destruction and the functional results of this procedure were generally reported to be good [28]. Pellegrini and Burton [29] reviewed a number of patients who had undergone different procedures for PIP joint destruction. They observed that arthrodesis in the radial digits brought an improvement in the lateral pinch, while arthroplasty in the ulnar digits gave reasonable functional mobility with good pain relief. Based on this analysis, the authors were not able to make a definitive recommendation on the optimal procedure for destroyed PIP joints. Since that publication, however, several authors have advocated the concept of reserving PIP arthroplasty for ulnar

digits and treating the index finger, which is the main partner for pinching with the thumb, with PIP joint fusion. We have adapted the concept in that PIP arthroplasty is indicated in all digits, but the rehabilitation programme in the index finger is modified. Functional exercises with the index finger are begun later and functional splinting is prolonged in order to protect the radial collateral ligament, which is most important for the lateral stability of this joint. The goal of index finger rehabilitation after PIP arthroplasty is not maximum mobility but an optimal balance between mobility and stability.

Contraindications to PIP joint replacement include the classic criteria of insufficient bone stock, missing or dysfunctional tendons, and severe tendon imbalance, especially contracted boutonnière and swan-neck deformities. In severely contracted joints with a long-standing history of immobility, PIP joint fusion in a functional position may be a better choice than implant arthroplasty. Severe joint instability and deformity of more than 30° is extremely difficult to correct with an implant and is a relative contraindication to arthroplasty.

The choice of implant depends on several factors, including the surgeon's experience, the local anatomical situation, especially the bone stock, and the surgical approach. Silicone devices, which act as joint spacers, are by far the most forgiving implants. They provide reproducible results even in cases with difficult bone stock and with limited surgical experience. They can be implanted easily using different surgical approaches. More complex, two-component joints need an adequate bone stock; no large cystic defects can be allowed to exist with implants, as they have to be inserted without cementing. Correct placement, with the goal of restoring the biomechanical centre of rotation, needs some experience. Some of these implants are supplied with resection guides, which can be used only with a dorsal approach. In addition, some prostheses need more space for implantation, which also means that a dorsal or lateral approach is required.

Different surgical approaches have been described to implant a PIP joint replacement. All of them have theoretical advantages and disadvantages. So far, no one approach has proved to be superior to the others, although the theoretical advantages of the volar approach are now being discussed. The dorsal approach is the most widely used and technically least demanding in comparison with the volar and lateral approaches. It is also required when certain soft tissue conditions, such as mild swan-neck or boutonnière deformity, are to be corrected at the same time. A straight or slightly curved longitudinal incision is performed. The dorsal veins should be preserved if possible and care taken with the dorsal nerve branch to the PIP joint. Several techniques have been described to access the joint. Swanson [25] advocated a midline split of the central slip of the extensor tendon. Care should be taken to preserve the insertion of the central slip at the base of the middle phalanx, since a good view of the joint is usually only possible if some of the insertion is released on the ulnar and radial sides. Transosseous reinsertion of the central slip may therefore be necessary after the implantation. A good alternative, and our choice, is the approach described by Chamay [30]. He uses a V-shaped extensor flap, which offers a good view of the joint and allows a long stable suture line for tendon closure. After exposing the joint, the most dorsal parts of the two collateral ligaments are released; this gives full access to the joint with a perfect overview. Dorsal and even volar osteophytes can now be removed. Care has to be taken not to violate the insertion of the central slip. The bone is prepared according to the needs of the selected implant. For silicone implants, the resection line is planed according to the implant size (most often size 1 in the original Swanson design) and care should be taken to preserve as much of the collateral ligaments as possible. After bone preparation, the trial implant is inserted and a trial reduction is performed. The tension should be chosen so that full flexion and, in particular, extension is possible. Either a smaller implant or more bone resection is needed if there is an extension lag. When there is significant joint deformity or deficient collateral ligaments, reinforcement suture of the ligaments and/ or a staged release is needed on the contracted side. The sutures are passed within the ligament and reinserted through drill holes in the proximal phalanx. The joint should now be well balanced but with a full passive range of motion still being possible. It is virtually impossible to correct any deformity remaining on the operating table, even with a well-applied rehabilitation programme.

After skin closure, a standard hand dressing is applied, including a volar splint.

Rehabilitation must be individualized according to the intra-operative stability, the collateral ligament status, and the finger ray. A more conservative rehabilitation programme is not started until 2-3 weeks after surgery for the index finger and for any joints that were severely deformed and consequently required collateral ligament re-balancing. Theoretically, the long suture line in the extensor tendon allows early active mobilization. Resting splints in the intrinsic plus position are worn for up to 6 weeks. Buddy splinting to the neighbouring radial finger, with a figure of eight dressing, is a good way of protecting the collateral ligaments and yet still allowing an active and passive range of motion. Individual adaptations need to be made during the rehabilitation programme. If the joints become stiff early, more vigorous mobilisation is needed. In general, dynamic splinting is rarely needed and not tolerated by the soft tissues until 4-6 weeks after surgery. In our experience, an extensor lag is the most commonly observed deficit following the dorsal approach. The reasons could be scarring of the extensor tendon with subsequent loss of free gliding or a certain excess length of the extensor mechanism. Night splints in extension and dynamic extensor splints may help. In cases of a mild, passively correctable swan-neck or boutonnière deformity in combination with destruction of the PIP joint, a dorsal approach is essential for joint replacement. Careful attention should be paid to the cause of the swan-neck deformity, as this is very often found at a different level from the PIP joint. These cases require release of the lateral bands, often in combination with lengthening of the central slip. A central slip reconstruction or reinforcement is needed with boutonnière deformity. Several techniques have been described for this difficult procedure. Overall, PIP arthroplasty has limited results in the presence of these deformities and there is an inherent danger that the joint will become stiff or that the deformity will recur.

The **volar approach** has, at least theoretically, several advantages over the other approaches. The

tendons are not violated with this technique and, in particular, the delicate extensor mechanism remains untouched. The venous drainage is less compromised, which results in less postoperative swelling and easier subsequent rehabilitation. However, the volar approach is technically more demanding and offers less space for the implantation of an artificial joint. In addition, pre-existing tendon imbalances are more difficult to correct. The technique described by Simmen offers good access to the joint [22]. A Bruner incision forms a radially based skin flap. The two neurovascular bundles are identified and protected. The ulnar bundle has to be mobilised, while the radial bundle remains with the skin flap. The flexor tendon sheet is opened transversely in the area of the A3-pulley on both the volar and the dorsal side. On the ulnar and the radial sides, the incision is continued to form a sleeve, which includes the release of the accessory collateral ligaments (Fig. 6.4). Access to the joint is now achieved with hyperextension. Some release of the ulnar collateral ligament may be needed if the joint is not supple enough to get a good exposure. The osteophytes, especially those on the volar side, can now be removed. This is important since they may be a potential site of impingement with the implant in flexion. The head of the proximal phalanx can now be resected but care has to be taken to identify the ulnar neurovascular bundle and protect it with retractors. Preparation of the bone and implantation of the prosthesis follow the same principles as for the dorsal approach. For closure, the pulley sleeve is retracted and reattached in its anatomical position. In cases with pre-existing deviation of the flexor tendon due to lateral deformity, the tendon can be recentred. If need be, the collateral ligaments can be reinforced with sutures. It is important to test the passive range of motion again before final closure. The rehabilitation programme follows the principles outlined for the dorsal approach but no special protection of the extensor tendons is needed, and even passive motion is allowed.

The **lateral approach** is the least common approach used for PIP implants. The incision goes along the midline on the ulnar side of the finger and curves dorsally on the middle phalanx. After releasing the oblique and transverse fibres

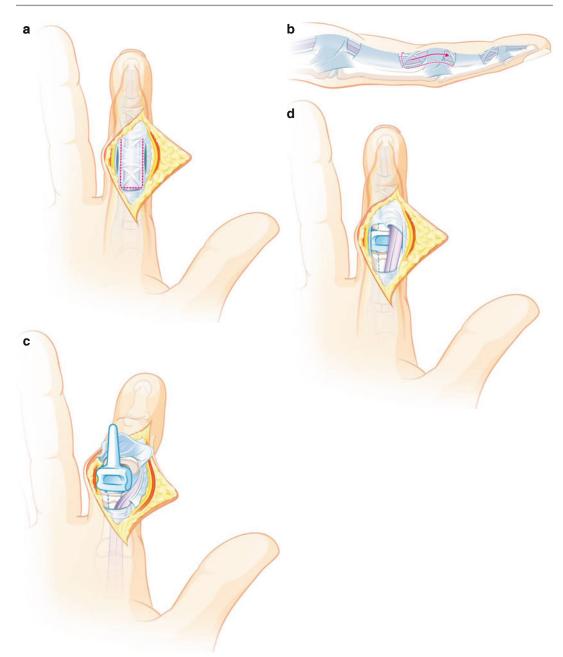


Fig. 6.4 Volar approach for PIP arthroplasty. (**a**, **b**) A sleeve of the flexor pulley system is formed starting at A3 pulley, including the release of the accessory collateral ligaments. (**c**) Reflexion of the flexor tendons and after the

of the retinacular ligaments, the extensor apparatus is elevated and can be mobilized laterally, with the insertion of the central slip remaining intact. The ulnar neurovascular bundle remains on the volar side of the joint. The ulnar collateral preparation of the bone the implant can be inserted (**d**) Final appearance after joint reposition. The flexor pulley sleeve can be re-fixed and immediate active and passive rehabilitation is possible

ligament has to be detached completely in such a way that the joint can be opened on the radial side. This is best done with a triangular proximally based flap that can be reflected proximally. The implant can be inserted as described previously. For closure, it is essential to reattach the ulnar collateral ligament in such a way that active rehabilitation is possible. The ulnar side has to be protected with buddy splinting for up to 6 weeks.

Arthrodesis of the joint may be indicated in cases of severe instability and deformity of the PIP joint or difficult bone situations. Several techniques have been described for this procedure. Tension band wiring, plate fixation, and screw arthrodesis are the most common techniques. Tension band wiring has the advantage that compression of the arthrodesis site occurs during active motion. This technique is also costeffective, using inexpensive hardware. The disadvantages are possible pin protrusion and painful hardware requiring subsequent metal removal [28]. Plate fixation, usually 2.0–2.4 mm in size, allows rigid fixation at the desired angle. It has the disadvantage of causing extensor tendon adhesions along the plate, thus limiting DIP motion. The newer-generation plates are so thin that hardware removal is not necessary in most patients. The screw fixation technique, preferably with a headless screw, is another option. Theoretically, a single screw has no rotational stability but in practical use this does not cause any problems [31]. The main challenge with the screw technique is to achieve the desired fusion angle. This is not so easy to accomplish, especially for angles less than 30°. The straighter the fusion position, the more difficult it gets to obtain adequate purchase on the distal volar fragment. The screw also has more potential for protrusion on the proximal dorsal cortex.

The joint is approached from the dorsal aspect. The central slip of the extensor tendon is split and the joint opened. After removing the osteophytes and releasing both collateral ligaments, there is a good view of the joint. The osteotomy should be performed in such a way that the desired fusion angle is set on the proximal part and a perpendicular bone cut is made on the distal part. Suitable fusion positions are usually $15-20^{\circ}$ of flexion angle in the radial digits and $25-40^{\circ}$ in the ulnar joints. Trial reposition is performed and can be held in place with a temporary K-wire. Fluoroscopic control should confirm good bone contact over the whole area of the osteotomy.

Once this preliminary fixation has been done, the hand is removed from the operating table to check the three-dimensional appearance of the finger. Rotational malpositioning, in particular, has to be avoided. Definitive fixation is then performed. The extensor tendon is sutured over the hardware to cover it, and the remaining DIP motion is checked.

Postoperatively, the PIP joint should be protected in a finger splint for 6 weeks. Early mobilisation of the DIP joint out of the protective splint is started after a few days. Bone healing should be confirmed with radiographs 6 weeks after surgery.

DIP Joint

The standard procedure for treating painful destroyed DIP joints is joint fusion. In OA, the DIP often presents with a marked deformity, significant osteophytes, and an active range of motion that is already limited. With DIP fusion, the joint can be brought into a functionally good position and a definitive solution can be offered with a single procedure. The cosmetic appearance is often much better once the deformity has been corrected, something that is important to the mainly female population with this disease. There are several techniques described for DIP arthrodesis. Screw fixation, K-wires, and tension band wiring are the most commonly used. Newer implants such as endomedullary staples, providing purely intramedullary fixation, are also available and have the advantages of not requiring any secondary hardware removal and not causing any irritation of the fingertip. The screw technique has the advantage of immediate strong fixation with a low complication rate [32]. K-wires may be needed in difficult bone situations where screw fixation is not possible or in cases with a significant mismatch between the size of the screw and the dimensions of the phalanx. Screw fixation with a 2.0 mm screw is possible in almost all cases of osteoarthritic destruction of the DIP joint, although sometimes a 1.5 mm screw is necessary for the small finger. Although several authors prefer a headless screw, it is our experience that a regular 2.0 mm AO screw can be placed in such a way that the head does not irritate

the fingertip. The length of the screw is somewhat critical, especially in patients suffering from concomitant destruction of the PIP joint, which might require subsequent joint arthroplasty. It is usually possible to get enough purchase with a screw length of about 30 mm, since the shape of the medullary canal of the middle phalanx is at its narrowest about 15-20 mm proximal to the joint line. In most cases, it is still possible to do a PIP arthroplasty with a screw of this length, either at the same time or later without removing the screw. K-wire fixation is often less convenient for the patient, since additional immobilization of adjacent joints is necessary due to the limited primary stability of the fixation. There is a higher risk of infection and secondary wire removal is required. If K-wires are needed, it is better to avoid penetration of the wires through the fingertip. The wires can be inserted from proximaldorsal to distal-volar. Two wires should be used in a crossed fashion in order to enhance stability and provide rotational fixation.

Surgically, the DIP joint is approached through a dorsal transverse incision; this usually gives adequate exposure of the joint. Proximal or distal extension is always possible if more space is needed. The extensor tendon is identified and a transversal tenotomy is performed. In most patients there are significant osteophytes proximal and distal to the joint, which have to be removed, especially for cosmetic reasons. Care should be taken distally to the germinative nail matrix, which starts a few millimetres distal to the joint line. Violation of this structure can lead to severe permanent nail deformity. Both parts of the joint are now prepared for fusion. A power-saw is used for the proximal part but the bone resection should be kept as minimal as possible, in order to avoid excessive shortening of the finger. The distal part can be prepared with the rongeur. The goal is to achieve joint fusion in 5-10° of flexion. This modest amount of flexion gives a functionally good position with an optimal cosmetic appearance. For screw insertion, the inside out technique on both parts is the easiest way to place the screw correctly. The insertion point is in the central part of the osteotomy surface on both sides. The screw channel should point to the dorsal recess of the

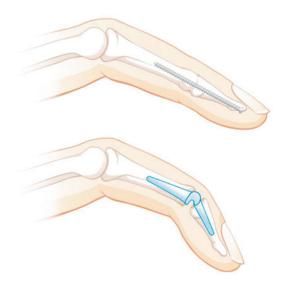


Fig. 6.5 Schematic drawing of a DIP fusion with a screw and a DIP arthroplasty with an original Swanson silicone implant

PIP joint in the middle phalanx, while a straight subungual direction is optimal in the distal phalanx. These positions achieve the desired slight flexion. The distal phalanx is over-drilled in the usual lag screw technique and the screw should be placed flush to the surface of the subungual bone. Correct screw placement and good contact of the osteotomy site have to be checked by fluoroscopy during the operation. It is important to lift the hand from the operating table and check the position of the fused joint relative to the other fingers. Care should be taken to ensure that the rotation of the finger is correct. Tightening the screw might rotate the finger to the ulnar side. It is not mandatory to re-suture the extensor tendon but, if this is done, only a few stitches of fine absorbable suture material should be used. The joint is protected for 6 weeks with a removable DIP splint. Bone healing is then confirmed radiographically.

DIP arthroplasty is another surgical option in this patient group (Figs. 6.5 and 6.6). The same surgical approach as for DIP fusion can be used. The tenotomy of the extensor tendon should be made in such a way that the extensor tendon can be reattached easily. Alternatively, an extensor tendon split can be performed, although it might be difficult to get full access to the joint, remove the osteophytes, and still preserve the extensor



Fig. 6.6 (a, b) DIP arthroplasty with a Silastic implant on the index finger and fusion of the DIP joint in the middle finger with a memory metal device

tendon insertion. After removing the osteophytes, the bone resection line is marked with the trial implant. A Swanson original implant size 00 usually fits well (Fig. 6.7). After bone resection, the two medullary canals have to be prepared and a trial reduction is performed. The implant should lie smoothly in the bone without bulking. The final implant is then inserted and the extensor tendon re-attached. Passive motion should not be tested, as it violates the extensor tendon fixation. The joint is then immobilized for 5–6 weeks with the aim of achieving around 30° active mobility with enough stability for pinching.

Pearls and Personal Recommendation: Surgical Treatment

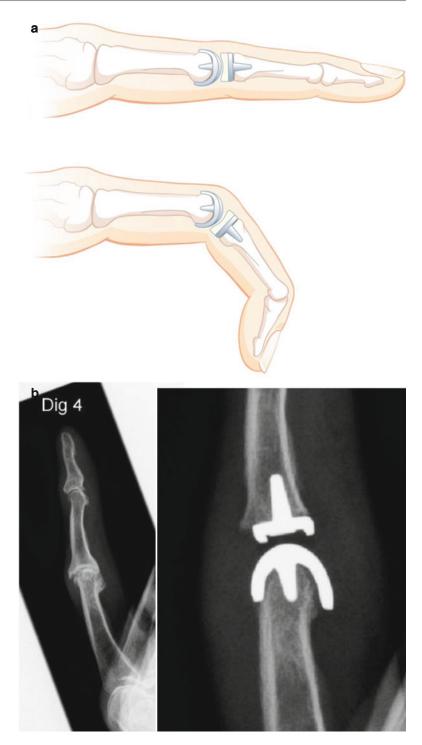
- The volar approach to PIP arthroplasty has at least theoretical advantages over the other approaches.
- DIP fusion with screw fixation allows immediate mobilization of the other finger joints.
- DIP arthroplasty is an interesting alternative to DIP fusion.

Outcome, Including Literature Review

Most publications on replacement of the proximal interphalangeal joint mix the indications and only very few authors focus on patients suffering from primary OA of the fingers. Most series have shown that patients with rheumatoid arthritis had a poorer outcome, due to pre-existing deformity that could not be corrected with the implant [23]. Overall, the results of this procedure in osteoarthritic joint destruction are quite uniform. Pain relief is good to excellent, the average range of motion for almost all implants, including the newer designs, is 40-60°, and there is a high recurrence of pre-existing deformities. In most series, the range of motion could not be improved, and no clear correlation between preoperative mobility and postoperative range of motion is to be expected [22]. The newer designs do not improve the active range of motion, moreover they have a greater potential for complications compared with silicone implants [33, 34].

We have started to use our own implant (CapFlex[®]), a non-cemented real resurfacing prosthesis with short stem fixation and a modular

Fig. 6.7 New PIP joint implant (CapFlex®) as resurfacing prosthesis with minimal bone resection and un-cemented fixation with short stems. (a) Schematic drawing. (b) Preoperative and postoperative radiograph 6 weeks after implantation with good osteointegration of the prosthesis and no secondary dislocation



polyethylene inlay, in different sizes to allow optimal collateral ligament tension. The preliminary results are promising and we plan to use this implant more widely.

PIP joint fusion gives reproducible results regardless of the technique used. Hardware irritation is possible with all techniques and might require metal removal [28].

DIP joint arthrodesis also gives reproducible results but screw fixation is more convenient in most cases [32]. Our own series of 107 DIP joint fusions [35], performed with 2.0 mm screw fixation, showed a solid fusion 6–8 weeks postoperatively in 96 % of the cases. The screw had to be removed in 35 % of the joints, interestingly more often in the two radial digits than in the two ulnar fingers. Mal-union with a residual deviation of more than 5° was observed in 14 % of the patients. Overall, patient satisfaction was high. From these results we can conclude that screw placement flush to the bone surface and careful positioning of the arthrodesis, especially of the rotation, is essential.

For DIP arthroplasty, only few series are available. Wilgis [26] report about 38 digits treated with Silicone arthroplasties with a mean of 10 years follow-up. Less than 10 % of the implants had to be removed and the average range of motion was 33°. Similar results were found by Brown [27], in 13 patients with 21 flexible silicone implant arthroplasties good to excellent results with only one complication was reported. He considers DIP arthroplasty as an alternative to DIP joint fusion. In a recent communication by Zweifel et al. [36] 123 consecutive DIP silicone arthroplasties with two different techniques: one with tendon sparing, and one with tenotomy and subsequent tendon refixation were analyzed. Good pain relief was achieved and 20-30° residual range of motion. No long-term results of this series are yet available.

Pearls and Personal Recommendation: Outcome

PIP arthroplasty, regardless of the implant, has quite uniform results with good pain relief and an average range of motion of 40–60°. The more complex implant designs have not yet proved superior to silicone implants.

DIP fusion with screw fixation provides good fusion rates and the procedure has high patient satisfaction. DIP arthroplasty may be an interesting alternative.

Complications of Treatment

The complication rate in PIP arthroplasty is significant and the following section addresses the problems. While the main problems of silicone devices are implant failure and cystic bone formation with time [23], more complex joints might show implant loosening and joint dislocation. In the long-term follow up, it is to be expected that 10-30 % of the silicone implants at PIP level show a fracture. This is clearly less than in the MCP joints and does not always mean revision surgery. In comparison with the MCP joint, the rate of silicone synovitis is less and in our experience only a few cases need revision for this problem. As already mentioned, recurrence of pre-existing deformity is high. The overall revision rate in the literature varies from 2% up to 13% [23]. Our own series of 612 consecutive PIP silicone arthroplasties over 10 years, with the majority of cases operated on for OA, showed a revision rate of 5.5 % [37]. The main reasons for revision were pain, limited range of motion and joint deformity, mainly ulnar deviation. Most patients showed a combination of these problems. Revision surgery gave good to moderate pain relief, no change in the range of motion, and a high recurrence of joint deformity.

The newer generation of prostheses, including pyrocarbon, ceramic and other resurfacing implants, show a relatively high complication rate with implant dislocation and problems in bone fixation in non-cemented devices [34, 38–40]. A permanent squeezing, unrelated to pain, was observed with some of the implants.

Conclusions/Personal View

Personal Top Ten Pearls: Osteoarthritis of the Fingers

- Primary osteoarthritis of the PIP and DIP joints is very common and will be one of the growing markets for surgical treatment in the future.
- There is increasing evidence that genetic predisposition is a major factor in disease development.
- OA of the fingers is an incurable disease and all attempts to treat this condition conservatively are limited to modifying the symptoms and, at best, slowing down the progression.
- Our standard treatment for painful destroyed PIP joints is arthroplasty, with joint arthrodesis for DIP joints.
- So far, the more complex two-component PIP prostheses have failed to perform better than silicone implants. At the moment, therefore, the silicone spacer remains our standard implant.
- We prefer the volar approach for PIP arthroplasty in patients with OA.
- PIP arthroplasty is indicated in the index finger as well as in the other fingers, but immobilisation during rehabilitation is prolonged in order to achieve sufficient lateral stability.
- Revision surgery for PIP arthroplasties gives good pain relief but does not improve the range of motion; recurrence of the deformity is to be expected.
- Screw fixation is our preferred method for DIP arthrodesis.
- There may be a future potential for DIP arthroplasty, especially in the ulnar fingers, but no long-term results of this procedure are available yet.

References

- Kalichman L, Cohen Z, Kobyliansky E, Livshits G. Patterns of joint distribution in hand osteoarthritis: contribution of age, sex, and handedness. Am J Hum Biol. 2004;16(2):125–34.
- Geyer M, Grassel S, Straub RH, Schett G, Dinser R, Grifka J, et al. Differential transcriptome analysis of intraarticular lesional vs intact cartilage reveals new candidate genes in osteoarthritis pathophysiology. Osteoarthritis Cartilage. 2009;17(3):328–35.
- Goekoop RJ, Kloppenburg M, Kroon HM, Dirkse LE, Huizinga TW, Westendorp RG, et al. Determinants of absence of osteoarthritis in old age. Scand J Rheumatol. 2011;40(1):68–73.
- Williams FM, Skinner J, Spector TD, Cassidy A, Clark IM, Davidson RM, et al. Dietary garlic and hip osteoarthritis: evidence of a protective effect and putative mechanism of action. BMC Musculoskelet Disord. 2010;11:280.
- Feldon P, Belsky MR. Degenerative diseases of the metacarpophalangeal joints. Hand Clin. 1987;3(3): 429–47.
- Hirsch JH, Killien FC, Troupin RH. The arthropathy of hemochromatosis. Radiology. 1976;118(3):591–6.
- Wilder FV, Barrett JP, Farina EJ. Joint-specific prevalence of osteoarthritis of the hand. Osteoarthritis Cartilage. 2006;14(9):953–7.
- Schouten JS, van den Ouweland FA, Valkenburg HA. Natural menopause, oophorectomy, hysterectomy and the risk of osteoarthritis of the dip joints. Scand J Rheumatol. 1992;21(4):196–200.
- Altman R, Alarcon G, Appelrouth D, Bloch D, Borenstein D, Brandt K, et al. The American College of Rheumatology criteria for the classification and reporting of osteoarthritis of the hand. Arthritis Rheum. 1990;33(11):1601–10.
- Kellgren JH, Lawrence JS. Radiological assessment of osteo-arthrosis. Ann Rheum Dis. 1957;16(4): 494–502.
- Varju G, Pieper CF, Renner JB, Kraus VB. Assessment of hand osteoarthritis: correlation between thermographic and radiographic methods. Rheumatology (Oxford). 2004;43(7):915–9.
- Arrestier S, Rosenberg C, Etchepare F, Rozenberg S, Foltz V, Fautrel B, et al. Ultrasound features of nonstructural lesions of the proximal and distal interphalangeal joints of the hands in patients with finger osteoarthritis. Joint Bone Spine. 2011;78(1):65–9.
- Grainger AJ, Farrant JM, O'Connor PJ, Tan AL, Tanner S, Emery P, et al. MR imaging of erosions in interphalangeal joint osteoarthritis: is all osteoarthritis erosive? Skeletal Radiol. 2007;36(8):737–45.

- Haugen IK, Lillegraven S, Slatkowsky-Christensen B, Haavardsholm EA, Sesseng S, Kvien TK, et al. Hand osteoarthritis and MRI: development and first validation step of the proposed Oslo Hand Osteoarthritis MRI score. Ann Rheum Dis. 2011;70(6):1033–8.
- Fioravanti A, Fabbroni M, Cerase A, Galeazzi M. Treatment of erosive osteoarthritis of the hands by intra-articular infliximab injections: a pilot study. Rheumatol Int. 2009;29(8):961–5.
- Strauss EJ, Hart JA, Miller MD, Altman RD, Rosen JE. Hyaluronic acid viscosupplementation and osteoarthritis: current uses and future directions. Am J Sports Med. 2009;37(8):1636–44.
- Huskisson EC. Glucosamine and chondroitin for osteoarthritis. J Int Med Res. 2008;36(6):1161–79.
- Uebelhart D. Clinical review of chondroitin sulfate in osteoarthritis. Osteoarthritis Cartilage. 2008;16 Suppl 3:S19–21.
- Ikeda M, Ishii T, Kobayashi Y, Mochida J, Saito I, Oka Y. Custom-made splint treatment for osteoarthritis of the distal interphalangeal joints. J Hand Surg Am. 2010;35(4):589–93.
- Rizzo M, Beckenbaugh RD. Treatment of mucous cysts of the fingers: review of 134 cases with minimum 2-year follow-up evaluation. J Hand Surg Am. 2003;28(3):519–24.
- Woodworth JA, McCullough MB, Grosland NM, Adams BD. Impact of simulated proximal interphalangeal arthrodeses of all fingers on hand function. J Hand Surg Am. 2006;31(6):940–6.
- Herren DB, Simmen BR. Palmar approach in flexible implant arthroplasty of the proximal interphalangeal joint. Clin Orthop Relat Res. 2000;371:131–5.
- Takigawa S, Meletiou S, Sauerbier M, Cooney WP. Long-term assessment of Swanson implant arthroplasty in the proximal interphalangeal joint of the hand. J Hand Surg Am. 2004;29(5):785–95.
- 24. Iselin F, Conti E, Perrotte R, Stephan E. Long-term results of proximal interphalangeal resectionarthroplasty using the Swanson silastic implant. Ann Chir Main Memb Super. 1995;14(3):126–33.
- Swanson AB, de Groot Swanson G. Flexible implant resection arthroplasty of the proximal interphalangeal joint. Hand Clin. 1994;10(2):261–6.
- Wilgis EF. Distal interphalangeal joint silicone interpositional arthroplasty of the hand. Clin Orthop Relat Res. 1997;342:38–41.

- Brown LG. Distal interphalangeal joint flexible implant arthroplasty. J Hand Surg Am. 1989;14(4): 653–6.
- Uhl RL. Proximal interphalangeal joint arthrodesis using the tension band technique. J Hand Surg Am. 2007;32(6):914–7.
- Pellegrini Jr VD, Burton RI. Osteoarthritis of the proximal interphalangeal joint of the hand: arthroplasty or fusion? J Hand Surg Am. 1990;15(2): 194–209.
- Chamay A. A distally based dorsal and triangular tendinous flap for direct access to the proximal interphalangeal joint. Ann Chir Main. 1988;7(2):179–83.
- Ayres JR, Goldstrohm GL, Miller GJ, Dell PC. Proximal interphalangeal joint arthrodesis with the Herbert screw. J Hand Surg Am. 1988;13(4): 600–3.
- 32. Tomaino MM. Distal interphalangeal joint arthrodesis with screw fixation: why and how. Hand Clin. 2006;22(2):207–10.
- Stoecklein HH, Garg R, Wolfe SW. Surface replacement arthroplasty of the proximal interphalangeal joint using a volar approach: case series. J Hand Surg Am. 2011;36(6):1015–21.
- Wijk U, Wollmark M, Kopylov P, Tagil M. Outcomes of proximal interphalangeal joint pyrocarbon implants. J Hand Surg Am. 2010;35(1):38–43.
- Baumgartner U, Simmen BR. Analyse von DIP Schrauben Arthrodesen. Doctoral thesis. 1996.
- 36. Zweifel CJ, Sirotakova M, Sierakowski A, Elliot D. DIP joint replacement: indications and outcome of a new technique. Interlaken: Annual congress Swiss Society for Surgery of the hand; 2010.
- Schindele S, Keuchel T, Herren DB. Revision arthroplasty of the PIP joint after primary failure of a silicone implant. Oslo: FESSH congress; 2011.
- Chung KC, Ram AN, Shauver MJ. Outcomes of pyrolytic carbon arthroplasty for the proximal interphalangeal joint. Plast Reconstr Surg. 2009;123(5): 1521–32.
- Herren DB, Schindele S, Goldhahn J, Simmen BR. Problematic bone fixation with pyrocarbon implants in proximal interphalangeal joint replacement: short-term results. J Hand Surg Br. 2006;31(6): 643–51.
- Drake ML, Segalman KA. Complications of small joint arthroplasty. Hand Clin. 2010;26(2):205–12.

Rheumatoid Hand and Wrist Problems

7

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Keywords

Rheumatoid arthritis • Hand • Wrist • Synovitis • Tenosynovitis • Diagnosis • Rehabilitation • Surgery • Splints • Arthroplasty • Fusion

Introduction

The function of the hand can be considered in four categories; pinch, grasp, precise manipulation and activities of daily living [1]. Even minor impairment in movement, strength, dexterity or sensation can have huge impact on function. In rheumatoid arthritis all these factors conspire to cause impairment, affect productivity and may create social stigma.

Rheumatoid arthritis is a progressive, systemic, inflammatory autoimmune condition that affects the synovial lining of diarthrodial joints and tendon sheaths. It is characterised by the production of pannus, which results in destruction

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Hand Therapist, Upper Limb Unit, Wrightington Hospital, Hall Lane, Appley Bridge, Wigan WN6 9EP, UK of articular surfaces through the release of activated neutrophils and soft tissue microvascular ischaemia. This causes alteration in joint kinematics and stretching or rupture of ligamentous restraints. The hand is affected in more than 70 % of individuals with rheumatoid arthritis. The estimated prevalence of rheumatoid arthritis in the UK for the twenty-first century is at least 1.16 % in women and 0.44 % in men [2]. The annual incidence for women is estimated to be 36/100,000, with an increase in incidence up to 45 years and a decline after 75 years. In men, the annual incidence is estimated at 14/100,000 with rare cases under 45 years and a steep rise with age [3].

The management of the rheumatological patient is complex and involves a multidisciplinary approach; the rheumatologist to control the systemic inflammation without which surgical intervention is futile, the hand therapist to help assess the patients needs and functional deficit, to provide splints and post-surgical rehabilitation and the hand surgeon, who will frequently be asked to address the pathological effects of the disease process or to address the sequelae of biomechanical disturbance in the hand.

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Presentation, Investigation and Treatment Options

The diagnosis of rheumatoid arthritis has often been made before the patient attends the hand clinic. Some patients, however, will present denovo with pauciarticular pain and inflammation and the hand surgeon must be alert to the possible diagnosis. The American Rheumatological Association has provided criteria for the diagnosis of rheumatoid arthritis based on analysis of over 260 patients with RA, compared to an equal number of controls with other rheumatic diseases [4]; morning stiffness in the joints for at least 1 h, soft tissue swelling of three or more joint areas observed by a physician, swelling of the proximal interphalangeal, metacarpophalangeal or wrist joints, symmetrical arthritis, rheumatoid nodules, rheumatoid factor positive serology and the radiographic detection of erosions and/or periarticular osteopenia in hand or wrist joints. The first four criteria listed must be present for at least 6 weeks. Rheumatoid arthritis is defined by the presence of four or more criteria. These criteria have been reported to be 91-94 % sensitive and 89 % specific for the diagnosis of RA, but have been criticised for lack of sensitivity in the diagnosis of early disease [4]. Early diagnosis of rheumatoid arthritis is aided by the use of serological tests. The presence of rheumatoid factors is associated with RA in up to 80 % of patients. Anti-cyclic citrullinated peptide antibody (ACPA) may be detected earlier in the disease and when present makes the diagnosis of RA highly likely. Antikeratin Antibody is less useful for diagnosis, but may have prognostic value [5]. The American College of Rheumatology and the European League Against Rheumatism have
 Table 7.1
 ACR/EULAR criteria for new diagnosis RA [6]

Patients should be tested if:	
1. they have at least 1 joint with definite clinical synovitis.	
2. the synovitis is not better explained by another disease.	r
Sum of score of categories A−D≥6/10 patient classified as definite RA	
A. Joint involvement	
1 large joint	0
2-10 large joints	1
1-3 small joints	2
4-10 small joints	3
>10 joints (at least 1 small joint)	5
B. Serology	
Negative RF and negative ACPA	0
Low-positive RF or low-positive ACPA	2
High positive RF or high-positive ACPA	3
C. Acute-phase reactants	
Normal CRP and normal ESR	0
Abnormal CRP or abnormal ESR	1
D. Duration of symptoms	
<6 weeks	0
≥6 weeks	1

developed a revised set of criteria, more able to diagnose early disease, using these serological tests, before some of the longer term sequelae have developed (Table 7.1) [6].

The assessment of a patient with rheumatoid arthritis may involve all members of the multidisciplinary team. Often the patient will have already been assessed by their rheumatologist and referred to the hand surgeon to address a specific problem. If the referral has come from another source and the symptoms are more systemic in nature, the surgeon may wish to refer to a rheumatologist in the first instance for medical control of the inflammatory process. Ongoing inflammation is likely to compromise the outcome of any surgical intervention. Early assessment by an experienced hand therapist is extremely valuable. The therapist is often able to take more time with the patient to assess the individual functional deficits and concerns and desires of the patient regarding treatment. In many instances the provision of amended implements, splints or orthoses can prevent the need for surgery and improve the patient's independence.

In performing an assessment, the surgeon should be mindful that the aims of the patient and the aims of the clinician may be at odds. Surgeons and rheumatologists both advocate surgery to relieve pain or improve function [7]. Souter found that most RA patients rate pain relief as the predominant aim of hand surgery [8]. More recent studies suggest that patients with hand deformity are predominantly looking for improved function and that pain relief is a secondary aim [9, 10]. The same authors also concluded that aesthetic considerations may be an important factor in determining satisfaction with surgical outcome. Generally, the rheumatoid surgeon will address any lower limb problems before upper limb, so not to compromise the outcome of any upper limb reconstructive procedures through the use of walking aids. In the upper limb, joints are addressed in a proximal to distal direction. The reasons are two-fold; firstly proximal deformity can result in distal imbalance and deformity that would result in early recurrence after reconstruction if not addressed and secondly, there is limited value in a functioning hand if it cannot be moved in space to a position to perform tasks. In the hand, the rheumatoid surgeon will want to consider performing preventative surgery before reconstructive interventions and in general to start with "winning" procedures in which outcomes are reasonably predictable and which provide the patient with significant improvement in quality of life. The three best examples include wrist fusion, rebalancing of the MCPJ with silicone internal splinting and thumb MCPJ fusion.

In the hand, the rheumatoid surgeon will want to start with "winning" procedures. The three best examples include wrist fusion, rebalancing of the MCPJ with silicone internal splinting and thumb MCPJ fusion.

Clinical assessment of the patient requires consideration of global function and, for the hand surgeon, the function of all joints of the upper limb. The assessment is aided by the recognition of characteristic patterns of deformity that will be discussed in detail in this chapter. Four stages of clinical presentation have been described. In stage 1 the synovitis has been present for less than 6 months and medical treatment is recommended, in stage 2 the synovitis has been present for more than 6 months and surgery may be indicated as an adjunct to medical management. In stage 3 specific deformities have developed that are suitable for surgical reconstruction and in stage 4, arthritis mutilans, salvage procedures are indicated [11]. It is important to make a careful assessment of the neurological status of the upper extremity and if considering surgical intervention, clinical and radiographic examination of the cervical spine is recommended. A significant proportion of rheumatoid patients will have atlanto-axial subluxation.

Plain radiographs are routinely employed for diagnosis, assessment and monitoring of treatment (Fig. 7.1). Plain PA and lateral radiographs are routinely used, with additional



Fig. 7.1 Plain PA radiograph of the rheumatoid hand showing characteristic features of metacarpophalangeal and radiocarpal erosion

Score		
0	Intact bony outlines and normal joint space	
1	Erosion less than 1 mm in diameter or joint space narrowing	
2	One or several small erosions, diameter more than 1 mm	
3	Marked erosions	
4	Severe erosions, where there is usually no join space left and the original bony outlines are partly preserved	
5	Mutilating changes, where the original bony outlines have been destroyed	

 Table 7.2
 Larsen scoring system for rheumatoid arthritis

oblique views occasionally required. Numerous radiographic scoring systems have been described to aid management decisions, monitor treatment and document progression. The Steinbrocker method used a four-point scale, from minimal damage (grade 1) to severe damage (grade IV), for global hand and wrist joint damage based on the worst changes in any joint [12]. The most widely used methods are those of Sharp et al. [13] and Larsen et al. [14] both of which have undergone revisions since their original description [15, 16]. The Larsen method employed reference radiographs to score any synovial joint on a scale from 0 to 5, based on joint space narrowing and erosions. Later, Larsen introduced guidelines for scoring (Table 7.2) [17]. Kaarela and Kautiainen proposed including ten metacarpophalangeal joints, both wrists and the second to fifth metatarsophalangeal joints to give a summative score, ranging from 0 to 100 [18]. Radiographs have their limitations, however. Changes occur slowly and their use is therefore limited for guiding response to treatment. Likewise, modern management requires intervention before radiographic changes occur. Radiographic joint space narrowing does not correlate with erosions [19, 20]. These are more reliably detected using high resolution B-mode ultrasound, although neither are as sensitive as clinic examination [21]. Magnetic resonance imaging is sensitive for the detection of erosions and has a role for diagnosis in patients, in whom ACPA antibodies are negative [22].

Surgical Techniques and Rehabilitation

Pathological Effects

Synovitis

One of the cardinal features of RA is the proliferation of synovial tissue within diarthrodial joints. In the hand, the metacarpophalangeal and proximal interphalangeal joints are most commonly affected. Acutely, this causes pain and decreased movement of the joint. Over time, persistent untreated synovitis leads to joint erosion and laxity of the ligaments, capsule and volar plate, which results in joint subluxation, disrupting tendon balance around the joint and leading to deformity. The role of surgery to prevent the long-term sequelae is contentious. There is little evidence that early synovectomy will alter the natural history of the condition, with high rates of recurrence, especially in the hand [23].

Medical control of MCPJ synovitis should be attempted for 6–9 months, with systemic drug therapy or intra-articular steroid injection. Surgery is performed where medical control is unsuccessful, with the aim of reducing symptoms and preventing further damage. Hand surgeons' perception of the outcome of small joint synovectomy is discordant from that of rheumatologists, the latter being rather less optimistic about the effectiveness of this intervention in preventing joint destruction [7]. The procedure can be performed as an open synovectomy or arthroscopically [24]. One series has reported a recurrence rate of 20 % at 84 months after arthroscopic synovectomy.

Tenosynovitis

Approximately 50 % of patients with RA will have tendon involvement [25]. Proliferation of the synovial lining of the tendon sheath predominantly affects the extensor tendons under the extensor retinaculum. The resulting swelling is not usually painful, but can be unsightly and is often the first presentation of the disease. If left untreated, the synovial proliferation can invade the substance of the tendon leading to adhesion and ultimately to tendon rupture. The mechanism of tendon rupture is poorly understood. Original descriptions attributed rupture to abrasion over sharp bony spurs, but in some cases no spur is found [26]. Invasion of the tendon by synovitis does not occur in all cases and is not time dependant, but is thought to be a result of a microvascular response to tissue hypoxia [27].

The risk factors for extensor tendon rupture have been investigated by Ryu et al. [28] They reported an association between volar subluxation of the radius at the distal radio-ulnar joint (DRUJ) resulting in a prominent ulna head dorsally, identification of the scallop sign of the DRUJ described by Freiberg and Weinstein and a duration of symptoms of greater than 6 months [29]. The ring and little finger extensor tendons were reported to be at greatest risk of rupture, with a slight predominance of rupture in the dominant hand [28]. They and others have reported a low rate of tendon rupture in patients undergoing prophylactic tenosynovectomy. Without a control it is impossible to be certain that this intervention altered the natural history [30-32].

Tenosynovectomy is performed dorsally through a longitudinal incision sited ulnar to Lister's tubercle. The extensor retinaculum can be elevated in a radial to ulnar direction, with care to expose all involved extensor compartments. The first extensor compartment does not need to be exposed unless clearly involved in the disease. The tenosynovitis is excised with care taken to preserve the extensor tendons [30]. If the patient has wrist pain then this procedure can be combined with wrist neurectomy. The elevated retinaculum can be split in two, axially, to enable one half to be passed under the extensor tendons to protect from underlying bony prominences and the other half to be sutured back over the tendons to prevent bow-stringing. The results of tenosynovectomy are generally superior to joint synovectomy, with lower rates of recurrence [30, 33].

The diagnosis of extensor tendon rupture is normally straight forward. However, other causes of extensor lag must be excluded; subluxation of the central slip at the MCPJ from the apex of the metacarpal head into the ulna gutter occurs when the radial sagittal band becomes attenuated or stretched, the diminished lever arm result in a

functional weakness (in some cases the tendon comes to lie volar to the axis of the MCPJ and acts as a flexor), MCPJ subluxation or dislocation leading to dysfunction that may manifest as an extensor lag and posterior interosseous nerve palsy secondary to synovitis around the radial neck. Where tendon rupture has occurred, primary tendon transfer can be performed at the time of tenosynovectomy [34]. Vaughan-Jackson was the first to report rupture of an extensor tendon, originally in two patients with osteoarthritis [26, 35], but later in a series of 25 patients with rheumatoid arthritis [35]. Other authors too reported cases of spontaneous extensor tendon rupture [36]. Vaughan-Jackson concluded that tendon ruptures occurred in the ulnar sided tendons and progressed radially and attributed this pathology to attrition from a prominent ulna head and this lesion continues to bear his name.

Rupture of the flexor tendons occur with less frequency. The tendons are at risk of rupture at the level of the wrist and within the synovial sheaths within the digits. As with extensor tendons, the cause is either synovial infiltration or abrasion on a bony spur. One review of over 100 cases reported that 4/5 occur at the wrist and that bony spurs were present in 2/3 of cases [37]. The presentation of flexor tenosynovitis is often painless, with the presence of swelling at the wrist level or, when in the digital sheaths, loss of finger flexion. Trigger finger, or carpal tunnel syndrome, may be the earliest symptoms. Ultrasound or MRI scan may be helpful in assessing the extent of the synovitis. As with extensor tenosynovitis, the treatment is medical in the first instance. Flexor pollicis longus is the flexor tendon at greatest risk of rupture, due to the proximity to the scapho-trapezo-trapezoid joint (Mannerfelt Syndrome) [38].

Rupture of the EPL Tendon

Rupture of the extensor pollicis longus (EPL) tendon is a frequent event in rheumatoid arthritis and is best demonstrated by asking the patient to place their palm flat on a table and to lift the thumb towards the ceiling. An isolated EPL rupture produces variable functional loss, depending on the state of the extensor pollicis brevis (EPB) tendon and the joints of the thumb, but significant functional deficit can be satisfactorily managed with extensor indicis proprius (EIP) transfer. Nalebuff advocated this transfer, as it results in no loss of function in the index finger and reproducibly restores EPL function [34]. The EIP is divided at the index MCPJ, where it lies just ulna to the extensor digitorum communis (EDC) tendon. It is then delivered through an incision over the wrist where it is found as the deepest tendon in the fourth extensor compartment and rerouted in the subcutaneous plane to attach to the EPL stump at the base of the thumb by means of a Pulvertaft weave. The muscle belly of EIP extends more distally than for the EDC, which aids identification of the EIP in the fourth compartment. Extensor carpi radialis longus (ECRL), or extensor digiti minimi (EDM) may be used where EIP is required or has been used for reconstruction on the ulna side of the hand. Before EIP is harvested, it is important to ensure that EDC to the index finger remains intact. Where the rupture is identified early and the EIP muscle remains healthy, function may be restored by use of a tendon graft to bridge the gap between the distal tendon stump and the muscle. Palmaris longus is most frequently used, but, where this is not available, grafts can be taken from ECRL, plantaris or the extensor tendon of the fourth toe. When the presentation is delayed, the distal stump of EPL may be sutured by means of Pulvertaft weave to the tendon of EPB with satisfactory results [39].

Rupture of Long Finger Extensors

Rupture can occur to any of the long finger extensors, but most frequently involves those on the ulnar side of the wrist. Significant extensor lag of the little finger normally implies rupture of both the EDM and EDC tendons. Isolated EDM rupture produces little functional loss and is often unnoticed. When suspected, the diagnosis can be confirmed by firstly asking the patient to make a fist, to defunction the EDC, and then to extend the little finger in isolation. If EDM is ruptured, there will be a lag of approximately 40°. End-toside repair of the distal EDM tendon stump to EDC of the little or ring finger will produce a satisfactory result.

More commonly, the patient will present with rupture of both EDM and EDC to the little finger.

This will result in significant extensor lag and is most commonly reconstructed by transferring EIP to EDM and EDC of the little finger. As with all transfers, tension of the reconstruction is important. Observation of the tenodesis effect under anaesthesia is important: as the forearm is passively pronated and supinated, the normal cascade of finger flexion and extension should be observed. Here, the tension should be set so that the little finger extends slightly further than the ring finger, but passive flexion is still possible in 40° of wrist extension. Great care and attention must also be paid to eliminate the cause of the tendon rupture and prompt surgery is recommended to prevent rupture of other tendons. Extensive tenosynovitis must be removed and, if a prominent ulna head is present (caput ulna syndrome), then the ulna head should be excised. The indications for the Darrach procedure are discussed further in the section on the wrist.

If more than one finger is involved, then other transfer options must be considered. Here end-toside repairs can be helpful. Rupture of EDM and EDC to ring and little finger can be reconstructed by transfer of EIP to EDM and EDC (little) and end-to-side repair of EDC (ring) to EDC (middle). When the EDC (middle) is ruptured too, then EIP can be transferred to both the ring and little fingers and end-to-side repair of EDC (middle) to EDC (index) can be undertaken. Clearly it is preferable to intervene, before this situation arises.

When the EDC (middle) is ruptured too, then EIP can be transferred to both the ring and little fingers and end-to-side repair of EDC (middle) to EDC (index) can be undertaken.

It will be clear to the reader that EIP is the workhorse for tendon reconstruction in the rheumatoid hand, but what if this is not available? Wrist extensors can be employed in reconstructions, but perform less well because the excursion is less than the long finger extensors. Extensor carpi ulnaris should not be used, as it is an important wrist stabiliser, especially in the rheumatoid patient in whom the wrist tends to drift into radial deviation. For the same reason, ECRL is preferred, as it is the more radial of the two radial wrist extensors and its removal for transfer may give some advantage to wrist rebalance. Alternatively, tendon grafts can be harvested from the palmaris longus, plantaris, ECRL or ECRB. These may be problematic, with a risk of secondary adhesions [40, 41]. Nakamura and Katsuki reported problems with restricted finger flexion after graft reconstruction of multiple extensor tendon ruptures and highlighted that patient satisfaction was more closely related to pulp-to-palm distance than to extensor lag [42].

If all the extensor tendons have ruptured, then flexor tendons will suffice using techniques described for nerve palsy. Flexor digitorum superficialis to the middle and ring finger can be employed, using Boyes technique of passing the tendons through the inter-osseous membrane to the extensor tendon stumps [43]. Alternatively, these tendons can be passed radially and dorsally in the subcutaneous plane to lie beneath the extensor retinaculum after harvesting [44]. Avoid passing the tendons ulnarly, as this may promote ulnar carpal drift.

Pass tendon grafts around the radial side of the wrist to avoid wrist imbalance.

Flexor Tendons

Tenosynovectomy is indicated in the flexor sheaths, when medical management has failed to control the disease. Care must be taken to preserve the pulleys during the dissection, with windows created at several levels to permit access for tenosynovectomy. In particular, excision of the A1 pulley has been reported to create instability of the MCPJ, bowstringing and increased ulnar deviation and should be avoided [45].

Isolated FPL rupture is the most common presentation and can be disabling, preventing the patient from performing pinch grip. The aim of surgery is of course to restore function, but also to prevent further ruptures by surgical treatment of the tenosynovitis and any bony spurs. Reconstruction may be performed, using either a graft (palmaris longus or FCR) or with tendon transfer using ring finger FDS. The rare situation of multiple flexor ruptures is likely to require use of grafts and tendon transfers and in extreme cases fusion of the proximal interphalangeal joint to restore function.

Rheumatoid Nodules

Rheumatoid nodules are firm fibrous lesions that are found in some patients with rheumatoid arthritis. They are not quite pathognemonic, as they can also occur in other conditions such as lupus and in healthy children [46]. They are typically found on the extensor surfaces and are commonly seen around the elbow, but also in the hands. The histology reveals a lesion with a shell of fibrous tissue surrounding a centre of fibrinoid necrosis. The boundary between these elements consists of palisades of macrophages and fibroblasts arranged in a radial manner. The presence of nodules is a marker for severity of the disease in most cases. A variant of rheumatoid arthritis exists, rheumatoid nodulosis of the hand, in which the patient develops multiple nodules, but the prognosis for joint disease is good [47]. The indications for surgical excision include erosion, infection, neural compression, pain and limitation of hand function [46].

Biomechanical Disturbance

Synovitis, joint erosion and tendon dysfunction lead to altered biomechanics in the hand in the rheumatoid patient. Joint subluxation and dislocation are frequent events, especially at the wrist and metacarpophalangeal joints. This joint subluxation disturbs the fine balance of flexor and extensor forces that maintain the normal cascade within the digits and can result in characteristic deformity.

Metacarpophalangeal Joint

The development of deformity at the metacarpophalangeal joint in RA is multifactorial. It used to be said that 1/3 of patients would develop ulnar drift, but, with improved medical control, the incidence appears to have decreased. Drift occurs as a result of both intrinsic and extrinsic factors. The fingers tend to drift into ulnar deviation with volar subluxation of the proximal phalanx. The intrinsic factors include anatomical predisposition (the ulna condyle of the metacarpal is smaller than the radial) and pathological causation (joint erosion and synovitis resulting in attenuation of the collateral ligaments, volar plate and preferentially the radial saggital bands). The extrinsic factors then exacerbate the problem; radial deviation of the rheumatoid wrist alters the line of pull of the long flexors and extensors into a more ulna position at the MCPJ, contraction of the ulnar intrinsic muscle and subluxation of the central slip into the ulna gutter accentuates this problem. Dynamic extrinsic factors are also present; the forces when a key-pinch is formed between thumb and index finger, push the digit in an ulna direction and gravity is also thought to play a role [48]. Recent research has pointed to a primary hypoxia within the ulnar intrinsics as a potential cause of this deformity [49].

Management of the MCPJ in RA depends on the stage of disease and successful treatment relies on the correction of proximal deforming forces. See the section on the wrist for further discussion. Where ulna deviation is present without marked joint subluxation or erosion the judicious use of splints may be beneficial. There is no evidence that splints will prevent ulna deviation, but their use may have a role in management of pain [50]. A well conducted, randomised trial including 120 patients with early RA observed over a period of 12 months found no difference in grip strength, deformity, hand function or pain in those with and without static splints applied to immobilise the wrist, MCPJ and PIPJ [51]. A challenge of all studies investigating the use of splints and orthoses, is the standardisation of splint use and amount of stress applied making true comparison difficult.

Synovectomy and Soft Tissue Balance

Although practiced less often in the UK, rebalancing of the MCPJ, in which the articular surface remains healthy and passive correction is possible, may provide some lasting benefit, both for cosmesis and function. The procedure can be performed through a single transverse incision, just proximal to the metacarpal heads, through four longitudinal incisions over each MCPJ, or through two longitudinal incisions situated between the index and middle MCPJ and ring and little MCPJ. It is the author's preference to use a single transverse incision, but care must be taken to preserve the venous bundles that lie in the gutters between the metacarpals. The joint is approached through the attenuated radial sagittal band using a paratendinous incision. Frequently, the ulna sagittal band is contracted and will need to be released to allow tendon centralisation at the end of the procedure. A midline capsulotomy is performed, to allow synovectomy and inspection of the joint surface. The ulna intrinsic tendon should be released from the base of the proximal phalanx. Transfer of this intrinsic tendon to the radial side of the adjacent digit has been shown, by some authors, to be beneficial in preventing recurrence [52, 53]. The transferred tendon should be sutured to the insertion site of the radial intrinsic at the base of the proximal phalanx. A bone anchor can be used [54]. It is the authors preference to reef the radial collateral ligament, using a 2/0 braided synthetic suture, with a grasping stitch at either end. The radial sagittal band is double-breasted to centralise the extensor tendon, or a distally based slip of extensor hood can be harvested, passed under the radial collateral ligament in a palmar to dorsal direction and sutured back to the EDC using a Pulvertaft weave to reconstruct the radial sagittal band (Fig. 7.2) [55]. Postoperatively, the hand should be placed in a volar slab with ulnar gutter initially. Static or dynamic



Fig. 7.2 Sagittal band reconstruction (From Dell et al. [55], Reproduced with permission)

splinting can then be used for 8–10 weeks to maintain the correction, with a further period of night splinting up to 6 months.

MCPJ Arthroplasty

The history of metacarpo-phalangeal joint arthroplasty began in the 1950's in America, with a hinged metal implant designed for use at the MCPJ and PIPJ. These devices were used in young service personnel for post-traumatic reconstruction [56]. In the 1960's, Swanson and Neibauer independently developed silicone rubber implants for the MCPJ, that were intended as internal splints to allow early motion of joints in which soft tissue balancing had been performed. The Neibauer implant was reinforced with a Dacron mesh, in an attempt to strengthen the implant. The antecedent to this had been resection of the metacarpal head, combined with the balancing procedures described previously. Acceptable results could be achieved with this procedure, if acceptable alignment could be maintained. However, frequently, this required prolonged immobilisation with pins, or external splints leading to joint stiffness. Swanson introduced the concept of fixation by fibrous encapsulation, whereby the inert silicone implant provides enough initial stability to allow early protected mobilisation, while a functionally adapted fibrous capsule forms around the joint with maintenance of the space between the bones [57]. Pistoning of the implant within the bone was considered to allow greater range of movement and reduce the risk of implant breakage. The Swanson implant has stood the test of time and is used in nearly every country in the world.

The surgical technique for implantation is straightforward and emphasis is placed on release and balance of the soft tissues. The joint is approached as described previously, the neck of the metacarpal is exposed subperiosteally and the head excised. This cut should be angled palmarward and care taken to remove any sharp edges of bone. This is followed by extensive soft tissue release, including release of the collateral ligaments, volar plate and ulnar intrinsic tendon, if necessary, until it is possible to dislocate the base of the proximal phalanx dorsal to the metacarpal. It is important to maintain the balance of this release, such that, when traction is applied to the digit, a rectangular space is left between the bone ends both in extension and flexion. It should not be necessary to remove any of the base of the proximal phalanx. Hand reaming of the bones starts with the phalanx for the index, middle and little fingers, as this is likely to determine the size of the implant. In the ring finger the metacarpal is narrower and this should be reamed first, to determine the implant size. The largest implant size possible should be inserted. The soft tissue balancing is completed with careful closure of the soft tissues around the implant using the techniques described previously. At completion of

the procedure, the fingers should not fall into

ulnar deviation when the hand is raised from the

table in neutral forearm rotation. The outcome of Swanson MCPJ arthroplasty, reported initially by the originator in 358 digits at 2-5 years follow-up, was good, with 97 % exhibiting less than 10° of ulnar drift, joint subluxation corrected in 99 % and an average 60° arc of motion [57]. Implant fracture was reported in 2 % and implant dislocation in 1 %. The infection rate was 0.5 %. In a more recent prospective series, from an independent centre, patient rated outcomes improved significantly in the short term, but subjective parameters, such as grip and pinch strength, were not significantly altered [58]. The short term outcome does not differ significantly between digits [59]. Long term followup studies demonstrated that, over time, the rate of implant fracture increases in a linear fashion, such that, at an average of 14 years, the implant fracture rate is as high as 67 % [60]. Silicone elastomer is known to be susceptible to crack propagation and tearing. The implants have been shown to fail in a very predictable manner, with fretting of the implant surface on the dorsal radial surface at the base of the distal stem with propagation in a volar and ulnar direction [61]. Swanson introduced titanium grommets to try to shield the neck of the implant from sharp bone edges, which has been shown to reduce the incidence of implant fracture in the short term. [62] Newer silicone implants have been developed in an attempt to address some of the issues of the Swanson implant, with more material around the

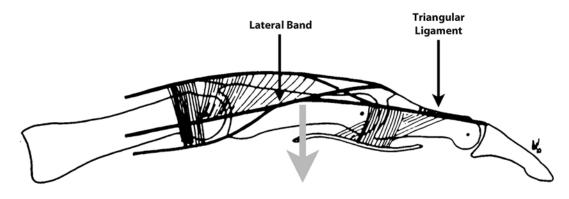


Fig. 7.3 Development of boutonniere deformity (Copyright A Watts)

base of the stems to try to prevent fracture, more volar hinges to give the long extensors greater biomechanical advantage and a preflexed implant to mimic the normal resting position of the MCPJ. Equivalent outcomes have been reported with these newer implants, the Sutter, Avanta and Neuflex, with no significant difference in fracture rate [63–67]. One study has reported a greater flexion arc with the Neuflex implant [68].

Following on from the success of the silicone implants, unlinked anatomical joint resurfacing arthroplasty has increased in popularity. Early implants were of the constrained-hinge design, but these often failed by fracture or loosening, leading to recurrent deformity. Other designs now use pyrocarbons and other composite materials. Pyrocarbon is a synthetic material with a high strength graphite core coated with a pyrolytic carbon layer, formed by heating a hydrocarbon gas to approximately 1,300 °C. In 1999, Cook et al. reported their experience in 53 predominantly rheumatoid patients, with 151 anatomical unconstrained MCPJ implants made from pyrocarbon (Fig. 7.1) [69]. The authors reported a revision rate of 12 %, with a 10-year survival rate of 81.4 %. Pyrocarbon MCPJ arthroplasty has been shown to reduce pain scores and increase range of movement in the short term, but implant subsidence has been documented [70]. Complications including subluxation or dislocations of the unconstrained components, loosening of the implants, implant fracture and joint stiffness have all been described for unconstrained anatomical implants [69, 71].

Digital Deformities

Digital deformities occur secondary to synovitis, joint destruction, ligament incompetence, muscle imbalance and proximal joint deformity. The success of reconstructive interventions depends on the mobility of the deformity. When the deformity becomes fixed, the options for treatment are limited, with joint fusion providing the most reliable outcome. The digits collapse into predictable patterns of deformity depending on the site of joint involvement. With established RA, over 1/3 of patients will develop a boutonnière deformity as a result of synovitis at the PIPJ, leading to incompetence of the triangular ligament between the lateral bands which sublux in a volar direction (Fig. 7.3). Swan neck deformity has approximately half the prevalence of boutonnière deformity and can occur as a result of imbalance at the wrist, MCPJ, PIPJ or DIPJ. Both deformities can be seen in the same hand of a patient with RA.

Swan Neck

A swan neck deformity is characterised by hyperextension at the PIPJ and compensatory flexion at the DIPJ. Assessment of swan neck deformity begins with assessment of the mobility of the joints, then with determination of the joint with the primary pathology. Synovitis at the DIPJ can present with a hot tender swollen joint. The synovitis can cause rupture of the extensor apparatus, leading to a mallet finger that relaxes the lateral bands and transmits all the force from the long



Fig. 7.4 Swan neck deformity

extensors to the base of the middle phalanx that, over time, leads to hyperextension of the PIPJ.

When the primary pathology is in the PIPJ, synovitis will result in pain and swelling. The synovitis leads to stretching and, ultimately, attenuation of the volar plate, check rein ligaments and flexor digitorum superficialis tendon. Incompetence of this volar restraint leads to hyperextension at the PIPJ, resulting in relaxation and dorsal migration of the lateral bands that can no longer extend the DIPJ.

If the primary pathology is a synovitis of the MCPJ, this joint will be more swollen and tender. Over time, the synovitis causes subluxation of the MCPJ and attenuation of the attachment of the central slip to the base of the proximal phalanx. The result is dorsal translation of the lateral bands and transmission of the forces of the long extensors to the base of the middle phalanx which results in hyperextension of the PIPJ and a characteristic sagittal "Z" collapse of swan neck deformity (Fig. 7.4).

Primary wrist pathology can lead to a swan neck deformity, by disrupting the balance of the intrinsic and extrinsic muscles. Carpal collapse results in functional lengthening of the long flexors and extensors, which means that the intrinsic muscles are relatively more powerful and cause MCPJ flexion and PIPJ hyperextension.

These deformities will be passively reducible and mobile initially. The use of a Silver ring, or prefabricated thermoplastic splint may provide

Nalebuff classification	Clinical signs	Pathoaetiology
Type I	Flexible hyperextension deformity PIPJ	Extensor tendon rupture Volar plate attenuation MCPJ subluxation Wrist subluxation
Type II	Tight PIPJ flexion with MCPJ in extension	Instrinsic tightness
Type III	Limited PIPJ flexion in all MCPJ positions	Fixed dorsal subluxation of lateral bands
Type IV	Immobile PIPJ Destruction of joint surface	Adhesion and joint erosion

 Table 7.3
 Nalebuff classification of swan neck deformity



Fig. 7.5 Swan neck deformity corrected with Silver ring

satisfaction by preventing hyperextension [72]. Over time, fixed deformities develop due to fibrosis between extensor and flexor elements and shortening of musculotendinous units. Nalebuff's classification categorises according to the clinical and radiographic findings, which then provides a guide to management (Table 7.3, Fig. 7.5). (*Hand Clin* 1989)

The first step in evaluation of swan neck deformity is to determine the passive range of movement of the PIPJ. If there is a full passive range at the PIPJ then the examiner moves to evaluate the MCPJ. Stability is assessed by passive manipulation and the joint integrity by plain

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radiographic examination. Intrinsic tightness can be assessed using the Bunnell test, in which short intrinsics lead to progressive PIPJ stiffness, as the MCPJ is passively extended. The DIPJ is then assessed to determine the integrity of the terminal extensor tendon and to radiographically assess for joint damage. If the MCPJ is involved, then this should be the first target for intervention, with soft tissue rebalancing and silicone arthroplasty internal splinting, if the joint is damaged or subluxated. If the primary cause is a mallet deformity, then DIPJ fusion will address this. The PIPJ hyperextension is usually addressed with a tenodesis, using either flexor digitorum superficialis (FDS) or the lateral band. FDS tenodesis is simple and reliable, but does not on its own address the DIPJ hyperextension. It is performed by incising the A1 pulley, performing a flexor synovectomy and identifying the radial slip of FDS, which is divided proximally and sutured to the neck of the metacarpal to maintain approximately 20° of PIPJ flexion. The digit can be mobilised immediately in a relative motion splint or dorsal blocking splint for 6 weeks.

If the PIPJ movement is restricted on passive testing, then soft tissue releases may be necessary before the steps outlined above. This can be performed by careful, controlled manipulation, or by surgical release of tight structures starting with dissection of the lateral bands from the central slip, excision of the dorsal PIPJ capsule, then release of the PIPJ collateral ligaments and "Z" lengthening of the central slip [73]. If the joint is fixed in flexion or the joint surface damaged or dislocated, then joint arthrodesis or arthroplasty may be considered. Silicone arthroplasty of the PIPJ for RA still has a role, but often fails to correct deformity [74, 75]. It has been abandoned by some in favour of resurfacing arthroplasty, but the results are less predictable in the rheumatoid patient than in osteoarthritis and some propose that it should be reserved for the ulnar two digits where preservation of motion is advantageous.

Boutonniere

The pathogenesis of boutonniere deformity is more straight forward in RA than that of swan neck deformity, but the management can be more challenging. Boutonniere deformity is characterised by a flexion deformity of the PIPJ and extension at the DIPJ. In RA it is caused by synovitis at the PIPJ, leading to attrition of the triangular ligament between the lateral bands and functional lengthening of the central slip. The result is that the lateral bands sublux volar to the axis of rotation of the PIPJ and become joint flexors. Attenuation and functional lengthening of the central slip means that the lumbricals and interossei become overpowered, a force that is transmitted via the lateral bands to extend the DIPJ. Further, the principle pull of the central band is transferred from the base of the middle phalanx, to the base of the proximal phalanx, resulting in MCPJ hyperextension. Secondary contracture of the volar plate, check rein ligaments, joint capsule and collateral ligaments eventually leads to a fixed boutonniere deformity in which passive correction is not possible.

Assessment of the hand with a boutonniere deformity will often reveal little in the way of functional compromise. The flexed position of the PIPJ is often well tolerated, as grasping functions are maintained. The clinician should assess the mobility of the MCPJ, PIPJ and DIPJ to guide treatment. Special manoeuvres include Elson's test for tension of the lateral bands. The finger is passively flexed at the PIPJ and the mobility of the DIPJ assessed, if in 90° PIPJ flexion the DIPJ remains rigid, then the lateral bands are overpowered. Nalebuff classified the boutonniere deformity according to the ability of the clinician to passively correct the deformity (Table 7.4).

A mobile boutonniere deformity is best treated with adequate medical management of the synovitis, with local steroid injection, if necessary and splinting of the PIPJ in extension. Synovectomy may be indicated, if medical management fails to address the synovitis. The DIPJ should be left free of the splint and mobilisation encouraged, to prevent contracture of the lateral bands. If the DIPJ is already tight in PIPJ extension, because of contracture of the lateral bands, then a Fowler's tenotomy of the distal extensor mechanism over the middle phalanx preserving the oblique retinacular ligament will reliably correct this deformity [76].

	Clinical signs	Pathoaetiology
Stage 1	Correctable extensor lag	Synovitis PIPJ
Stage 2	Marked flexion deformity partially correctable or fixed	Attenuation of triangular ligament Volar subluxation of lateral bands
		Contraction of transverse retinacular ligament
Stage 3	Destruction of PIPJ surface	Erosion

 Table 7.4
 Nalebuff classification of Boutonniere deformity

A mild mallet finger may result, but can be addressed with splinting in most cases.

In boutonniere deformity, with some passive correction, secondary changes have already occurred and reconstruction can be a significant challenge. If the joint is well preserved on plain radiographs, then soft tissue rebalancing with retensioning of the central band through detachment and repair, combined with volar release and reefing of the lateral bands in a dorsal position, may correct the deformity, but runs the risk of limiting PIPJ flexion [77]. Distal Fowler's tenotomy may be required if the DIPJ remains stiff. A static splint is applied to the PIPJ for 6 weeks, allowing movement at the DIPJ.

For static boutonniere deformity the options are more limited. Isolated distal Fowler's tenotomy will address the hyperextension deformity of the DIPJ, which may create a more functional digit. Soft tissue correction of the deformity may be attempted after serial splinting to stretch out the soft tissues, but this is rarely indicated. Occasionally, PIPJ fusion may help especially if joint destruction and pain are a problem. Arthrodesis can be performed using a number of techniques (wires, screws, staples, plates), but it is the authors' preference to use a tension band wire technique with cup in cone preparation of the surfaces.

It is important to remember that the outcome of surgery of the digits is less predictable than surgery to the wrist and MCPJ and careful consideration should be given before undertaking these procedures.



Fig. 7.6 "Z" deformity of thumb

Thumb Instability

The importance of the thumb to hand function needs no elaborating here. In RA, preservation of thumb function is vital to enable basic pinch grips. The system described by Nalebuff provides the basis for a classification system of thumb deformity in RA (Fig. 7.6). The judicious use of medical therapies may slow or prevent progression of thumb deformity and should be considered as the first line of treatment in all cases. Splints and braces may help in the case of passively correctable deformity, such as a Silver ring splint for a swan neck deformity of the thumb. Functional bracing of boutonniere deformity has been shown to decrease pain [78].

Table 7.5 Nalebuff classification of rheumatoid thumb deformity

	Clinical Signs	Pathoaetiology
Туре І	Boutonniere Deformity	MPJ Synovitis
	CMC Unaffected	EPB attenuation
		Volar and Ulnar Subluxation EPL
Type II	Fixed boutonniere CMC effected	As above with involvement of CMCJ
Type III	Swan Neck and adduction	CMCJ subluxation Compensatory MCPJ hyperextension
Type IV	Gamekeeper	Ulnar collateral ligament attenuation
Type V	Swan neck alone	Volar plate laxity of MCPJ
		CMCJ spared
Type VI	Skeletal Collapse	Bone loss leading to telescoping

The surgical management of RA thumb deformity can be challenging. Soft tissue reconstruction alone is likely to fail in the thumb and will often be combined with a fusion procedure. Synovectomy is associated with high rates of recurrence [79]. Fusion of both the MCPJ and IPJ should be avoided.

Passively correctable boutonniere deformity is the most common thumb deformity in RA and can be addressed with MCPJ synovectomy and EPL re-routing to the dorsal capsule of the MCPJ, but recurrence rates are high [80]. Insertion of the EPL into bone has been shown to improve the long term success of this procedure [81]. Fusion of the MCPJ produces a more reliable and robust solution. Stanley et al. reported that in a series of 42 cases successful fusion was achieved in 83 % [82]. Arthrodesis may be performed with a chevron osteotomy, or with a cupand-cone decortication of the joint. In the author's opinion, the latter is more forgiving and allows the joint to be set in the preferred position with ease. Fixation with K-wires, tension band, screws or staples may be performed. In cases where the CMCJ or IPJ are involved, silicone MCPJ arthroplasty with balanced reconstruction of the extensor apparatus may be preferred to maintain motion [83]. This may need to be combined with IPJ fusion, where this is destroyed. In Swanson's original series, 42 out of 44 thumbs assessed at two to six and a half years after surgery were reported to be pain free, with improved range of movement and function. These results have been replicated in other series [80, 84]. Five out of six patients who had had contralateral MCPJ fusion were reported to favour the arthroplasty because of the retained motion [83]. As with other silicone joint replacement, implant failure is a problem with an 11 % fracture rate reported in Swanson's series.

For swan neck deformity, the second most commonly encountered deformity in RA, MCPJ fusion is usually indicated [77], but where the CMCJ is painful, excision arthroplasty with or without tendon autograft may be required and produce excellent long-term outcomes [85]. Attempts at CMCJ arthroplasty, using silicone spacers have not shown good long term outcomes, with high rates of subluxation and silicone synovitis [86–88], even when combined with tendon augmentation [89]. For patients with type V deformity, capsulodesis to address the volar plate insufficiency has been reported to produce good outcomes, with no recurrence in ten thumbs [90].

Rheumatoid Wrist

Introduction

A painless, stable wrist joint is crucial for normal hand function. Involvement of the wrist joint is common and within 2 years of diagnosis more than 50 % of patients will have wrist pain and over 10 years 90 % will have wrist disease [91].

Rheumatoid arthritis of the wrist can cause significant disability and pain and is thus extremely important in the hierarchy of priorities of surgery to be considered in a patient with rheumatoid arthritis of the upper limb. A number of distinct patterns can occur at the wrist, as described by JK Stanley and can be summarised as supination of the carpus, translocation and translation, collapse deformity, volar subluxation and distal radio-ulnar instability.

Patterns of Arthritis (Prof JK Stanley) Carpal Supination Translocation and Translation Collapse Volar Subluxation DRU Instability

Carpal Collapse

Interosseous ligament attenuation between the scapho-lunate and triquetro-lunate joints as well as capsular weakening, causes rotary instability and eventual carpal collapse with a dorsal intercalated segment instability appearance. This can result in significant restriction and instability of the wrist.

Translocation and Translation

Rupture of the Volar RLT (Radio Luno Triquetral) and Dorsal RLT can cause carpal translation, i.e. movement of the carpus wholly towards the ulnar side. Translation is the radial deviation of the carpus, resulting in a manus varus type of deformity of the hand with ulnar deviation of the fingers.

Volar Subluxation

Volar subluxation of the lunate from the lunate fossa, results in encroachment on the carpal tunnel, with formation of a shelf of bone from the distal radius secondary to degenerative changes.

Carpal Supination

A rupture of the dorsal ulnar carpal ligament causes the ulnar side of the wrist to drop away from the radius (supinate), resulting in an apparently prominent distal ulna. This results in significant distortion of the extensor retinaculum. The extensor digitorum minimus and the extensors to the fifth, fourth and third digit are firmly pressed against the ulnar head and any erosions that may be associated with the rheumatoid process. Wrist attrition ruptures described by Vaughan-Jackson are associated with Caput Ulnae syndrome and associated instability of the distal radio-ulnar joint is diagnosed using the piano-key sign.

Distal Radio-Ulnar Instability

Erosions around the insertion of the triangular cartilage, with attenuation of the volar and dorsal ulnar carpal ligaments, the extensor retinaculum and the displacement of the extensor carpi ulnaris from its groove, cause significant distal radio-ulnar instability.

Assessment

Most patients present with a painful swelling in the region of the wrist, with some functional limitation due to pain. A complete evaluation should include a full medical history, including an examination of the cervical spine, the entire upper limb as well as a neurological examination. Again, it is advisable to address lower limb problems first as the patient may require crutches which may interfere with major upper limb reconstructions.

In addition to an assessment of pain, the wrist should be examined to determine the range of motion, the synovitis (swelling) around the wrist joint any deformity, instability and pinch and grip strength. Tenderness and/or pain on resisted flexion or extension may suggest "tendons at risk" [92]. Similarly, assessment of passive versus active motion may indicate a tendon rupture [93]. Other causes of tendon dysfunction should be evaluated, including palsies of the posterior interosseous nerve, ulnar subluxation of the extensor tendons at the MCP joints, due to sagittal band involvement, or subluxation of the MCP joints.

The symptoms of chronic rheumatoid arthritis often mask severe compression palsies, usually affecting the median nerve and, as such, a careful assessment of this should be made at all times. A carpal tunnel decompression in these situations is usually accompanied by a formal synovectomy and carries a favourable prognosis [94].

Like the hand, plain radiographs are routinely employed for diagnosis, assessment and monitoring of treatment. Plain PA and lateral radiographs are routinely used, with additional views occasionally required.

Radiographic evaluation is critical for purposes of operative planning and to help determine what can and cannot be done from a surgical standpoint. Specific patterns of loss of joint space have been observed. The process tends to be most aggressive in the radio-lunate fossa occasionally causing spontaneous ankylosis of the radius to the lunate [95] and/or the scaphoid (Fig. 7.7). This phenomenon represents part of the logic behind a chamay fusion [96]. Midcarpal changes occur at a much later stage and may be associated with a spontaneous ankylosis. The Wrightington Classification [97] is based on radiographic changes within the wrist joint. The classification also outlines treatment recommendations. In stage 1, radiographs

Fig. 7.7 Plain radiograph demonstrating spontaneous fusion of radius to lunate and scaphoid

have cysts and erosions with juxta-articular osteoporosis and synovectomy is recommended. The second stage is the stage of instability and treatment involves soft tissue stabilizations and/or partial arthrodeses of the wrist. A stage 3 wrist shows the presence of significant dislocations and subluxations and only fusions or arthroplasties can be offered as possible treatments. In stage 4 disease the destruction of the distal radius is catastrophic (mutilans type) and the only options are bony fusions (Fig. 7.8). Simmen described three groups of radiographic changes; Type 1 is the ankylotic type, Type 2 is the osteoarthritic type with some stability and Type 3 the disintegrative type [98].

Non-surgical Treatment

It has been shown that wearing splints in acute RA for as little as 4 weeks can help with pain [99]. However, from a functional point of view the results were not significantly different when



Fig. 7.8 Plain radiograph demonstrating significant destruction of carpus and distal radius

compared with controls. Other studies have shown static splinting in early RA do not help function or reduce pain, when compared with placebo [51]. Intra-articular steroid instillation in the wrist, combined with splintage, does not improve pain related outcome measures [100]. Bliddal et al. compared the use of Etanercept with methyl predinisolone in a randomized trial and found no significant difference in outcome measures. The researchers concluded that, in view of the prohibitive costs of Etanercept, it should be reserved for patients with adverse effects to steroid injections [101, 102].

Anti-TNF agents and other newer pharmacologic agents have shown great promise in addressing problems with the progression of rheumatoid arthritis and have limited the damage done by synovitis, resulting in fewer patients needing surgical intervention. Although Grennan et al. showed that continuing methotrexate does not increase the risk of peri-operative infection [103], the newer generation of medications appear to have a degree of peri-operative morbidity, particularly in patients on corticosteroids [104].



Fig. 7.9 (a) Persistent dorsal wrist synovitis despite medical treatment. (b) Dorsal approach to wrist with synovitis exposed

Surgical Treatment

Patients requiring surgery can be divided broadly into three groups. In the conservative group, patients have normal joint spaces and some erosive radiographic changes with florid soft tissue disease. These patients would respond to synovectomy and dorsal stabilization [105, 106]. Patients in the reconstructive group who have localized radio-carpal arthritis require bony procedures aimed at stabilizing the radio-lunate articulation, or the distal radio-ulnar joint. In the salvage group, the mid-carpal joint is grossly damaged, with no satisfactory residual joint space and no pain-free articulation. In these situations, the treatment of choice is either an arthrodesis or replacement surgery. The timing for surgical intervention is controversial, but recalcitrant swelling and pain, despite conservative measures over a 3-6 month period, would be a reasonable indication for operative treatment [28, 107].

Synovectomy

This is usually reserved for patients with synovial disease, in whom the joint spaces are well preserved. Dorsal synovectomy is performed through a midline dorsal incision and the wrist joint is approached through the third compartment (Fig. 7.9a, b). The extensor retinaculum is raised as a flap from the ulnar side allowing for an extensor tenosynovectomy to be performed. At the end of the procedure, if the ulnar displacement of the extensor tendons persist, a dynamic sling of the extensor carpi radialis longus can be fashioned, leaving it attached distally and taking it deep to the tendons and bringing it back to be sutured to the extensor carpi radialis brevis. A posterior interosseous neurectomy can be performed deep to the fouth compartment tendons, usually at the level of the distal radio-ulnar joint. Attention should be paid to prominent bony spurs over the DRUJ and the wrist joint and these should be removed. Arthroscopic synovectomy is preferred in patients with isolated wrist disease, without tenosynovitis. Although short-term benefits are obtained, these do not correlate with long term disease progression. End to side tendon repairs are often used to restore tendon function. Tendons most commonly involved include the extensor digiti minimi at the DRUJ, the extensor pollicis longus at the Listers tubercle and the extensor digitorum communis.

A flexor synovectomy is done through an extended carpal tunnel approach. Volar disease is often masked, thus making flexor tendons vulnerable to injury. The situation is accentuated by the fact that flexor tendons run deep to the transverse carpal ligament and are crowded into a relatively inelastic carpal tunnel space. The flexor pollicis longus (STT joint) and the flexor digitorum profundus are especially at risk. In addition to a complete carpal tunnel release, the flexor tendons are visualized in the forearm space of Parona and any prominent bone spurs and ostephytes are excised. Tendon ruptures are not usually amenable to primary repair and tendon grafting, or tendon transfers are utilized as appropriate. Flexor pollicis longus ruptures are dealt with by interphalangeal joint arthrodeses. With the other fingers, isolated FDS tears are found to do well with non-operative treatment. However, if both the FDS and FDP are lost, tendon transfers and/or grafting is essential.

Management of the Distal Radio-Ulnar Joint

Dorsal subluxation of the distal ulna, with supination of the carpus, causes significant functional problems, due to pain and instability. Various treatment methods include, the Darrach procedure (excision of the distal ulna), Sauve Kapanji Procedure (arthrodesis of the DRUJ) and ulnar head arthroplasty.

Darrach's Procedure

This involves the resection of the distal ulna, through a dorsal exposure to the DRUJ, through the fifth dorsal compartment. The capsule of the DRUJ is elevated and preserved, thus protecting the ECU subsheath. The head of the ulnar is then osteotomised, taking care that the bare minimum is resected (Fig. 7.10a, b). Stump stabilization has been described using the distal aspect of the pronator quadrats and tightening of the dorsal capsule using a slip of the ECU.

The literature on the outcomes of a Darrach excision is mixed. Some investigators have published good outcomes, stressing on the importance of good technique. Other studies have shown high failure rates, with few options for salvage. Common complications include impingement and stump instability, as well as ulnar drift of the carpus.

Complications of Darrach Procedure include impingement, stump instability and ulnar drift of the carpus.

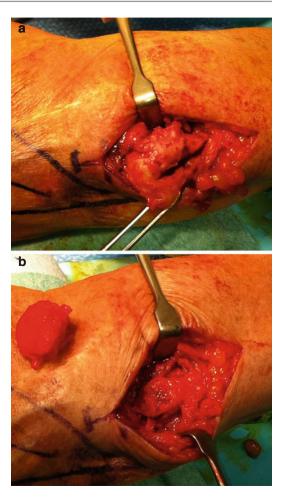


Fig. 7.10 (a, b) Excision of distal ulna through 5th dorsal compartment

Sauve Kapandji Procedure

This procedure involves an arthrodesis of the head of the ulna to the radius, followed by an excision of a segment of the distal ulnar shaft to allow prono-supination (Fig. 7.11) [108]. The procedure is performed through a similar approach to a Darrach's excision and following exposure of the DRUJ articular cartilage from the sigmoid notch and ulnar head is removed and compression screws are passed across the DRUJ, keeping the ulnar variance at neutral. A section of the ulna, usually 8–10 mm can then be removed proximal to the fusion site. Stump stabilization can be achieved using a strip of either extensor or flexor carpi ulnaris. Several studies report excellent results with the Sauvé -Kapandji procedure [109, 110]. However,



Fig. 7.11 Plain radiograph demonstrating Sauve Kapandji procedure

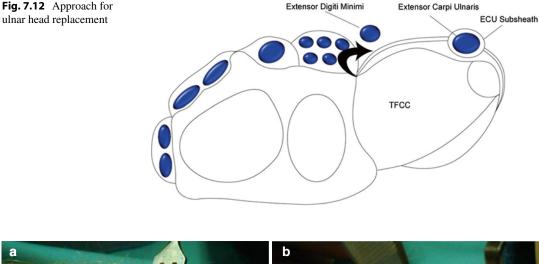
other investigators have shown little superiority on radiographic assessment [111, 112]. Complications include proximal ulnar stump instability and convergence, as well as non-union of the fusion [113, 114]. The treatment of a failed Sauvé- Kapandji procedure can be managed with either further proximal stump resection or custom ulnar head arthroplasty [115].

Ulnar Head Arthroplasty

Several DRUJ arthroplasty systems are available. These include constrained and unconstrained ulnar head designs. Additionally, hemi-head and complete ulnar head replacements are also available. An ulnar head arthroplasty has the theoretical advantage of providing stability to the forearm, with the elimination of convergence and stump instability. Sigmoid notch implants for DRUJ implant arthroplasty have been described. However, long term results are awaited [116].

The surgical approach is through the fifth compartment, taking care to elevate thick soft tissue flaps in order to facilitate a firm dorsal repair following the replacement (Fig. 7.12). The ECU sub sheath should be protected at all times. In a hemi-arthroplasty a jigging system is used to determine the amount of distal ulnar resection and a trial stem is implanted after broaching the proximal ulna. Most investigators comment on the role of local tissues, such as the ECU subsheath and the dorsal capsule, to ensure stability in these procedures (Fig. 7.13).

A retrospective review was conducted at Wrightington of 57 patients who underwent ulnar



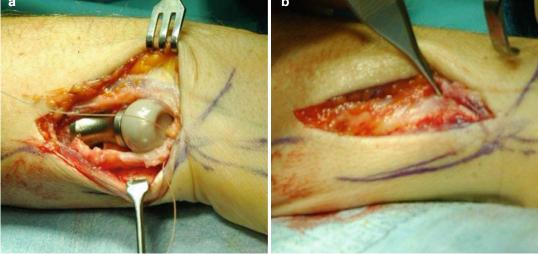


Fig. 7.13 A good repair of the dorsal structures ensures stability following ulnar head replacement. (a) distal ulna showing sharp osteophytes, (b) resected distal ulna

head replacement for recalcitrant DRUJ problems at a mean follow up of 4.4 years, with an average patient age of 54 years. Patients had an average of three prior operations. The mean visual analogue score for pain improved from 7.9 to 3.9. It was reported that 70 % felt that they had a good to excellent result and 67 % of the flexion extension range and 80 % of the prono-supination range was preserved, compared with the opposite side.

Resorption of the distal ulna was seen in 24 patients without any clinical consequence (Fig. 7.14). Six cases had radial scalloping, three of which were progressive. Five prostheses had

been removed and two implants were radiologically loose. One implant fractured and was subsequently removed. Six patients had surgical exploration of ECU for persistent pain.

Revision surgery, following a failed ulnar head replacement, is difficult. One option available is the creation of a one-bone forearm (Fig. 7.15). Cooney et al. reported satisfactory results at 2 years follow-up. Although early outcomes have been encouraging, numbers are few and a longer follow-up will be useful in assessing the role of ulna head arthroplasties in rheumatoid arthritis [117–119].



Fig. 7.14 Complications of ulnar head replacement Scalloping Breakage Resorption



Fig. 7.15 Salvage following a failed ulnar arthroplasty is difficult and may involve the creation of a one bone forearm

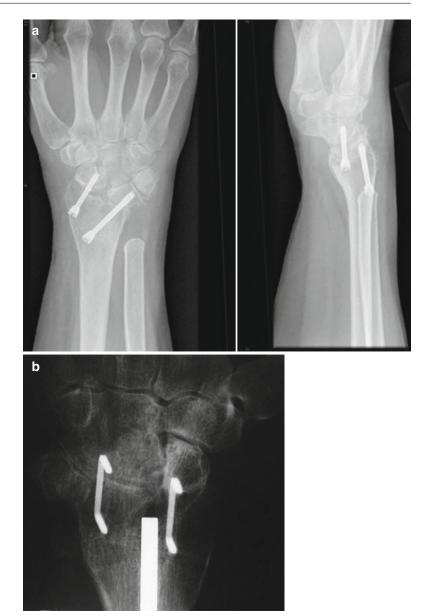


Fig. 7.16 (a) Radioscapholunate fusion using differential pitch screws (b) Partial wrist arthrodesis using staples

Partial Wrist Arthrodesis

In rheumatoid arthritis the midcarpal joint is usually affected later and partial fusion at the radiocarpal level helps to preserve some range of motion. Commonly performed procedures include radiolunate fusions and radio-scapholunate arthrodeses. Midcarpal arthrosis, or rapidly progressive inflammatory changes are contraindications.

The approach is through a standard dorsal approach, with removal of inflamed synovium and

diseased cartilage with approximation of the bone ends. It is crucial to reduce the lunate to the radius in a neutral position, as extension will limit wrist flexion. It is also important to recognize patients with long standing deformities and collapse who do not respond well to an overcorrection, due to exacerbation of the arthritic process [120, 121]. Fixation can be performed using differential pitch screws (Fig. 7.16), K-wires or staples [122, 123]. Bone grafting is recommended and may be



Fig. 7.17 (a) Wrist fusion in rheumatoid arthritis using a Stanley pin (b) Fusion using plates

harvested from the distal radius or the iliac crest. Outcomes tend to be good. Complications include non-union and development of midcarpal arthritis. Tendon attrition and rupture can occur, as well as painful metalwork that may require removal.

Total Wrist Arthrodesis

This is indicated in patients with pan-carpal disease, or in those wrists with significant bone destruction incapable of taking the stems of a total joint replacement. A trial of fusion can be given to the patient by wearing a wrist splint preoperatively, as a total wrist arthrodesis severely limits motion. It is important that the patient is aware of the limitations that a fusion will impose. The ideal position of fusion is slight extension and ulnar deviation and should be discussed with the patient [124]. A fusion in a neutral position will make prono-supination easier [125]. In bilateral disease, one should fuse the dominant side in limited extension and the non-dominant side in neutral or slight flexion [92]. The approach is once again through a dorsal longitudinal incision and the wrist joint is exposed through the retinaculum, between the third and fourth compartment. The carpus is exposed through a distally based flap and a synovectomy is performed. The minimum number of joints included in a wrist arthrodesis includes the radiolunate, radioscaphoid scaphocapitate, capitolunate and capitometacarpal joints. Bone graft from the iliac crest, the distal radius or a resected ulna can be used to supplement the fusion. Contoured dorsal wrist fusion plates are available and are designed to provide some extension and ulnar deviation at the wrist joint. In patients with severe bone loss the Stanley pin can be used through the head of the third metacarpal into the carpus, which is morselized into the distal radius (Fig. 7.17). These pins are counter-sunk to avoid them backing out.

Wrist arthrodesis is a safe and reliable technique. Intramedullary pin fixation seemed to have a higher rate of complications, when compared with fusion plates [126, 127]. However, another paper using a Rush Nail (Mannerfelt technique) demonstrated a similar reduction in pain and functional improvement as plate fixation [124]. Complications include, non-union (usually at the level of the carpometacarpal joint), prominent painful plates and screws causing tendon irritation and rarely attrition ruptures.

Total Wrist Arthroplasty

The primary advantage of total wrist arthroplasty (TWA) is the ability to preserve a functional arc of wrist motion. Generally, this is considered a higherrisk, higher reward procedure. Swanson developed the first generation of wrist implants in 1967. Principally it was a larger version of earlier metacarpal joint replacements, which were also developed by Swanson. It only acts as a spacer device and is therefore sometimes termed an 'Interposition Arthroplasty' rather than a true TWA.

Volz® and Meuli® implants were both developed in the 1970s, with articulating bearing for joint motion, which reduces the implant stresses. Meuli® was the first true TWA, with the design based on ball and socket configuration similar to a hip joint arthroplasty and represent a second generation of wrist implants.

The third generation of implants placed greater emphasis on the correct centre of rotation. These included the Biaxial Wrist, Trispherical® implant and the Universal. They were available in left and right versions, in order to achieve correct placement of the axis of rotation. However, due to problems with loosening of the carpal component these were discontinued.

The most recent designs include screw fixation into the carpus to improve distal carpal fixation. The newer designs also have superior wrist kinematics and allow minimal bone resection that helps to maintain wrist length, thus allowing for better soft tissue balancing. These newer designs appear to resurface the radial side of the joint and are thus more precise and technically demanding.

The Universal[®] Wrist Implant was designed by Menon in 1988 and is an anatomical implant that has better load transfer from the carpus to the radius. The carpal component is made of titanium

and it has an ovoid shaped base to match the cut surface of the carpal bones. The base plate had three screw holes. The centre was for a 6.5 mm diameter for screw insertion in to capitate and possible extension into third metacarpal, if required. It was primarily fixed in the capitate, rather than the third metacarpal as compared to other implants. There were two 4.5 mm screw holes for additional fixation into the carpal bones. These support the first and fifth rays to prevent proximal migration of the columns. The procedure involves intercarpal fusion, which provides a solid bony support for the carpal plate. Menon claimed this should improve longevity. Carpal height could also be restored by using corticocancellous bone graft.

The Universal® implant was modified in the early quarter of the last decade to the Universal-2® wrist implant (Fig. 7.18). The carpal component now has a peg in the centre of the base plate for fixation to the capitate and 3rd metacarpal. The side holes have been maintained, which also allowed screw insertion at variable angles. The articular surface was changed from toroidal to ellipsoid, which reduced the risk of point loading, therefore decreasing the possibility of early wear and loosening. The surface-area of articular tray of the radial component was increased to provide better joint capture. Porus coating was introduced on the carpal component, at the base and peg and on the proximal portion of the stem of the radial component to encourage osteointegration.

The procedure is performed through a dorsal approach and the joint is exposed using T or H shaped flaps. Using radial and carpal jigs, appropriate cuts are made in the distal radius and the carpus to center the prosthesis accurately using intra-operative fluoroscopy to enhance accuracy of placement.

Murphy et al. and other investigators have shown that a successful TWA is better tolerated than an arthrodesis [128]. A cost-utility analysis also suggests that arthroplasty and arthrodesis are cost effective and that a total wrist arthroplasty is only minimally more costly than an arthrodesis [129]. The investigators conclude that arthroplasty is worth considering and is not as costprohibitive as previously thought.



Fig. 7.18 Universal two total wrist replacement arthroplasty

Early follow-up studies of total wrist arthroplasty have shown that patient satisfaction is high and the outcomes are encouraging [130]. However, in a recent publication from the same unit [131], results for the Universal wrist prosthesis, at a minimum of 5 years of follow-up, include a high rate of failure, most often because of carpal component loosening, resulting in revision of ten (50 %) of 20 wrists at the time of the latest follow-up. In a series of 21 consecutive patients, Ferres et al. [132] had no major complications at 5 year follow-up. Their series included patients with Kienbock disease, degenerative arthrosis and chondrocalcinosis. Strunk and Bracker [133], in their review of 41 implants, had a failure rate of 15 % at a 5.3 years.

In a retrospective review of a series from Wrightington Hospital of 73 wrist replacements post-operative pain relief was achieved in 72 % within 6 months and patient satisfaction was 86 %. Movements were preserved with mean dorsiflexion of 23° and palmar flexion of 21°. An interim study of 34 patients showed VAS pain score improved from 5.4 to 1.7 and a DASH score improvement of 14 points. The complications included joint stiffness (11 %, n=10), wrist pain (9 %, n=8) and 2 cases of superficial infection (2.3 %). There was one intra-operative fracture of the distal radius which was successfully managed with a period of immobilization in plaster. Major complications were revision of wrist arthroplasty in 4.7 % (n=4), 3.5 % salvage arthrodesis (n=3) and one patient (1.1 %)required removal of prosthesis, due to chronic synovitis.

Further long term prospective studies are required to best ascertain the role of total wrist arthroplasty in the management of rheumatoid arthritis of the wrist.

Rheumatoid Hand Therapy

Introduction

Although hand structures are frequently affected by rheumatoid arthritis, the hand therapist should not view them in isolation. In addition to a detailed knowledge of hand anatomy, the therapist needs to appreciate the changing nature and varied course of the disease, as this is what dictates the direction of therapeutic emphasis. Therapeutic evaluation and treatment needs to reflect that this disease affects multiple joints and organs and has not just a physical impact on the individual, but also social, economic and psychological consequences for the individual and their family.

In addition to a detailed knowledge of hand anatomy, the therapist needs to appreciate the changing nature and varied course of the disease, as this is what dictates the direction of therapeutic emphasis. A multidisciplinary approach, with medical management at its core, is essential [134]. Individuals should be encouraged and empowered to have input into the decision making processes that determine treatments, as treatment plans that are relevant to individual needs and personal beliefs will increase compliance with suggested treatment and improve outcomes [135].

Therapeutic interventions can be divided into treatments used for conservative management of rheumatoid arthritis and those associated with rehabilitation following a specific surgical procedure. The therapeutic emphasis will be dictated by the stage of the disease and the individuals specific needs, with the overall aim of minimising functional impairment for as long as possible.

Conservative Treatment

Individual treatments are often multifactoral in their aims, which are:-

- 1. Relieve pain.
- 2. Maintain/ increase muscle strength and endurance.
- 3. Maintain/ increase joint range of movement.
- 4. Prevent deformity.
- 5. Educate the patient and their family about the disease, its effects and management.
- 6. Assist with emotional adjustment to a chronic progressive disease.
- 7. Maintain/ increase functional ability.

These aims are achieved by means of specific treatment modalities, which are used throughout the course of the disease.

 Treatment commences with evaluation. Evaluation is usually a global assessment of function, as in the Health Assessment Questionnaire [136] or the Arthritis Impact Measurement Scale [137], but the SODA [138] assessment is a useful tool for assessing function in the rheumatoid hand and is also sensitive to changes brought about by treatment, surgery or just the passage of time [68]. Objective assessments such as pain, grip strength and range of movement can also be useful as a baseline, against which to measure the effects of treatments.

- 2. Pain relief is at the core of most therapeutic interventions. The experience of pain in rheumatoid arthritis can be variable, the pain of an acutely inflamed joint is often constant and described as an ache, the joint that is no longer inflamed and has suffered severe erosive changes may be pain free or only painful with certain activities or movements. Patient education, joint protection, transcutaneous nerve stimulation, provision of splints and aids, all aim to reduce pain. It is worthy of note to recognise that the perception of pain will vary with the individual, it is influenced not only by disease activity, but by family and piers, employment and social situation, culture and education as well as prior experience. Pain relieving techniques must reflect this pain spectrum.
- 3. The application of cold or heat is a frequently employed technique. Ice is the commonest form of applying cold, flaked ice in the hospital environment or frozen peas at home. Cold produces physiological effects on the nervous system and local circulation, alternating vasoconstriction and vasodilatation eliminates the toxins produced by inflammation and thus reduces pain as well as acting as a powerful stimulus that reduces muscle spasm [139]. The application of cold is particularly beneficial during periods of acute inflammation, where it can be used twice a day if necessary, but is contraindicated when raynauds phenomenon or vasculitis are present. Heat is contraindicated for the acutely inflamed joint, as it increases inflammation, but its sedative effect on sensory nerve endings is effective in the sub acute and chronic phases of rheumatoid arthritis, where muscle spasm is frequently the source of pain. In the hospital environment heat is often applied by wax treatment or infrared lamps, at home a hot water bottle or microwave wheat pack is recommended. Heat is particularly effective prior to exercise, as collagen is more extensible at higher temperatures [140].
- Exercise in the rheumatoid hand should never produce pain during or after its performance. If pain is experienced, exercise should be

reduced or ceased completely. For the rheumatoid hand, exercise aims to prevent contractures, maintain range of movement in the joints as well as to encourage differential tendon glides and to prevent deformity. The amount of exercise will be different, according to the phase of the disease:-

- (a) Acute phase, minimal repetitions only, as the aim is to maintain joint range of movement.
- (b) Sub-acute phase, repetitions are increased and light resistance is introduced to compensate for the loss of grip strength, brought on through pain and disuse of acute inflammation.
- (c) Chronic phase, functional exercise is encouraged, as well as an increase in specific exercises as indicated by deformity and range of movement.

The most commonly taught exercises include: wrist flexion/extension and radial/ ulnar deviation, as well as pro and supination to maintain ROM and alignment, finger bridge (bridging of the hand with the digits straight) for intrinsic strengthening, finger walking (radial deviation exercise for the MCP joints to strengthen interossei and prevent ulnar deviation), fist formation (finger flexion involving MCP, PIP and DIP joints to maintain ROM and discourage deformity), finger lift (full extension of MCP, PIP and DIP joints to maintain ROM and prevent contracture) and finger hook (flexion of the PIP and DIP joints with the MCP joints in full extension to prevent intrinsic tightness).

5. The use of joint protection and energy conservation techniques for the rheumatoid hand are particularly relevant, as the hand is the tool that manipulates our environment. They aim to maintain joint integrity and muscle strength, as well as reduce pain and fatigue [141]. In order to be effective, joint protection techniques need to be incorporated into daily life, even on good days. Therefore, the individual needs knowledge of how articular functions can be maintained and improved and joint protection is therefore generally taught as part of a long term

education programme. The techniques are: to maintain joint range and strength through functional activity and specific exercise; to respect pain and stop an activity before pain commences; to balance rest and work, in order to improve overall endurance for a task, as well as to combat a systemic disease; to reduce the effort required to perform a task by the use of work simplification; adaptive techniques and appropriate aids, such as jar openers and sharp kitchen tools or enlarged handles; and to avoid positions of deformity and reduce internal and external deforming forces. In the hand, this can be illustrated by the MCP joints, where several factors contribute to the development of ulnar deviation and volar subluxation. One of them is functional use of the hand. All functional tasks that involve MCP joint flexion and pinch magnify ulnar force up to three times and for the joint that is already weakened by the internal forces of inflammation, this is potentially deforming. Using less deforming grips, or enlarging the handles of tools, can significantly reduce external forces and should be encouraged throughout the course of the disease, as well as following reconstructive surgery to the joints. For example, to spread the strain over several joints, or to use a larger joint to perform a task is easily achieved; for instance opening a heavy door with the pelvis or lifting objects with the forearm and the palms of the hand rather than the fingers, to avoid prolonged static positioning in order to reduce joint compression and positional stress to weakened structures, a change of position every 20 min will avoid the stiffness and pain associated with static positions. The use of assistive equipment is also recommended, not just to compensate for deformity, but prior to its development. Assistive equipment that increases the diameter of grip, or increases leverage, is particularly useful for the hand.

6. Splinting is a technique frequently used for the treatment of the rheumatoid hand, but before any splint is provided, there needs to be a sound clinical reason for their provision. To this end, splinting can be subdivided into therapeutic, prophylactic, functional and post-operative.

Therapeutic splinting is most often used to immobilise a joint during periods of inflammation. Supporting a joint allows the muscles surrounding the joint to relax and pain associated with movement is eliminated, bringing about a local reduction in inflammation [142]. The wrist/hand resting splint is commonly used in the acute phase of rheumatoid arthritis, but other small joints can also be successfully treated through a short period of immobilisation. There is no empirical evidence to support prophylactic splinting, but clinical reasoning suggests that maintaining optimal alignment during function should eliminate deforming forces and the "knock on" effect of deformity. An example of a prophylactic splint is a wrist support worn for activity. The splint maintains the alignment of extensor carpi ulnaris and discourages supination of the carpus, by eliminating radially deforming forces. This in turn discourages the development of MCP joint ulnar deviation. Stabilising joints and reducing pain during activity is what functional splinting aims to do, many sufferers of this disease have incorporated a wrist brace into their daily routine, as it can significantly improve their function [143]. The use of postoperative splinting aims to maintain/protect surgically achieved alignment and mobility, as well as to prevent adhesions and assist with strengthening. As would be expected, there is a huge variety of postoperative splints, since each splint is individually made to suit the individuals needs and particular surgical programme. Some splints are designed to immobilise, for instance, following a wrist arthrodesis, others allow movement in a prescribed arc or plane, such as following MCP joint reconstruction. Other splints may be used to facilitate movement, for instance following flexor tenosynovectomy, blocking mobile MCP joints directs movement to stiff PIP joints.

Splinting the rheumatoid hand is a potentially dangerous modality, not least because there are often multiple instabilities, stiffness, fragile skin and already diminished hand function. Due consideration and care should be given to a splints design and construction and their effectiveness and usefulness should be reviewed on a regular basis. Sometimes not providing a splint is the right treatment choice, particularly if it will exacerbate other problems, interfere with function or have a negative affect on the individual's morale.

Postoperative Treatment

Possibly the most valuable treatment a hand therapist can contribute to the surgical team is a detailed assessment of the rheumatoid hand prior to surgery. Reducing pain, providing stability, minimising stiffness and correcting deformity should improve the function of the hand, providing that surgery is appropriately targeted. When so many joints and soft tissue structures are involved, targeting the structure that is contributing most to functional deficit can be difficult and time consuming to establish. A pre-operative assessment can prioritise functional difficulties for the patient and highlight the anatomical structures that are causing them for the surgeon. By breaking an activity down into its component parts, it is usually possible to establish at which point in the chain the difficulty arises and what is causing it. For instance, the task of feeding has a number of components to it, picking up a utensil, applying pressure to pick up or cut the food, getting the food to the face and placing the utensil in the mouth. To do this task, the individual requires dexterity in the fingers, stability and strength in the wrist, elbow extension to reach the plate and flexion to reach the face, as well as supination at the distal radioulnar joint to place the utensil in the mouth. Targeting surgery at deformed MCP joints, when the reason the person can't feed themselves properly is because they cannot supinate sufficiently, will not resolve the difficulty, whilst targeting the DRUJ may. In addition to identifying functional difficulties and their causes, the experienced hand therapist should be able to evaluate if the expected outcome of a given procedure will bring about a functional change for that person, help the individual decide if they wish to proceed with the surgery and prepare them for the demands of rehabilitation following the operation.

By breaking an activity down into its component parts, it is usually possible to establish at which point in the chain the difficulty arises and what is causing it.

As well as functional factors, there are medical and biomechanical factors that need consideration prior to surgery. Intact tendon mechanisms, as well as the motor power to use them, are essential for hand function and for this reason, tendon and nerve integrity is a surgical priority. As stated previously, when considering joint surgery, the usual practice is to attend to lower limb joints before upper limb so that reconstructed joints can be spared the trauma of walking aids, the one exception to this would be for a wrist fusion, as a pain free and stable wrist can be a bonus when using walking aids and recovering from lower limb surgery. Subluxated MCP joints, in conjunction with stiff swan-neck deformity, require MCP joint subluxation to be corrected first, otherwise the swan-neck will recur. MCP disease, in conjunction with gross PIP joint flexion contractures, requires the PIP joint deformity to be corrected first, since this deformity predisposes the MCP joint towards hyperextension, making MCP joint flexion difficult to achieve following MCP joint surgery. Extensor tendon rupture in conjunction with MCP disease requires the MCP joints to be corrected first, otherwise it is difficult to accurately tension the tendon repair and the outcome will be compromised. Flexor tendon disease, in conjunction with MCP joint disease, requires the flexor tendons integrity to be preserved and the tendons mobile. It may, however, be possible to rectify both at once, by flexor synovectomy and MCP joint reconstruction, but this subjects the patient to surgery on the dorsal and palmar surface and is a difficult postoperative regime for them to follow [144]. Thumb surgery is frequently performed at the same time as other procedures, but if this is not possible then finger surgery should be performed first, so that the thumb can be accurately positioned in opposition to the fingers.

When considering joint surgery, the usual practice is to attend to lower limb joints before upper limb so that reconstructed joints can be spared the trauma of walking aids, the one exception to this would be for a wrist fusion, as a pain free and stable wrist can be a bonus when using walking aids and recovering from lower limb surgery.

In summary, careful assessment is required before surgery, of the rheumatoid hand, is embarked upon. Identification of the anatomical structures contributing most to functional deficit is essential, so that surgery can be properly targeted and the patient needs to be informed what the expected outcome of the procedure will be and its effect upon them as individuals. They also need to be prepared for what can be a prolonged rehabilitation programme.

References

- 1. Jarus T, Poremba R. Hand function evaluation: a factor analysis study. Am J Occup Ther. 1993;47:439–43.
- Symmons D, Turner G, Webb R, Asten P, Barrett E, Lunt M, et al. The prevalence of rheumatoid arthritis in the United Kingdom: new estimates for a new century. Rheumatology (Oxford). 2002;41:793–800.
- Symmons DP, Barrett EM, Bankhead CR, Scott DG, Silman AJ. The incidence of rheumatoid arthritis in the United Kingdom: results from the Norfolk Arthritis Register. Br J Rheumatol. 1994;33:735–9.
- Arnett FC, Edworthy SM, Bloch DA, McShane DJ, Fries JF, Cooper NS, et al. The American Rheumatism Association 1987 revised criteria for the classification of rheumatoid arthritis. Arthritis Rheum. 1988;31:315–24.
- Schoels M, Bombardier C, Aletaha D. Diagnostic and prognostic value of antibodies and soluble biomarkers in undifferentiated peripheral inflammatory arthritis: a systematic review. J Rheumatol Suppl. 2011;87:20–5.
- Aletaha D, Neogi T, Silman AJ, Funovits J, Felson DT, Bingham 3rd CO, et al. 2010 rheumatoid arthritis classification criteria: an American College of Rheumatology/ European League Against Rheumatism collaborative initiative. Ann Rheum Dis. 2010;69:1580–8.
- Alderman AK, Chung KC, Kim HM, Fox DA, Ubel PA. Effectiveness of rheumatoid hand surgery: contrasting perceptions of hand surgeons and

rheumatologists. J Hand Surg Am. 2003;28:3–11; discussion 12–13.

- 8. Souter WA. Planning treatment of the rheumatoid hand. Hand. 1979;11:3–16.
- Chung KC, Kotsis SV, Kim HM, Burke FD, Wilgis EF. Reasons why rheumatoid arthritis patients seek surgical treatment for hand deformities. J Hand Surg Am. 2006;31:289–94.
- da Silva JA, Ramiro S, Pedro S, Rodrigues A, Vasconcelos JC, Benito-Garcia E. Patients- and physicians- priorities for improvement. The case of rheumatic diseases. Acta Reumatol Port. 2010;35: 192–9.
- Millender LH, Nalebuff EA. Evaluation and treatment of early rheumatoid hand involvement. Orthop Clin North Am. 1975;6:697–708.
- Steinbrocker O, Traeger CH, Batterman RC. Therapeutic criteria in rheumatoid arthritis. J Am Med Assoc. 1949;140:659–62.
- Sharp JT, Lidsky MD, Collins LC, Moreland J.Methods of scoring the progression of radiologic changes in rheumatoid arthritis. Correlation of radiologic, clinical and laboratory abnormalities. Arthritis Rheum. 1971; 14:706–20.
- Larsen A, Dale K, Eek M. Radiographic evaluation of rheumatoid arthritis and related conditions by standard reference films. Acta Radiol Diagn (Stockh). 1977;18:481–91.
- Sharp JT. Radiographic evaluation of the course of articular disease. Clin Rheum Dis. 1983;9:541–57.
- Rau R, Herborn G. A modified version of Larsen's scoring method to assess radiologic changes in rheumatoid arthritis. J Rheumatol. 1995;22: 1976–82.
- Larsen A. How to apply Larsen score in evaluating radiographs of rheumatoid arthritis in long-term studies. J Rheumatol. 1995;22:1974–5.
- Kaarela K, Kautiainen H. Continuous progression of radiological destruction in seropositive rheumatoid arthritis. J Rheumatol. 1997;24:1285–7.
- Kirwan J, Byron M, Watt I. The relationship between soft tissue swelling, joint space narrowing and erosive damage in hand X-rays of patients with rheumatoid arthritis. Rheumatology (Oxford). 2001;40: 297–301.
- Smolen JS, van der Heijde DM, Aletaha D, Xu S, Han J, Baker D, et al. Progression of radiographic joint damage in rheumatoid arthritis: independence of erosions and joint space narrowing. Ann Rheum Dis. 2009;68:1535–40.
- Weidekamm C, Koller M, Weber M, Kainberger F. Diagnostic value of high-resolution B-mode and doppler sonography for imaging of hand and finger joints in rheumatoid arthritis. Arthritis Rheum. 2003;48:325–33.
- 22. Solau-Gervais E, Legrand JL, Cortet B, Duquesnoy B, Flipo RM. Magnetic resonance imaging of the hand for the diagnosis of rheumatoid arthritis in the absence of anti-cyclic citrullinated peptide antibodies: a prospective study. J Rheumatol. 2006;33:1760–5.

- 23. McEwen C. Multicenter evaluation of synovectomy in the treatment of rheumatoid arthritis. Report of results at the end of five years. J Rheumatol. 1988;15:765–9.
- Wei N, Delauter SK, Erlichman MS, Rozmaryn LM, Beard SJ, Henry DL. Arthroscopic synovectomy of the metacarpophalangeal joint in refractory rheumatoid arthritis: a technique. Arthroscopy. 1999;15: 265–8.
- Ferlic DC. Rheumatoid flexor tenosynovitis and rupture. Hand Clin. 1996;12:561–72.
- Vaughan-Jackson OJ. Rupture of extensor tendons by attrition at the inferior radio-ulnar joint; report of two cases. J Bone Joint Surg Br. 1948;30B:528–30.
- Sivakumar B, Akhavani MA, Winlove CP, Taylor PC, Paleolog EM, Kang N. Synovial hypoxia as a cause of tendon rupture in rheumatoid arthritis. J Hand Surg Am. 2008;33:49–58.
- Ryu J, Saito S, Honda T, Yamamoto K. Risk factors and prophylactic tenosynovectomy for extensor tendon ruptures in the rheumatoid hand. J Hand Surg Br. 1998;23:658–61.
- Freiberg RA, Weinstein A. The scallop sign and spontaneous rupture of finger extensor tendons in rheumatoid arthritics. Clin Orthop Relat Res. 1972;83:128–30.
- Millender LH, Nalebuff EA, Albin R, Ream JR, Gordon M. Dorsal tenosynovectomy and tendon transfer in the rheumatoid hand. J Bone Joint Surg Am. 1974;56:601–10.
- Moore JR, Weiland AJ, Valdata L. Tendon ruptures in the rheumatoid hand: analysis of treatment and functional results in 60 patients. J Hand Surg Am. 1987;12:9–14.
- Brown FE, Brown ML. Long-term results after tenosynovectomy to treat the rheumatoid hand. J Hand Surg Am. 1988;13:704–8.
- Straub LR, Ranawat CS. The wrist in rheumatoid arthritis. Surgical treatment and results. J Bone Joint Surg Am. 1969;51:1–20.
- Nalebuff EA. Surgical treatment of tendon rupture in the rheumatoid hand. Surg Clin North Am. 1969;49: 811–22.
- Vaughan-Jackson OJ. What can be done for the deformed rheumatoid hand? Postgrad Med J. 1964; 40:280–6.
- Straub LR, Wilson Jr EH. Spontaneous rupture of extensor tendons in the hand associated with rheumatoid arthritis. J Bone Joint Surg Am. 1956;38-A:1208–17; passim.
- Ertel AN, Millender LH, Nalebuff E, McKay D, Leslie B. Flexor tendon ruptures in patients with rheumatoid arthritis. J Hand Surg Am. 1988;13:860–6.
- Mannerfelt L, Norman O. Attrition ruptures of flexor tendons in rheumatoid arthritis caused by bony spurs in the carpal tunnel. A clinical and radiological study. J Bone Joint Surg Br. 1969;51:270–7.
- Harrison S, Swannell AJ, Ansell BM. Repair of extensor pollicis longus using extensor pollicis brevis in rheumatoid arthritis. Ann Rheum Dis. 1972; 31:490–2.

- 40. Bora Jr FW, Osterman AL, Thomas VJ, Maitin EC, Polineni S. The treatment of ruptures of multiple extensor tendons at wrist level by a free tendon graft in the rheumatoid patient. J Hand Surg Am. 1987; 12:1038–40.
- 41. Mountney J, Blundell CM, McArthur P, Stanley D. Free tendon interposition grafting for the repair of ruptured extensor tendons in the rheumatoid hand. A clinical and biomechanical assessment. J Hand Surg Br. 1998;23:662–5.
- Nakamura S, Katsuki M. Tendon grafting for multiple extensor tendon ruptures of fingers in rheumatoid hands. J Hand Surg Br. 2002;27:326–8.
- Boyes JH. Flexor-tendon grafts in the fingers and thumb; an evaluation of end results. J Bone Joint Surg Am. 1950;32-A:489–99; passim.
- Nalebuff EA, Patel MR. Flexor digitorum sublimis transfer for multiple extensor tendon ruptures in rheumatoid arthritis. Plast Reconstr Surg. 1973;52:530–3.
- 45. de Jager LT, Jaffe R, Learmonth ID, Heywood AW. The A1 pulley in rheumatoid flexor tenosynovectomy. To retain or divide? J Hand Surg Br. 1994; 19:202–4.
- McGrath MH, Fleischer A. The subcutaneous rheumatoid nodule. Hand Clin. 1989;5:127–35.
- Fleischer A, McGrath MH. Rheumatoid nodulosis of the hand. J Hand Surg Am. 1984;9:404–11.
- Flatt AE. Some pathomechanics of ulnar drift. Plast Reconstr Surg. 1966;37:295–303.
- 49. Akhavani MA, Paleolog EM, Kang N. Muscle hypoxia in rheumatoid hands: does it play a role in ulnar drift? J Hand Surg Am. 2011;36:677–85.
- Egan M, Brosseau L, Farmer M, Ouimet MA, Rees S, Wells G, et al. Splints/orthoses in the treatment of rheumatoid arthritis. Cochrane Database Syst Rev. 2003;(1):CD004018.
- Adams J, Burridge J, Mullee M, Hammond A, Cooper C. The clinical effectiveness of static resting splints in early rheumatoid arthritis: a randomized controlled trial. Rheumatology (Oxford). 2008;47: 1548–53.
- Ellison MR, Flatt AE, Kelly KJ. Ulnar drift of the fingers in rheumatoid disease. Treatment by crossed intrinsic tendon transfer. J Bone Joint Surg Am. 1971;53:1061–82.
- Oster LH, Blair WF, Steyers CM, Flatt AE. Crossed intrinsic transfer. J Hand Surg Am. 1989;14: 963–71.
- Muzaffar AR, Orenstein HH. Use of the Mitek Mini G2 anchor for crossed intrinsic transfer. Plast Reconstr Surg. 2000;105:469–70.
- Dell PC, Renfree KJ, Below Dell R. Surgical correction of extensor tendon subluxation and ulnar drift in the rheumatoid hand: long-term results. J Hand Surg Br. 2001;26:560–4.
- Brannon EW, Klein G. Experiences with a fingerjoint prosthesis. J Bone Joint Surg Am. 1959;41-A: 87–102.
- 57. Swanson AB. Flexible implant arthroplasty for arthritic finger joints: rationale, technique, and

results of treatment. J Bone Joint Surg Am. 1972;54: 435–55.

- Chung KC, Kotsis SV, Kim HM. A prospective outcomes study of Swanson metacarpophalangeal joint arthroplasty for the rheumatoid hand. J Hand Surg Am. 2004;29:646–53.
- Chung KC, Kotsis SV, Wilgis EF, Fox DA, Regan M, Kim HM, et al. Outcomes of silicone arthroplasty for rheumatoid metacarpophalangeal joints stratified by fingers. J Hand Surg Am. 2009;34:1647–52.
- Goldfarb CA, Stern PJ. Metacarpophalangeal joint arthroplasty in rheumatoid arthritis. A long-term assessment. J Bone Joint Surg Am. 2003;85-A:1869–78.
- Joyce TJ. Analysis of the mechanism of fracture of silicone metacarpophalangeal prostheses. J Hand Surg Eur Vol. 2009;34:18–24.
- 62. Schmidt K, Willburger R, Ossowski A, Miehlke RK. The effect of the additional use of grommets in silicone implant arthroplasty of the metacarpophalangeal joints. J Hand Surg Br. 1999; 24:561–4.
- McArthur PA, Milner RH. A prospective randomized comparison of Sutter and Swanson silastic spacers. J Hand Surg Br. 1998;23:574–7.
- 64. Parkkila T, Belt EA, Hakala M, Kautiainen H, Leppilahti J. Comparison of Swanson and Sutter metacarpophalangeal arthroplasties in patients with rheumatoid arthritis: a prospective and randomized trial. J Hand Surg Am. 2005;30:1276–81.
- 65. Moller K, Sollerman C, Geijer M, Kopylov P, Tagil M. Avanta versus Swanson silicone implants in the MCP joint–a prospective, randomized comparison of 30 patients followed for 2 years. J Hand Surg Br. 2005;30:8–13.
- 66. Pettersson K, Wagnsjo P, Hulin E. NeuFlex compared with Sutter prostheses: a blind, prospective, randomised comparison of Silastic metacarpophalangeal joint prostheses. Scand J Plast Reconstr Surg Hand Surg. 2006;40:284–90.
- Kimani BM, Trail IA, Hearnden A, Delaney R, Nuttall D. Survivorship of the Neuflex silicone implant in MCP joint replacement. J Hand Surg Eur Vol. 2009;34:25–8.
- Delaney R, Trail IA, Nuttall D. A comparative study of outcome between the Neuflex and Swanson metacarpophalangeal joint replacements. J Hand Surg Br. 2005;30:3–7.
- Cook SD, Beckenbaugh RD, Redondo J, Popich LS, Klawitter JJ, Linscheid RL. Long-term follow-up of pyrolytic carbon metacarpophalangeal implants. J Bone Joint Surg Am. 1999;81:635–48.
- Parker WL, Rizzo M, Moran SL, Hormel KB, Beckenbaugh RD. Preliminary results of nonconstrained pyrolytic carbon arthroplasty for metacarpophalangeal joint arthritis. J Hand Surg Am. 2007; 32:1496–505.
- Syed MA, Smith A, Benjamin-Laing H. Pyrocarbon implant fracture after metacarpophalangeal joint arthroplasty: an unusual cause for early revision. J Hand Surg Eur Vol. 2010;35:505–6.

- 72. van der Giesen FJ, Nelissen RG, van Lankveld WJ, Kremers-Selten C, Peeters AJ, Stern EB, et al. Swan neck deformities in rheumatoid arthritis: a qualitative study on the patients' perspectives on hand function problems and finger splints. Musculoskeletal Care. 2010;8:179–88.
- Nalebuff EA. Surgical treatment of finger deformities in the rheumatoid hand. Surg Clin North Am. 1969;49:833–46.
- 74. Adamson GJ, Gellman H, Brumfield Jr RH, Kuschner SH, Lawler JW. Flexible implant resection arthroplasty of the proximal interphalangeal joint in patients with systemic inflammatory arthritis. J Hand Surg Am. 1994;19:378–84.
- Takigawa S, Meletiou S, Sauerbier M, Cooney WP. Long-term assessment of Swanson implant arthroplasty in the proximal interphalangeal joint of the hand. J Hand Surg Am. 2004;29:785–95.
- Stern PJ. Extensor tenotomy: a technique for correction of posttraumatic distal interphalangeal joint hyperextension deformity. J Hand Surg Am. 1989;14:546–9.
- Nalebuff EA, Millender LH. Surgical treatment of the boutonniere deformity in rheumatoid arthritis. Orthop Clin North Am. 1975;6:753–63.
- Silva PG, Lombardi Jr I, Breitschwerdt C, Poli Araujo PM, Natour J. Functional thumb orthosis for type I and II boutonniere deformity on the dominant hand in patients with rheumatoid arthritis: a randomized controlled study. Clin Rehabil. 2008;22:684–9.
- Salgeback S, Eiken O, Haga T. Surgical treatment of the rheumatoid thumb. Special reference to the metacarpophalangeal joint. Scand J Plast Reconstr Surg. 1976;10:153–6.
- Terrono A, Millender L, Nalebuff E. Boutonniere rheumatoid thumb deformity. J Hand Surg Am. 1990;15:999–1003.
- Manueddu CA, Bogoch ER, Hastings DE. Restoration of metacarpophalangeal extension of the thumb in inflammatory arthritis. J Hand Surg Br. 1996;21: 633–9.
- Stanley JK, Smith EJ, Muirhead AG. Arthrodesis of the metacarpo-phalangeal joint of the thumb: a review of 42 cases. J Hand Surg Br. 1989;14:291–3.
- Swanson AB, Herndon JH. Flexible (silicone) implant arthroplasty of the metacarpophalangeal joint of the thumb. J Bone Joint Surg Am. 1977;59: 362–8.
- Figgie MP, Inglis AE, Sobel M, Bohn WW, Fisher DA. Metacarpal-phalangeal joint arthroplasty of the rheumatoid thumb. J Hand Surg Am. 1990;15:210–6.
- Tomaino MM, Pellegrini Jr VD, Burton RI. Arthroplasty of the basal joint of the thumb. Long-term follow-up after ligament reconstruction with tendon interposition. J Bone Joint Surg Am. 1995;77:346–55.
- Swanson AB, de Goot Swanson G, Watermeier JJ. Trapezium implant arthroplasty. Long-term evaluation of 150 cases. J Hand Surg Am. 1981;6: 125–41.

- Creighton Jr JJ, Steichen JB, Strickland JW. Long-term evaluation of Silastic trapezial arthroplasty in patients with osteoarthritis. J Hand Surg Am. 1991;16: 510–9.
- Bezwada HP, Sauer ST, Hankins ST, Webber JB. Long-term results of trapeziometacarpal silicone arthroplasty. J Hand Surg Am. 2002;27:409–17.
- Eaton RG. Replacement of the trapezium for arthritis of the basal articulations: a new technique with stabilization by tenodesis. J Bone Joint Surg Am. 1979;61:76–82.
- Schuurman AH, Bos KE. Treatment of volar instability of the metacarpophalangeal joint of the thumb by volar capsulodesis. J Hand Surg Br. 1993;18: 346–9.
- Trieb K. Treatment of the wrist in rheumatoid arthritis. J Hand Surg Am. 2008;33:113–23.
- Rizzo M, Cooney 3rd WP. Current concepts and treatment for the rheumatoid wrist. Hand Clin. 2011;27:57–72.
- Wilson RL, DeVito MC. Extensor tendon problems in rheumatoid arthritis. Hand Clin. 1996;12:551–9.
- Shinoda J, Hashizume H, McCown C, Senda M, Nishida K, Doi T, et al. Carpal tunnel syndrome grading system in rheumatoid arthritis. J Orthop Sci. 2002;7:188–93.
- Hindley CJ, Stanley JK. The rheumatoid wrist: patterns of disease progression. A review of 50 wrists. J Hand Surg Br. 1991;16:275–9.
- Chamay A, Della Santa D, Vilaseca A. Radiolunate arthrodesis. Factor of stability for the rheumatoid wrist. Ann Chir Main. 1983;2:5–17.
- Hodgson SP, Stanley JK, Muirhead A. The Wrightington classification of rheumatoid wrist X-rays: a guide to surgical management. J Hand Surg Br. 1989;14:451–5.
- 98. Simmen BR, Huber H. The wrist joint in chronic polyarthritis–a new classification based on the type of destruction in relation to the natural course and the consequences for surgical therapy. Handchir Mikrochir Plast Chir. 1994;26:182–9.
- 99. Veehof MM, Taal E, Heijnsdijk-Rouwenhorst LM, van de Laar MA. Efficacy of wrist working splints in patients with rheumatoid arthritis: a randomized controlled study. Arthritis Rheum. 2008;59:1698–704.
- Weitoft T, Ronnblom L. Randomised controlled study of postinjection immobilisation after intraarticular glucocorticoid treatment for wrist synovitis. Ann Rheum Dis. 2003;62:1013–5.
- 101. Bliddal H, Terslev L, Qvistgaard E, Konig M, Holm CC, Rogind H, et al. A randomized, controlled study of a single intra-articular injection of etanercept or glucocorticosteroids in patients with rheumatoid arthritis. Scand J Rheumatol. 2006;35:341–5.
- 102. Bliddal H, Terslev L, Qvistgaard E, Recke P, Holm CC, Danneskiold-Samsoe B, et al. Safety of intraarticular injection of etanercept in small-joint arthritis: an uncontrolled, pilot-study with independent imaging assessment. Joint Bone Spine. 2006;73: 714–7.

- 103. Grennan DM, Gray J, Loudon J, Fear S. Methotrexate and early postoperative complications in patients with rheumatoid arthritis undergoing elective orthopaedic surgery. Ann Rheum Dis. 2001;60:214–7.
- 104. Favalli EG, Desiati F, Atzeni F, Sarzi-Puttini P, Caporali R, Pallavicini FB, et al. Serious infections during anti-TNFalpha treatment in rheumatoid arthritis patients. Autoimmun Rev. 2009;8: 266–73.
- 105. Kessler I, Vainio K. Posterior (dorsal) synovectomy for rheumatoid involvement of the hand and wrist. A follow-up study of sixty-six procedures. J Bone Joint Surg Am. 1966;48:1085–94.
- 106. Koka R, D'Arcy JC. Stabilisation of the wrist in rheumatoid disease. J Hand Surg Br. 1989;14: 288–90.
- 107. Millender LH, Nalebuff EA. Preventive surgery tenosynovectomy and synovectomy. Orthop Clin North Am. 1975;6:765–92.
- Kapandji AI. Technical improvement of the Kapandji-Sauve operation, called "Technique III". Ann Chir Main Memb Super. 1998;17:78–86.
- 109. Fujita S, Masada K, Takeuchi E, Yasuda M, Komatsubara Y, Hashimoto H. Modified Sauve-Kapandji procedure for disorders of the distal radioulnar joint in patients with rheumatoid arthritis. Surgical technique. J Bone Joint Surg Am. 2006; 88(Suppl 1 Pt 1):24–8.
- Vincent KA, Szabo RM, Agee JM. The Sauve-Kapandji procedure for reconstruction of the rheumatoid distal radioulnar joint. J Hand Surg Am. 1993;18:978–83.
- 111. George MS, Kiefhaber TR, Stern PJ. The Sauve-Kapandji procedure and the Darrach procedure for distal radio-ulnar joint dysfunction after Colles' fracture. J Hand Surg Br. 2004;29:608–13.
- 112. Kobayashi A, Futami T, Tadano I, Fujita M, Watanabe T, Moriguchi T. Radiographic comparative evaluation of the Sauve-Kapandji procedure and the Darrach procedure for rheumatoid wrist reconstruction. Mod Rheumatol. 2005;15:187–90.
- 113. Daecke W, Martini AK, Schneider S, Streich NA. Amount of ulnar resection is a predictive factor for ulnar instability problems after the Sauve-Kapandji procedure: a retrospective study of 44 patients followed for 1-13 years. Acta Orthop. 2006; 77:290–7.
- 114. Daecke W, Martini AK, Schneider S, Streich NA. Clinical results after Sauve-Kapandji procedure in relation to diagnosis. Unfallchirurg. 2004;107: 1057–64.
- 115. Fernandez DL, Joneschild ES, Abella DM. Treatment of failed Sauve-Kapandji procedures with a spherical ulnar head prosthesis. Clin Orthop Relat Res. 2006;445:100–7.
- Murray PM, Adams JE, Lam J, Osterman AL, Wolfe S. Disorders of the distal radioulnar joint. Instr Course Lect. 2010;59:295–311.
- 117. Willis AA, Berger RA, Cooney 3rd WP. Arthroplasty of the distal radioulnar joint using a new ulnar head

endoprosthesis: preliminary report. J Hand Surg Am. 2007;32:177–89.

- Van Schoonhoven J, Herbert TJ, Fernandez DL, Prommersberger KJ, Krimmer H. Ulnar head prosthesis. Orthopade. 2003;32:809–15.
- 119. van Schoonhoven J, Fernandez DL, Bowers WH, Herbert TJ. Salvage of failed resection arthroplasties of the distal radioulnar joint using a new ulnar head prosthesis. J Hand Surg Am. 2000;25:438–46.
- Borisch N, Lerch K, Grifka J, Haussmann P. Adaptive patterns of the rheumatoid wrist after radiolunate arthrodesis. Z Rheumatol. 2004;63: 326–30.
- Borisch N, Haussmann P. Radiolunate arthrodesis in the rheumatoid wrist: a retrospective clinical and radiological longterm follow-up. J Hand Surg Br. 2002;27:61–72.
- 122. Doets HC, Raven EE. Radiolunate arthrodesis: a procedure for stabilising and preserving mobility in the arthritic wrist. J Bone Joint Surg Br. 1999;81: 1013–6.
- 123. Stanley JK, Boot DA. Radio-lunate arthrodesis. J Hand Surg Br. 1989;14:283–7.
- 124. Toma CD, Machacek P, Bitzan P, Assadian O, Trieb K, Wanivenhaus A. Fusion of the wrist in rheumatoid arthritis: a clinical and functional evaluation of two surgical techniques. J Bone Joint Surg Br. 2007;89:1620–6.
- Clayton ML, Ferlic DC. Arthrodesis of the arthritic wrist. Clin Orthop Relat Res. 1984;187:89–93.
- Howard AC, Stanley D, Getty CJ. Wrist arthrodesis in rheumatoid arthritis. A comparison of two methods of fusion. J Hand Surg Br. 1993;18:377–80.
- 127. Rehak DC, Kasper P, Baratz ME, Hagberg WC, McClain E, Imbriglia JE. A comparison of plate and pin fixation for arthrodesis of the rheumatoid wrist. Orthopedics. 2000;23:43–8.
- Murphy DM, Khoury JG, Imbriglia JE, Adams BD. Comparison of arthroplasty and arthrodesis for the rheumatoid wrist. J Hand Surg Am. 2003;28: 570–6.
- 129. Cavaliere CM, Chung KC. A cost-utility analysis of nonsurgical management, total wrist arthroplasty, and total wrist arthrodesis in rheumatoid arthritis. J Hand Surg Am. 2010;35:379–391 e372.
- Divelbiss BJ, Sollerman C, Adams BD. Early results of the Universal total wrist arthroplasty in rheumatoid arthritis. J Hand Surg Am. 2002;27:195–204.
- 131. Ward CM, Kuhl T, Adams BD. Five to ten-year outcomes of the Universal total wrist arthroplasty in patients with rheumatoid arthritis. J Bone Joint Surg Am. 2011;93:914–9.
- Ferreres A, Lluch A, Del Valle M. Universal total wrist arthroplasty: midterm follow-up study. J Hand Surg Am. 2011;36:967–73.
- Strunk S, Bracker W. Wrist joint arthroplasty: results after 41 prostheses. Handchir Mikrochir Plast Chir. 2009;41:141–7.
- 134. Ahlmen M, Sullivan M, Bjelle A. Team versus nonteam outpatient care in rheumatoid arthritis.

A comprehensive outcome evaluation including an overall health measure. Arthritis Rheum. 1988; 31:471–9.

- 135. Donovan J. Patient education and the consultation: the importance of lay beliefs. Ann Rheum Dis. 1991;50 Suppl 3:418–21.
- 136. Kirwan JR, Reeback JS. Stanford Health Assessment Questionnaire modified to assess disability in British patients with rheumatoid arthritis. Br J Rheumatol. 1986;25:206–9.
- 137. Meenan RF, Mason JH, Anderson JJ, Guccione AA, Kazis LE. AIMS2. The content and properties of a revised and expanded Arthritis Impact Measurement Scales Health Status Questionnaire. Arthritis Rheum. 1992;35:1–10.
- 138. van Lankveld W, van't Pad Bosch P, Bakker J, Terwindt S, Franssen M, van Riel P. Sequential occupational dexterity assessment (SODA): a new test to measure hand disability. J Hand Ther. 1996; 9:27–32.

- 139. Palastanga NP. Heat and cold. In: Pain management by physiotherapy. 2nd ed. Oxford: Butterworth-Heinmann; 1994.
- 140. Lehmann JF, Masock AJ, Warren CG, Koblanski JN. Effect of therapeutic temperatures on tendon extensibility. Arch Phys Med Rehabil. 1970;51: 481–7.
- Cordery JC. Joint protection; a responsibility of the occupational therapist. Am J Occup Ther. 1965;19: 285–94.
- 142. Gault SJ, Spyker MJ. Beneficial effect of immobilization of joints in rheumatoid and related arthritides: a splint study using sequential analysis. Arthritis Rheum. 1969;12:34–44.
- 143. Mercer C, Davis M. A survey of the uses and benefits of prefabricated wrist and thumb supports. Br J Ther Rehabil. 1995;2:599–603.
- 144. Nalebuff EA. The rheumatoid hand. Reflections on metacarpophalangeal arthroplasty. Clin Orthop Relat Res 1984;182:150–9.

Crystalline and Other Arthritides

8

lan A. Trail

Keywords

Psoriasis • Arthritis mutilans • Shish kebab fusion • Scleroderma • Raynaud's • SLE • Gout • Pseudogout • Acute calcification

Introduction

Whilst rheumatoid and osteoarthritis are responsible for the majority of arthritic conditions affecting the hand and wrist, other conditions can sometimes be manifest. These include psoriasis, scleroderma, systemic lupus erythematosis as well as gout, pseudo-gout and acute calcification. These conditions will be considered separately.

Psoriasis

The association between psoriasis and arthritis was first noted by Alibert in (1822) and confirmed in detail by Bourdillon in (1888). Psoriatic arthropathy as a disease entity, however, only became accepted in 1964 (American Rheumatism Association). Subsequently, Moll and Wright

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(1973) classified psoriatic arthropathy of the hand into five sub-groups [1]:

- 1. Classical psoriatic arthritis in which the D.I.P. joints are predominantly involved.
- 2. "Arthritis mutilans": a severe destructive form of arthritis affecting the hands and feet.
- 3. A symmetrical pattern of arthritis indistinguishable from rheumatoid arthritis.
- 4. A symmetrical oligo- (or mono-) articular presentation.
- 5. The predominant feature is ankylosing spondylitis.

Incidence

Several series have reported the incidence and distribution of arthritis affecting the various joints in the hands and wrists (Table 8.1).

In the study by Trail and Stanley (1990) on 56 patients, 30 being male and 26 female, with a mean age of 48.1 years (± 15.4), all but 3 were seronegative and arthritis had been present for a variable period [2]. In the fingers, the PIP joint was the most frequently involved.

Of the 19 patients who were found to have deformities, eight had a flexion contracture

	Martel et al. (1980)	Belsky et al. (1982)	Kapasi et al. (1982)
Number of patients	50	25	30
Joints affected (% of patients)			
Radio-carpal	32	68	35
Inferior radio-ulnar	14		
Finger M.P.	J	100	27.5
P.I.P.	50	88	51
D.I.P.	26	68	51.3
Thumb C.M.C.)	20
M.P.		60	48.3
I.P.			43.3
Nail involvement		J	100
Tenosynovitis		4	

Table 8.1 Frequency of involvement of wrists and hands in psoriatic arthritis according to three previous papers



Fig. 8.1 Flexion contracture of the PIP joint in psoriasis

(Fig. 8.1), seven had a boutonnière and four had swan-neck deformities. Two more were found to be completely dislocated.

At the metacarpophalangeal joint of the fingers, the incidence of involvement is almost the same. Eleven joints were found to be subluxed: five with volar subluxation, two ulnar subluxation and four had both. For the distal interphalangeal joint, clinically this was the least frequently involved, in only four patients having deformities, two a flexion deformity, one an extension deformity and one an ulnar deviation. One joint was found to have ankylosed spontaneously.

In the thumb the metacarpophalangeal joint was more frequently involved, with five patients having deformity; two a flexion contracture and three an abduction deformity. Three others had again spontaneously fused. The interphalangeal joint of the thumbs was the next most frequently involved, with five having deformities, three being flexion contactures and two radially deviated. Finally the least involved joint in the thumb was the carpo-metacarpal joint, with three patients having an adduction deformity and subluxation.

In addition, at the wrist joint, five patients had a prominent lower end of the ulna with a supinated wrist, one of these was bilateral and one had marked radio-ulnar synovitis. Three patients had a swollen wrist and a reduced range of motion; one of these had a flexion contracture



Fig. 8.2 Nail involvement with psoriasis

and a second had symptoms of carpal tunnel syndrome.

Finally, four patients had arthritis mutilans; two however only had involvement of one finger, whilst the others were more widespread. In addition 29 patients (49 %) had nail involvement (Fig. 8.2), 11 patients were found to have clinical evidence of synovitis, five of the extensor tendons and six of the flexor. Indeed one patient had a ruptured extensor pollicis tendon.

Investigations

Plain x-rays of the hands and wrists remain the cornerstone of diagnosis and staging. In 2006 Siannis et al. studied a large study of patients with psoriatic arthritis under the care of the University of Toronto [3]. They concluded that radiological evidence of joint involvement is more often detected before clinical changes are observed. However, signs of clinical inflammation often precede the detection of radiological damage.

In our study, the distribution of positive x-ray findings is set out in Table 8.2. These were matched for age and sex with a similar group of patients attending the Accident and Emergency Department. As can be seen, the commonest affected joints were both the distal (Fig. 8.3) and proximal interphalangeal joints, particularly of the finger. This was followed by the metacarpophalangeal joints of the fingers and thumbs and thereafter the radio-carpal joint (Fig. 8.4).

MRI scans have also been used to investigate psoriatic arthritis. Tehranzadeh et al. (2008) reviewed 23 contrast-enhanced MRI imaging studies of the hands and wrists in 10 patients with this condition [4]. As well as changes to the articular surfaces, they were also able to demonstrate bone marrow abnormalities, tears in the triangular fibrocartilage and articular synovitis in all cases. Added to this wrist soft tissue involvement was seen in 9 out of the 10 scans and 12 out of the 13 hands. Finally compression of the median nerve was seen in 8 of the 10 wrists. Many of these findings were confirmed by Lee et al. in their study on juvenile psoriatic arthritis undertaken again in 2008 [5].

Treatment

The treatment options available for patients with psoriatic involvement of the hand and wrist are similar to those patients who suffer with other inflammatory arthritis, particularly rheumatoid. At the wrist, arthrodesis and arthroplasty are both viable options. At the distal radio-ulnar joint either excision (Darrach's), fusion (Sauve Kapandje, or again joint replacement can be performed. In the hand, at the distal interphalangeal joint the standard treatment would be arthrodesis. At the proximal interphalangeal joint, again arthrodesis is an option particularly if there is significant bone loss or deformity such as boutonnière, swan-neck deformity as well as varus/ valgus angulation. For others however, joint replacement is an increasingly favoured option. Traditionally, hinged silastic implants have been used. However, the newer two piece implants are now being used more frequently, particularly in stable joints with good bone stock. Finally, in patients with little or no articular cartilage damage, various soft tissue procedures similar to the ones used in rheumatoid arthritis can be utilized [6].

Psoriatic X-rav					Controls X-rav			
642.22	Narrowing	Erosions	Expanded	Ankylosis/arthrodesis	Narrowing	Erosions	Expanded	Ankylosis
Radio-carpal	19(33.9)	14(25.0)	I	2(3.6)	5(8.9)	1	I	1
Inferior radio-ulnar	13(23.2)	21(37.5)	I	1	2(3.6)	1(1.8)	I	1
Carpus	12(21.4)	20(35.7)	I	6(10.7)	3(5.4)	1(1.8)	I	1
Finger M.Ps	22(39.3)	26(46.4)	I	1	2(3.6)	I	I	I
PIPs	25(44.6)	27(48.2)	I	1	10(17.9)	2(3.6)	I	1
DIPs	26(46.4)	20(35.7)	9(16.1)	1(1.8)	13(23.2)	5(8.9)	I	I
Thumb CMC	14(25.0)	1	I	1	9(16.1)	6(10.7)	I	1
M.P	20(35.7)	21(37.5)	1	4(7.1)	5(8.9)	2(3.6)	I	1
IP	21(37.5)	14(25.0)	1(1.8)	1(1.8)	10(17.9)	5(8.9)	1	1
Figures in brackets are percentages of patients	ercentages of paties	nts involved						

Table 8.2 Frequency of involvement of wrists and hands in psoriatic and control patients in our series	
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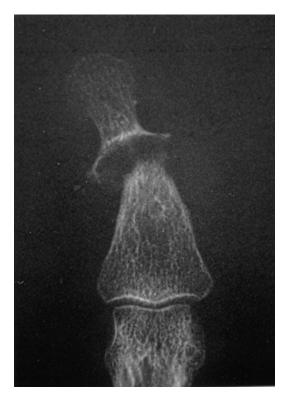


Fig. 8.3 X-ray image of psoriasis at the distal interphalangeal joint



Fig. 8.4 X-ray image of a psoriatic wrist

At the metacarpophalangeal joint, silastic joint replacement is the most common option, although again synovectomy and realignment would be an option in the less advanced cases. For the thumb, arthrodesis of the inter-or metacarpophalangeal joint is the most commonly performed procedure. At the base of the thumb

Table 8.3	Surgical	procedures	performed	on	psoriatic
hands					

Wrist arthrodesis	2
Wrist arthroplasty	1
Excision of ulnar head	1
M.P. replacement	1
P.I.P. replacement	3
P.I.P. arthrodesis	1
P.I.P. joint soft-tissue correction for boutonnière deformity	1
Percutaneous suspensory ligament release at P.I.P. joint	1
D.I.P. arthrodesis	1
Trapezium replacement	1
M.P. arthrodesis (thumb)	1
Flexor tenosynovectomy	4
E.P.L. graft	1
Release trigger finger	1
Total	20

however, a trapeziectomy or joint replacement are again options.

One particular clinical presentation that is however more unique to psoriasis is "arthritis mutilans". The treatment here is more difficult and if necessary takes the form of arthrodesis with the use of interposition bone grafts. Fixation can be undertaken either by a longitudinal k wire in a "shish kebab" fashion or by plate and screws. The number of surgical procedures undertaken in our series are set out in Table 8.3.

Results

In our series, a range of procedures undertaken is given in Table 8.3.

As can be seen the most commonly performed operation was flexor tenosynovecotomy with only one M.C.P. joint replacement in a total of 112 hands. This contrasts sharply with Belsky et al., who had only one tenosynovectomy but 58 metacarpophalangeal joint replacements [7]. In both series, however, the P.I.P. and wrist joints were commonly operated upon. Zangger et al. (2000), reported 71 operations in 43 patients; the commonest performed procedure being distal or proximal interphalangeal joint fusion [8]. The results being reported as good to excellent. Liebovic et al. in 1994, however, reported relatively high non-union rates [9]. Generally, however, the results of most of these procedures are similar to those posted for rheumatoid arthritis.

Clinical Pearl

- Whilst uncommon, psoriatic arthritis often affects the hands, resulting typically in an inflammatory type picture
- The condition is usually symmetrical, involving predominantly the finger, PIP and DIP joints, although all joints can be involved
- Nail involvement is also seen, but is not necessarily related to disease of the DIP joint
- Treatment is similar to other inflammatory conditions, although "mutilans" may require additional bone grafting.

Scleroderma

Systemic sclerosis or scleroderma is a rare connective tissue disorder which can affect the skin but also other end organs. Typically the disease has two patterns, firstly predominantly affecting the skin of the forearms and hands with calcification within the subcutaneous tissues. A more diffuse disease process also involves the skin, but also the major organs including renal, cardiac and pulmonary. Histologically the disease process appears as vasculitis and fibrosis either in the skin or deeper organs. The overall pathogenesis is felt to be immunological, predominantly an over active T-cell response.

Clinical Presentation

The disease is fortunately rare, but appears to be more common in women than men. The age of onset being in the middle years, particularly the fifth and sixth decades. As stated above, the disease can affect multiple end organs. In the hands,



Fig. 8.5 Finger contracture in diffuse cutaneous systemic sclerosis (published with the permission of Professor Ariane Herrick, Hope Hospital, Salford)

however, initially there is pain, swelling and oedema. Progression of the disease leads to painful contractures, usually due to skin tightening (Fig. 8.5) [10]. Later findings include Boutonnière or Swan-neck deformities, osteopenia and bone resorption. Soft tissue calcification is also characteristic [11]. In addition, the majority of patients with scleroderma demonstrate Raynaud's phenomenon. This is manifest by vascular involvement, with subsequent ischemic demarcation (Fig. 8.6), finger ulceration (Fig. 8.7) and ultimately auto-amputation [12]. More recently Low et al. (2009), have demonstrated the usefulness of MRI of the hands in patients with systemic sclerosis, by way of identifying undetected synovitis, erosions, joint effusion and tenosynovitis [13].



Fig. 8.6 Subsequent ischemic demarcation



Fig. 8.7 Digital ulcers in diffuse cutaneous systemic sclerosis (published with the permission of Professor Ariane Herrick, Hope Hospital, Salford)

Treatment

Again the predominant treatment for systemic sclerosis is medical, patients often receiving antiinflammatory agents or low dose cortico-steroids.

Surgical treatment can be divided into modalities, specifically those that attempt to improve circulation to the digit or treat joint involvement, specifically contracture. If the articular surfaces of the joint are well preserved, then release of capsule, collateral ligaments and volar plate are all possible. Followed by appropriate hand therapy, this can improve function [14]. In a large series Melone et al. described their results of 272 operations with generally good outcome [15]. These included various procedures, including arthrodesis and arthroplasty, combined with release of contracture; often healing occurred by secondary attention. For peri-arterial insufficiently both sympathectomy and bypass grafts have been undertaken. Hartzell et al. (2009) reported a series of 28 patients with a minimum follow-up of 23 months and showed that sympathectomy alone can either lead to complete healing or a decrease in ulcer numbers [16]. However, they also felt that this procedure may be of little or no benefit in patients with chronic digital ischaemia or in the presence of severe atherosclerotic disease. Finally, Kwon et al. (2009) reported two patients with severe digital ischaemia and ulceration, who underwent microvascular reconstruction with success [17].

Clinical Pearl

- Presents with ischaemia (Raynaud's) and skin involvement.
- Surgical treatment is twofold, firstly to improve circulation and secondly correct deformity.
- Wound healing is often a problem.

Systemic Lupus Erythematosus (SLE)

Systemic lupus erythematosus (SLE) is an autoimmune disease characterised by the presence of a positive anti-nuclear antibody. The disease process ultimately results in a loss of self-tolerance and as a consequence the production of auto antibodies. The latter results in damage to various end organs. The disease is again more common in females, particularly of child bearing years. Whilst numerous organs can be involved, arthritis is not uncommon, particularly involving the small joints of the hand.

Clinical Presentation

As stated above, the disease is more common in females, particularly of child bearing years. The disease process affects multiple end organs and is characterised by the presence of antinuclear



Fig. 8.8 Vasculitis of the finger in a patient with SLE

antigen (ANA) in the blood stream. In the hand, the disease process can affect the skin, often producing painful finger pulp inflammation. Added to this, patients can present with vasculitis (Fig. 8.8). Thrombosis of the dermal vessels is also sometimes seen [18]. In the finger joints, unlike rheumatoid arthritis, SLE is typically nonerosive. Soft tissue swelling is common, although fusions are rare. As with rheumatoid arthritis, however, ulna deviation of the MCP joint can occur with subluxation and contracture. Finally, tenosynovitis and tendon rupture are also quite common in SLE.

Treatment

Treatment is again predominantly medical and outside the remit of this chapter. Surgery, however, as with rheumatoid arthritis, can be undertaken in extreme cases. Certainly arthrodesis and joint replacement are options.

Clinical Pearl

- Typically seen in young females of child bearing age.
- Diagnosis confirmed by the presence of positive anti-nuclear antibodies.
- Surgical treatment can be undertaken in extreme cases.

Gout

Introduction

Gout is a condition predominantly of middle aged males characterized by intermittent painful swelling of the joints. The inflammation occurs as a result of a reaction to monosodium urate (MSU) crystals. The principal pathogenesis for most patients is an inability to excrete uric acid in sufficient quantities. Secondary causes include an overproduction of uric acid often as a result of dietary indiscretion. Histologically the appearances are of a florid white cell reaction to the presence of MSU crystals. The presence of intracellular, negatively birefringent, needle-shaped crystals being diagnostic.

Clinical Presentation

Whilst not exclusively a disease of middle aged men, the condition is significantly less common in children and females. In addition, it sometimes follows an incidence of trauma to a particular location. Classically patients present with intermittent severe pain and swelling of specific joints. These would typically include the great toe meta-tarsophalangeal joint, but can include the thumb, wrists and elbows amongst other joints. The joint itself being red and swollen and as such is often mistaken for infection. As well as joint involvement, tophi can develop (Fig. 8.9). These are essentially hard swellings predominantly located in the soft tissues. Whilst they can occur acutely and again be mistaken for infection, more commonly they occur in chronic gout. Indeed they may occur in up to 30 % of patients [19].

In addition, tendons or their surrounding synovium can also be involved. This can manifest as tenosynovitis or in chronic cases as a tendon rupture. Added to this, inflammation within the carpal tunnel can lead to median nerve compression. Finally, gout can also affect the wrist joint, not only the radio-carpal joint itself, but also the distal radio-ulna.



Fig. 8.9 Gouty tophi at the distal interphalangeal joint

Investigations

The key to diagnosing gout is to have a high index of clinical suspicion. Certainly in a middle aged male presenting with what externally looks like an infected joint gout should form part of the differential diagnosis. The definitive diagnosis of gout, however, is made by aspiration and microscopy with the detection of MSU crystals. These are needle-shaped and negatively bio-refringent when reviewed under a polarising microscope. Whilst intracellular MSU crystals signify an acute attack, it should be remembered that blood uric acid level may be normal. An x-ray of an acute joint may also be normal, however, in the chronic case various changes can be seen, including the loss of joint space and articular erosions. The latter characterised by punched out or small bite type lesions. Somewhat later, signs of secondary osteoarthritis are seen.

Other investigations including CT and MRI scans are sometimes undertaken. These more clearly demonstrate gouty tophi, particularly in deeper structures such as tendons. Certainly MRI scans allow a better understanding of the distribution of the disease process.

Management

The principal management of gout is medical. During an acute attack the aim is to reduce inflammation and reduce any excess uric acid. This is usually achieved by the administration of nonsteroidal anti-inflammatories.

Prevention is also important, particularly in patients who have frequent attacks. Classically this will involve drugs such as allopurinal or probenecid. Added to this it is important that patients with gout receive appropriate dietary advice.

The role of surgery is more limited. Certainly biopsies or drainage of acute gouty tophi can be undertaken. However usually the acute situation can be managed solely by medical means. However, once the acute condition has settled, surgery may have a role in returning movement and function to the wrist and hand. Procedures would include debridement and releases of the joint, debulking of tophi, release or repair of tendons and finally nerve decompression. In 1958, Larmon and Kurtz reported on their experience in 23 patients [20]. The principal operation was excision or curettage of tophi. However, they also performed carpal tunnel release. Gelberman et al. in 1980, described disruption of the extensor mechanism at the PIP joint, which required surgery by way of tophectomy and reconstruction of the extensor tendon [21]. Similarly Moore and Weiland (1985) described their experience with urate deposition within the extensor and flexor tendons of the wrist and fingers [22]. Operative intervention was required to debulk tophaceous deposits and to improve tendon gliding. Finally Wilczynski et al. (2009) reported their experience of wrist arthroscopy in seven patients with gout [23]. In all wrists they identified diffuse synovitis and crystalline deposits. Added to this there were focal precipitates on the scapholunate and lunotriquetral ligaments but not on the triangular fibrocartilage complex. Scapholunate or lunotriquetral ligament disruption was noted in 6 and 5 wrists, respectively. An early scapholunate advanced collapse pattern of arthritis was present in five patients.

Clinical Pearl

Gout typically affects middle aged males and can follow minor trauma.

- The principle differential diagnosis is acute infection.
- Diagnosis is confirmed by the identification of MSU crystals under a polarising microscope.

Treatment is predominantly medical.

In the long term, however, patients can develop secondary osteoarthritis as well as problems in and around tendons.

Pseudo Gout or Calcium Pyrophosphate Dihdrate Deposition

Calcium pyrophosphate dihdrate (CPPD) is a calcium containing crystal that can again deposit in joints resulting in an acute inflammatory response. Most patients however are elderly, that is over 65, and the condition appears equally in both sexes. Sometimes there is an association with conditions such as hyperparathyroidism, haemachromatosis and amyloidosis etc.

The pathological process is similar to gout in that the crystal is phagocytosed by bipolymorphonuclear leukocytes and this results in an inflammatory response. As a result of this enzymes are released, which can result in damage to the articular surface of the joint.

Presentation

This condition can present in various ways, either as an acute attack of synovitis, chronic arthritis, or indeed simply appear as an x-ray finding. Acute attacks often involve a single joint and take 2–3 weeks to resolve. Typical joints in the upper limb include the metacarpophalangeal, wrist and elbow. However, the disease can also affect the lower limb. The disease itself can be quite painful resulting in swelling and warmth of the affected area.

Investigations

The diagnosis of pseudo gout or chondrocalcinois is often made by plain x-ray, specifically calcific deposits are seen around the joint in ligaments, tendons and capsule. Aspiration of the joint will reveal a raised white cell count in the acute case. Histology will demonstrate the presence of intracellular weakly positive birefringent rod shaped crystals.

As with gout, the majority of cases can be managed by medical means alone. This may involve aspiration followed by an injection of a local anaesthetic and corticosteriod. This often results in a rapid resolution of symptoms. Added to this, other pain relieving modalities, including splintage as well as oral anti-inflammatories may be useful.

Surgical Management

As with gout, surgical management involves the restoration of movement of the particular joint if long term stiffness ensues and if appropriate carpal tunnel release. Ultimately however, patients may develop secondary osteoarthritis, treatment of which is discussed separately in (see Chap. 4).

Clinical Pearl

Often an incidental finding Differential diagnosis again is infection Diagnosis is confirmed by aspiration and microscopy Treatment predominantly medical

Acute Calcification

Acute calcification in the shoulder is well recognised. However acute calcification at other sites is much rarer. In the hand it was first described by Cohen in 1924. In the majority of cases these involve the carpal and metacarpal areas. Only 8 % in a series of 100 cases reviewed by Carroll (1955) were at the proximal interphalangeal joint level [24].

The aetiology of acute classification is still uncertain. Originally it was felt by Moschowitz (1915) that a injury or strain to a particular structure resulted in localised necrosis with a resultant deposition of calcium phosphate and oxalate crystals [25]. These crystals locally irritate the surrounding tissues resulting in acute inflammatory response. However, not all cases have a history of trauma. Secondly pathological studies reveal no evidence of scarring or indeed any acute inflammatory response.

Subsequently Uhthoff (1976) postulated that there was a local transformation of tissue into fibrocartilage [26]. This transformation was probably due to increased hydrostatic pressure and a lowering of the tissue oxygen tension. The chondrocytes present mediating the deposition of calcium crystals. As a consequence they described acute calcification, not as a degenerative but as a reactive process.

Gravanis (1983) having undertaken an histological examination did not find any calcification within the tendon itself but in the surrounding hyperplastic synovium [27]. They postulated that these calcific bodies are a reaction of the synovium to a primary tendinous lesion which may occur as a result of an episode of ischaemia or trauma.

Finally, other authors Greene (1980) have proposed that acute calcification may be similar to other crystal-induced diseases such as gout and pseudo-gout [28]. This is based on the fact that clinically they are very similar to these conditions although hypercalcaemia is not found in acute calcification.

Clinical Presentation

The condition in the fingers seems to affect women more than men with a ratio of 5:2. Ages range from 15 to 60. However, no particular occupation seems to predispose nor is there any difference between heavy, normal and lighter work. The lesion more often involves the dominant hand in the cases reported; where the finger



Fig. 8.10 Acute calcification

is named the index is the more usual. Within the finger, the proximal interphalangeal joint is the most common location (Fig. 8.10).

Pain is the most striking symptom. Often it is of acute onset, severe and throbbing in nature. Many patients also experienced a prodromal ache for anything up to 6 months prior to presentation [29].

Swelling is also a frequent sign with a localised tender nodule at the exact site of the pain [24].

Added to this, joint movement is also restricted and in a small number of cases patients experienced a pyrexia [30].

Overall most cases lasted for at most 3 weeks [31].

Obviously many of the above clinical features are shared by infective lesions of the finger e.g. cellulitis, suppurative arthritis, infected foreign bodies and indeed insect bites. However, the lack of systemic symptoms, axillary lymphadenopathy, the absence of skin abrasions together with the characteristic radiological appearances should enable the correct diagnosis to be made. Other conditions to be excluded are crystalline diseases such as gout or pseudo-gout, trauma and an acute exacerbation of rheumatoid arthritis or another connective tissue disorder.

Investigations

The diagnosis of acute calcification in the hand and wrist is generally made by x-ray. It is however important to include oblique views in addition to true antero-posterior and lateral images as many deposits can be small and difficult to see. The maximum point of tenderness localises the deposit well.

The deposit itself can be either poorly or welldefined. It is said that the more well-defined the deposit the more mature the lesion. The size can also vary from 3 mm to 2 cm and be either circular or linear in shape, depending upon the structure involved. In the fingers, it is generally held that the supporting structures around the interphalangeal joints are the more frequently involved. Added to that, however, tendon or tendon sheath can also be involved. Finally radiological features can persist up to 3 months and sometimes longer.

Treatment

Several modalities of treatment have been used in the past, including short-wave diathermy, ultraviolet light, heat and radiotherapy. However, the basis of a successful treatment would appear to be a period of immobilisation until the pain has resolved. Added to this, an injection of local anaesthestic could also appear to be beneficial [28]. Results are often dramatic with pain relief being achieved quickly and remaining long lasting. The rationale for that is unclear, but it may be that local anaesthetic improves the circulation to the area, increasing the local oxygen tension, thus aiding in the reabsorption of calcium.

The use of local steroid injections are a little more controversial, in so much that they do not seem to add any advantage over local anaesthetic alone. Various non-steroidal anti-inflammatory agents have also been used all with reported success the pain often subsiding within a few days [32].

Finally, operative intervention is rarely indicated in what is a self-limiting disease.

Clinical Pearl

Diagnosis is made by clinical suspicion Confirmed by appropriate x-ray

Treatment is by injection of local anaesthetic into the area of calcification

References

- 1. Moll JM, Wright V. Psoriatic arthritis. Semin Arthritis Rheum. 1973;3(1):55–78.
- Trail IA, Stanley JK. The hand in psoriasis. J Hand Surg Br. 1990;15B:79–83.
- Siannis F, Farewell VT, Cook RJ, Schentag CT, Gladman DD. Clinical and radiological damage in psoriatic arthritis. Ann Rheum Dis. 2006;65(4): 478–81.
- Tehranzadeh J, Ashikyan O, Anavim A, Shin J. Detailed analysis of contrast-enhanced MRI of hands and wrists in patients with psoriatic arthritis. Skeletal Radiol. 2008;37:433–42.
- Lee EY, Sundel RP, Kim S, Zurakowski D, Kleinman PK. MRI findings of juvenile psoriatic arthritis. Skeletal Radiol. 2008;37:987–96.
- Stanley JK, Jones WA, Lynch MC. Percutaneous accessory collateral ligament release in the treatment of proximal interphalangeal joint flexion contracture. J Hand Surg Br. 1986;11B:360–3.
- Belsky MR, Feldon P, Millender LH, Naleburr EA, Phillips C. Hand involvement in psoriatic arthritis. J Hand Surg Am. 1982;7(2):203–7.
- Zangger P, Esufali ZH, Gladman DD, Bogoch ER. Type and outcome of reconstructive surgery for different patterns of psoriatic arthritis. J Rheumatol. 2000; 27:967–74.
- Leibovic SJ, Strickland JW. Arthrodesis of the proximal interphalangeal joint of the finger: comparison of the use of the Herbert screw with other fixation methods. J Hand Surg Am. 1994;19A:181–8.
- Erre GL, Marongiu A, Fenu P, Faedda R, Masala A, Sanna M, Soro G, Tocco A, Piu D, Marotto D, Passiu G. The "sclerodermic hand": a radiological and clinical study. Joint Bone Spine. 2008;75: 426–31.
- Avouac J, Guerini H, Wipff J, Assous N, Chevrot A, Kahan A, Allanore Y. Radiological hand involvement in systemic sclerosis. Ann Rheum Dis. 2006;65: 1088–92.
- Hachulla E, Clerson P, Launay D, Lambert M, Morell-Dubois S, Queyrel V, Hatron PY. Natural history of ischemic digital ulcers in systemic sclerosis: singlecentre retrospective longitudinal study. J Rheumatol. 2007;34:2423–30.
- Low AHL, Lax M, Johnson SR, Lee P. Magnetic resonance imaging of the hand in systemic sclerosis. J Rheumatol. 2009;36:961–4.
- Anandacoomarasamy A, Englert H, Manolios N, Kirkham S. Reconstructive hand surgery for scleroderma joint contractures. J Hand Surg Am. 2007;32A: 1107–12.
- Melone CP, McLoughlin JC, Beldner S. Surgical management of the hand in scleroderma. Curr Opin Rheum. 1996;11:514–20.
- Hartzell TL, Makhni EC, Sampson C. Long-term results of periarterial aympathectomy. J Hand Surg Am. 2009;34A:1454–60.

- Kwon S, Eun S, Baek R, Minn K. Peripheral arterialbypass grafts in the hand or foot in systemic sclerosis. J Plast Reconstr Aesthet Surg. 2009;62:216–21.
- Bouaziz JD, Barete S, Le Pelletier F, Amoura Z, Piette JC, Francès C. Cutaneous lesions of the digits in systemic lupus erythematosus: 50 cases. Lupus. 2007;16:163–7.
- Holland NW, Jost D, Beutler A, Schumacher HR, Agudelo CA. Finger pad tophi in gout. J Rheumatol. 1996;23:690–2.
- Larmon WA, Kurtz JF. The surgical management of chronic tophaceous gout. J Bone Joint Surg. 1958; 40A(4):743–72.
- Gelberman RH, Doty DH, Hamer ML. Tophaceous gout involving the proximal interphalangeal joint. Clin Orthop. 1980;147:225–7.
- Moore JR, Weiland AJ. Gouty tenosynovitis in the hand. J Hand Surg Am. 1985;10:291–5.
- Wilczynski MC, Gelberman RH, Adams A, Goldfarb CA. Arthroscopic findings in gout of the wrist. J Hand Surg Am. 2009;34A:244–50.
- 24. Carroll RE, Sinton W, Garcia A. Acute calcium in the hand. J Am Med Assoc. 1955;157(5):422–6.

- Moschowitz E. Histopathology of calcification of spinatus tendons as associated with subacromial bursitis. Am J Med Sci. 1915;150:115–26.
- Uhthoff HK, Sarkar K, Maynard JA. Calcifying tendinitis: a new concept of its pathogenesis. Clin Orthop Relat Res. 1976;118:164–8.
- Gravanis MB, Gaffney EF. Idiopathic calcifying tenosynovitis. Histopathological features and possible pathogenesis. Am J Surg Pathol. 1983;7(4):357–61.
- Greene TL, Louis DS. Calcifying tendinitis in the hand. Ann Emerg Med. 1980;9(8):438–40.
- Hamilton AR. Calcinosis. J Bone Joint Surg. 1951; 33B:572–7.
- Selby CL. Acute calcific tendinitis of the hand: an infrequently recognised and frequently misdiagnosed form of periarthritis. Arthritis Rheum. 1984;27(3):337–40.
- DePalma AF. Calcareous deposits in soft tissues about the proximal interphalangeal joint of the index finger; report of a case. J Bone Joint Surg Am. 1947;29(3):808.
- Yelton CL, Dickey Jr LE. Calcification about the hand and wrist. South Med J. 1958;51(4):489–95.

Kienbock's Disease and Other Avascular Necrosis

Luc De Smet

Keywords

Kienböck's disease • Lunatomalacia • Lunate • Preisser's disease • Scaphoid • Johnson's disease • Capitate • Avascular Necrosis • Bone Necrosis • Carpus • Arthritis • Wrist • Leveling procedure • Proximal row carpectomy • Vascularised bone graft

Avascular (bone) necrosis can be caused by trauma, intake of (high doses) corticosteroids, alcoholism and various connective tissue disorders. However, in the majority of cases no clear aetiology can be found. In the wrist, avascular necrosis (AVN) of carpal bones involves mostly the lunate, scaphoid and sometimes the capitate. These bones have what is called a "vascular pattern-at-risk" as described by Gelberman and Gross [1]. AVN of other carpal bones has been reported, although these are even rarer. New imaging techniques, especially magnetic resonance imaging (MRI), have improved the early diagnosis of osteonecrosis, (as radiographs may be normal in the initial stages) although the ultimate diagnosis is histological.

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Kienbock's Disease

Introduction

The radiological appearances of avascular osteonecrosis of the lunate were first described by Kienbock in 1911 as "lunatomalacia" and since then this condition has been named after him [2]. The necrotic lunate becomes soft and flattens under the constant load of the wrist. This disturbs the normal architecture and kinematics of the carpus, with the ultimate development of secondary osteoarthritis. Generally, however, this progression is slow. Unfortunately with Kienbock's much is still unknown, specifically with regards to aetiology, the natural course of the disease and most importantly, treatment. There is a lack of evidence based treatment protocols. Most published series are simply case series.

History and Examination

Kienbock's disease mainly affects young males (third and fourth decade), although it can occur in

9

women, teenagers and sometimes older people. It is usually unilateral and isolated. A history of trauma is often mentioned, although this is often minor. A wrist subjected to repetitive trauma is also a common feature. Insurance companies however, usually reject the association between work and AVN. It is slowly progressive and the symptoms and signs are not very specific. Diffuse dorsal wrist pain, weakness and restricted motion are the first and often the only symptoms. On physical examination there is tenderness over the dorsal aspect of the wrist, often well localised over the dorsal side of the lunate. Later, swelling due to synovitis, loss of wrist motion and decreased grip strength occurs [3]. Early signs of synovitis include a restricted anteroposterior drawer sign at the wrist (pseudoinstability), as well as the obliteration of the anatomical snuffbox. In the anteroposterior drawer test, the examiner grasps the forearm of the patient, just proximal of the wrist joint with one hand and with the other hand holds the patient's hand just distal of the wrist joint; the joint itself being held in full pronation. The patient's hand is then moved in a dorso-palmar direction. Normally there is some translation possible, but not when there is synovitis or pain. Watson's scaphoid shift test is also usually painful. Sometimes, lunate collapse is detected on a routine wrist x-ray in an otherwise asymptomatic patient.

Investigations

The diagnosis and staging of Kienbocks disease is based on the x-ray findings [3, 4] (Table 9.1). In the early stages, plain radiographs can be normal, with the diagnosis being made by an abnormal bone scan or MRI. For the latter, an inversion of the signal on T1 and T2 weighted images is seen. Upon injection of Gadolinium, the lunate does not enhance. Schmitt et al. [3] have proposed a classification based on the MRI findings (Table 9.2).

With further progression, the typical sclerosis of the lunate appears on the anteroposterior radiographs, followed by collapse and fragmentation of the lunate. This causes a loss of the normal carpal height with rotation of the scaphoid. This change in configuration can lead to

Table 9.1 Lichtman's classif	ication
Table 9.1 Lichtman's classif	ication

Stage 1. The radiographic appearance of the lunate is normal, bone scintography is positive, MRI is bonormal.
Stage 2. The lunate exhibits increased density, but its ize and shape are normal.
Stage 3. The lunate has collapsed, allowing the capitate on migrate proximally.
In stage IIIA the scaphoid maintains a normal position relative to the rest of the carpus;
In stage IIIB there is rotatory subluxation, leading to a scaphoid "ring" sign.
Stage 4. Secondary osteoarthritis of the radiocarpal oint

altered force transmission across the wrist, with the development of secondary osteoarthritis. During this process it is postulated that new bone can be laid down at the lunate fossa, giving the impression of lengthening of the radius, compared to the ulna. Added to that and usually seen on a CT scan, a subchondral fracture of the lunate in the coronal plane can be seen. This radiological evolution forms the basis of the classification system proposed by Lichtman et al. [4]: (Fig. 9.1).

This classification has been found to be reliable and reproducible.

Another way of staging the disease is by arthroscopy. Baine and Begg [5] described four different stages of damage to the articular surfaces (Table 9.3).

In comparison to the new approach, this gives a more detailed view on the different articular surfaces, predicting the possible outcome after (surgical) treatment.

Aetiology and Evolution

The pathogenesis in Kienbocks disease is unknown. The blood supply of the lunate is variable and is thought to be the key factor in the pathogenesis of the disorder. The extra-osseous and intra-osseous vascular anatomy has been well studied [6]. According to Gelberman and Gross [1], all scaphoids and capitates and 8–20 % of lunates have a vascular pattern called "at risk" or type 1. These are carpal bones, which are mostly supplied by only

	Pattern A	Pattern B	Pattern C
Gaudolinium enhanced	Increased	Mixed signal (inhomogeneous)	Increased (homogeneous)
T1	Decreased or normal	Decreased	Decreased
T2	Increased	Decreased	Decreased

Table 9.2 Classification by MRI scan findings

Pattern A: marrow oedema and viable bone trabeculae

Pattern B: bone marrow necrosis

Pattern C: necrotic bone marrow and collapse

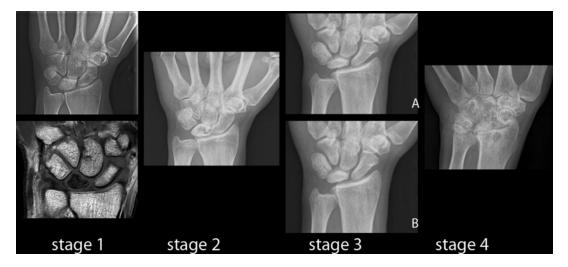


Fig. 9.1 Lichtman stages of Kienbock's disease. A and B are parts of the classification system

0: Normal articular	surfaces
1: Only the proxim	al surface of the lunate is damaged
2A: Distal and proz are involved	ximal articular surfaces of the lunate
2B: Fracture in the	coronal plane
	both articular surfaces of the lunate a of the distal radius
4: Finally, also the	head of the capitate is damaged

 Table 9.3
 Classification by wrist arthroscopy findings

one vessel without additional anastomoses, therefore occlusion of this vessel would lead to necrosis. Besides arterial insufficiency, venous stasis has also been suggested as a possible cause of (carpal) bone necrosis. Extrinsic factors, such as fractures or repetitive minor trauma, can damage the intra-osseous blood supply leading to osteonecrosis, although the evidence for this is poorly documented. Even in lunate and perilunate dislocations the lunate seldom, if ever, undergoes avascular necrosis. The presence of a fracture therefore seems to be more a consequence rather than a cause of the condition.

Hulten [7] reported on the association between Kienbock's disease and an ulnar minus variance. Several authors have reported a change in ulnar variance with age, sex and position of the wrist, as well as in osteoarthritis due to Kienbock's disease [8, 9]. Other authors have confirmed this finding. However, more recently an alternative explanation has been put forward [10, 11] involving a (pseudo) lengthening of the radius occurring due to bone apposition on the lunate facet of the distal radius (Fig. 9.2). In our personal series, we found that a negative ulnar variance did not predispose to Kienbock's disease, although there were indications that a negative ulnar variance could predispose to a more rapid progression of the disease process [12]. A recent biomechanical study, using finite element modelling, demonstrated that ulnar minus variance was instrumental to further collapse of the lunate [13].

bone apposition on the lunate fossa facing the necrotic

Other morphological factors of both the distal radius and the lunate may also play a role in the aetiology of Kienbocks [14]. Comparing the contralateral wrist with an age and sex matched control group of patients with Kienbocks, the lunates were smaller and had more of a radial tilt than the controls and the radial slope of the distal radius was less. Antuna-Zapico [15] distinguished different types of lunate, one of which was more prone to necrosis.

There are also different patterns of evolution once the lunate becomes necrotic. The general pattern was described by Lichtmann et al. [4] who modified the initial grading by Stahl [16]. The lunate itself can undergo different types of collapse. The lunate can also fracture, usually in a coronal plane. Subsequently and in some incidences the proximal row remains stable. However, in most cases, with collapse of the lunate the normal architecture of the carpus is lost. Generally this results in flexion of the scaphoid. Depending on other factors, this will result in either dorsal or volar angulation of the collapsed lunate. This carpal collapse ultimately leads to (painful) degenerative changes: Kienbock related advanced collapse (KRAC) - wrist.

Management

A thorough evaluation of the involved wrist is necessary, prior to treatment. Not all wrists need treatment. When the pain is mild there is no need to recommend surgery. Acute pain may also disappear rapidly with rest and analgesics. Only when symptoms are long standing (more than 1 year) and are affecting the quality of life of the patient, should surgery be considered. With regard to the surgery itself, treatment is very much decided by the stage of the disease process. If the lunate itself has not collapsed, or there is little evidence of carpal collapse, then some form of reconstructive surgery, by way of a combination of both decompression and the insertion of a local vascular graft, is recommended. If, however, the lunate is fragmented and there is severe carpal collapse then salvage surgery, by way of either arthrodesis or excision (proximal row carpectomy), is more suitable.

- Not all patients with Kienbock's disease need surgical intervention.
- The need for surgery is based on the level of symptomatology, particularly pain.
- If surgery is required, the procedure undertaken is dictated by the staging of the disease process.

Other patient factors are equally important; including, age and demands of the patient, profession and hobbies, smoking habits, their views on surgery and acceptance of the risks. The clinical condition of the wrist, particularly any synovial swelling or rigidity, also has a bearing on decision making. Obviously, it is ill advised to operate on an acutely inflamed wrist, and a wrist that is already stiff will become no more mobile following arthrodesis.

Fig. 9.2 Pseudolengthening of the distal radius due to lunate



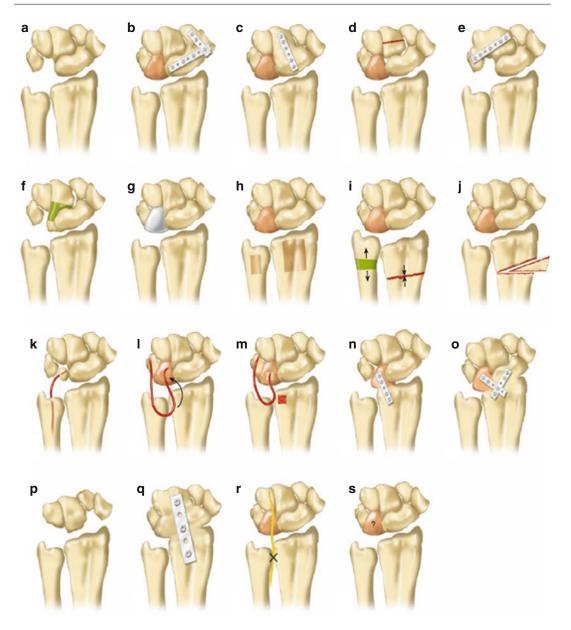


Fig. 9.3 Overview of the most popular procedures to treat Kienbock's disease. (a) Resection of the lunate. (b) STT arthrodesis with or without lunate resection. (c) Scaphocapitate arthrodesis with or without lunate resection. (d) Capitate shortening. (e) Lunate resection with ulnar column arthrodesis. (f) Lunate resection and capitate lengthening. (g) Lunate prosthesis. (h) Metaphyseal osteotomy (Core decompression). (i) Ulna lengthening (*arrows*) and/

or radial shortening (*arrows*). (**j**) Wedge (opening or closing) osteotomy. (**k**) Lunate resection and substitution with the vascularised pisiform. (**l**) Direct vessel implantation. (**m**) Vascular bone graft (VBG). (**n**) Radio-lunate (Chamay) arthrodesis. (**o**) Radio-lunate-scaphoid arthrodesis. (**p**) Proximal row resection. (**q**) Full wrist arthrodesis. (**r**) Denervation. (**s**) whatever the future brings

Treatment options include both conservative and operative methods. As a rule, surgical treatment is preferred, since it generally leads to quicker improvement in symptoms and better outcomes. Many operative treatments have been devised for the various stages of Kienbock's disease. Generally, however, they can be grouped into three broad categories (Fig. 9.3):

- (i) Direct revascularisation by vessel implantation or a vascularized bone graft (VBG) (stage 1 and 2).
- (ii) Joint decompression. This not only relieves the symptoms, but it allows spontaneous revascularisation of the lunate, whilst diminishing the compressive forces acting on it (stage 1, 2 and 3A). This can be done by levelling the distal radioulnar joint (DRUJ), intracarpal arthrodesis (scaphotrapezialtrapezoidal (STT) or scaphocapitate arthrodesis) or capitate shortening. Restoring the normal carpal height is the key element in preventing osteoarthritis.
- (iii) Salvage procedures for pain relief. These include wrist denervation, (resection) arthoplasty or radiocarpal arthrodesis (stage 3B and 4).

Non Operative Treatment

The rationale for this approach lies in the observation that spontaneous recovery can occur in some early onset cases. Cases of asymptomatic patients, with longstanding radiographic evidence of Kienbock's have also been reported. Previous reports suggest that the pain can subside within a few years. Kristensen et al. reported good results after short periods of immobilisation [17]; whereas Mikkelsen and Gelineck noted poor outcomes after non-operative treatment [18]. The debate continues, with some authors advocating a conservative approach, whereas others have observed progressive clinical and radiographic deterioration [19], or have demonstrated better outcomes with surgery [20]. A "watch and wait" policy, or, in a very painful wrist, a temporary short period of immobilisation with casting or bracing, however, can result in an asymptomatic wrist. A non-operative approach is also preferred in younger patients (Fig. 9.4). What we and others have observed in these, albeit rare, cases, is that there is some adaptation of the carpus through remodelling, rather than collapse.

Revascularisation by Vascularised Bone Implantation

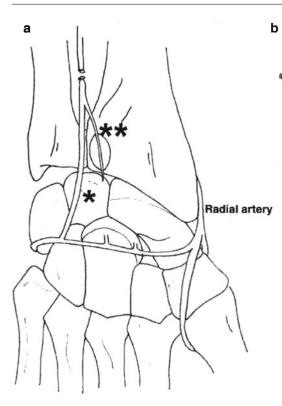
Direct revascularisation, or replacement of necrotic bone by well-vascularised bone, is a logical

Fig. 9.4 Juvenile Kienbock's disease

approach to the problem. The basic principles involve revascularisation of the lunate and temporary stabilisation of the carpus (to prevent collapse during the revascularisation period), which preserves the integrity of the lunate and the architecture and kinematics of the wrist. Historically, the lunate has been replaced by the pisiform bone, which is pedicled on the ulnar artery [21]. This procedure is usually called Saffar's technique. Other authors have removed the necrotic bone by curettage, followed by cancellous bone grafting and vessel implantation, using the posterior interosseous artery. There is some evidence that the bone grafts were revascularised by this technique. We found only one paper using this technique in 11 wrists [22]. They reported stabilisation of the lunate with good pain relief in nine patients. More recently Jones et al. described one case of vessel implantation, in combination with Bone Morphologic Protein (BMP) [23].

Vascularised bone grafts (VBG) have the advantage of immediate implantation of viable bone, which simplifies matters by substituting the bone defect for a healing fracture. One does not have to wait for the secondary revascularisation of a cancellous bone graft (creeping substitution) and it avoids the period of temporary weakening that occurs with non-vascularised bone grafts.

The Mayo group studied the vascularity of the distal radius. Based on anatomical studies



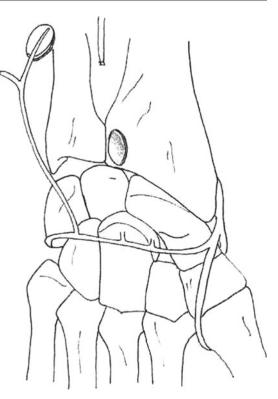


Fig. 9.5 Schematic presentation of the VBG. (a) Anatomy of the fifth extensor compartment artery (ECA) (*) and the fourth ECA (**). (b) Ligation of interosseous

and animal experimentation they developed the use of the so-called 4, 5 extensor compartment vascularised bone graft [24]. In their clinical series of 26 patients they reported pain relief in 92 % and a significant improvement in grip strength and maintenance of carpal height in 77 %. We have also applied this technique in the treatment of early Kienbock's. Contraindications to VBG would include smoking, the older patient, a patient with a stiff wrist and a patient where the lunate is fractured.

Surgical Technique of the Dorsal VBG (Fig. 9.5)

A straight skin incision is made over the dorsal aspect of the wrist, from the third metacarpal base to the distal forearm. The fifth dorsal extensor compartment is exposed and incised. The extensor tendons are retracted radially. The fifth extensor compartment artery (ECA), which originates from the dorsal branch of the anterior interosseous artery, is identified. The fourth extensor

artery and harvesting of the graft. Retrograde vascularisation of the graft from the fifth ECA towards the fourth ECA

compartment artery originates from the fifth ECA or anterior interosseous artery and supplies nutrient branches to the dorsal aspect of the radius within the fourth extensor compartment. A bone graft proximal to the radiocarpal joint and overlying the fourth ECA, including the nutrient vessels, is outlined. The dorsal wrist capsule is opened, according to Berger's ligament sparing incision. The lunate is then exposed and the necrotic part is removed through a dorsal cortical window. The anterior interosseous artery is ligated proximal to the fourth and fifth ECA. The vascularised bone is now mobilised (Fig. 9.6) and is placed into the gap in the lunate. In patients with stage IIIa lunatomalacia, an STT-fusion is also performed at the same time, in order to control the rotary subluxation of the scaphoid and to prevent carpal collapse. Postoperatively, the patients are immobilised in a below-elbow splint for 6 weeks.

The metacarpal head of the index ray is another donor site for VBG. It was first described

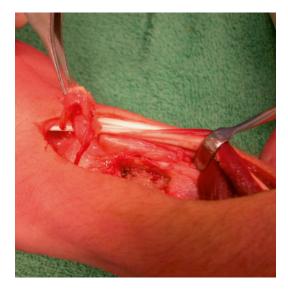


Fig. 9.6 Intra-operative view: the VBG is pre-elevated and will be moved distally

by Bengoechea et al. (one case) [25] and then by Zafra et al. (five cases) [26]. All cases obtained a satisfactory outcome, but all had an additional procedure on the radius (shortening or wedge osteotomy).

Finally, free vascularised bone grafts from the iliac crest have also been used [27, 28]. Both authors mentioned above performed this procedure in 18 wrists and both observed good bony integration in 16 wrists.

Leveling Procedures of the DRUJ: Radial Shortening or Ulna Lengthening

Based on Hulten's finding [7] that Kienbock's disease was more frequent in ulna minus variant wrists, levelling the ulna to the radius by shortening the radius or lengthening the ulna has been proposed. This is one of the oldest and most accepted techniques for the treatment of Kienbock's disease. Radial shortening is now preferred to ulna lengthening, as the latter has a high incidence of non-union. Several large series have been reported using these procedures [20, 24–39] (Tables 9.4 and 9.5). The basic mechanism seems to be an unloading of the lunate and prevention of further collapse. More sophisticated procedures include lateral closing and opening wedge osteotomies of the distal radius.

Tab	le 9	9.4	Result	ts of	radial	shortenin	g
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Author	Year	Ν	% good
Axelson	1973	19	100
Rosemeyer	1976	19	69
Schattenkerk	1987	20	70
Nakamura	1990	23	83
Weiss	1991	29	87
Gomis	1994	28	85
Siala	2000	31	80

 Table 9.5
 Results of ulna lengthening

Author	Year	Ν	% good
Armistead	1982	20	90
Sundberg	1983	19	95
Quenzer	1993	64	90
Trail	1996	20	100

Most classical papers and handbooks still recommend an osteotomy for stages one and two and sometimes for stage 3, provided there is an ulna minus variance. The morphology of the sigmoid notch and the ulnar head must be evaluated prior to any joint levelling procedure. Morphological studies of the DRUJ [40, 41] have distinguished three different types of sigmoid notch, which creates the potential for DRUJ incongruity or impingement following a change in the length of one of the forearm bones. Other types of decompressive procedure should be considered in these cases. These would include various intracarpal procedures [42, 43].

Surgical Technique of the Radial Shortening Osteotomy

A standard volar approach is used, radial to the flexor carpi radialis. The distal one-third of the radius is exposed. A six hole dynamic compression plate (DCP) or a three hole distal radial fracture fixation plate is applied on the volar surface. Markings are made to determine the level of the osteotomy and the correct rotation of the radius. The drill holes for the proximal three holes are made and the plate is provisionally fixed with one screw, but not tightened. The plate is rotated over 90° and the osteotomy is performed. A 2 mm slice of bone is removed. This is sufficient to decompress the lunate. Next, the plate is swung

back and the distal part of the radius is brought into close contact with the proximal part and the plate is fixed under compression, according to the standard techniques of osteosynthesis. Immediate postoperative mobilisation is allowed.

Adequate pain relief has been obtained with levelling procedures (radial shortening and ulna lengthening). The fate of the lunate in these procedures is, however, not clear. Weiss [38] found no progression, whilst Wada et al. [44] and our previous study found further progression of the disease process.

Proponents of more sophisticated procedures, including lateral closing or opening wedge osteotomies and medial closing wedge osteotomies of the radius claim that the osteotomy changes the morphological aspects of the distal radius [44, 45]. In general the results are also good. However, it is felt that radial opening osteotomies produce a better decompression than the closing wedge osteotomy [46]. The clinical outcomes, however, between the two were not significantly different.

Some authors believe that the biological effect of the osteotomy is responsible for the pain relief. Based on this, Illarramendi et al., proposed a simple fenestration of the distal radius and ulna, rather than changing the length or orientation [47]. Their series consisted of 22 patients, of which 16 were pain free and 4 had only moderate pain. Schultz et al. confirmed these findings in his series of 10 patients [48].

Intracarpal Procedures

One of the most important consequences of lunate necrosis and the subsequent collapse, is the disturbance of the overall carpal architecture, in particular rotational subluxation of the scaphoid. Based on the work by Watson et al. on the treatment of scapholunate ligament tears, some authors have reported good results with an STT arthrodesis for treating Kienbock's disease [49–51] (Fig. 9.7). It has also been proven that this procedure unloads the lunate to a similar degree as a joint levelling procedure. The STT arthrodesis can also be combined with a VBG or other revascularisation procedures (in Lichtman's stage 3a). In earlier stages, temporary fixation of the scaphoid to the capitate, using K wires, for a period of



Fig. 9.7 STT arthrodesis with the use of Shapiro staples. The geometry of the lunate remain unchanged with time

6–8 weeks, rather than a formal arthrodesis is a good alternative.

Other intracarpal procedures include scaphocapitate arthrodesis, a capitate shortening osteotomy and triquetro-capito-hamate fusion combined with, or without, a lunate resection.

Pre-existing wrist stiffness is a relative contraindication for a partial arthrodeses. A stiff wrist rarely gets better with a fusion. The main complications of these procedures include non-union, hardware failure and, last but not least, impingement between the radial styloid and the scaphoid, with the development of osteoarthritis. This complication is hard to deal with, since it appears to progress even when a radial styloidectomy has been undertaken.

Surgical Technique of the STT

We prefer a palmar Russe approach. The FCR tendon sheath is incised on its radial side and the tendon retracted ulnarly. The wrist joint is accessed through the bed of the FCR sheath. The STT joint is exposed distally and the capsule is incised transversally. The scaphoid is reduced by manual pressure and fixed to the capitate with a K-wire. The cartilage and subchondral bone of the distal pole of the scaphoid and the proximal side of the trapezium and trapezoid are removed. The gap is filled with cancellous bone from the iliac crest, or the distal radius. The STT joint is fixed with K-wires, staples or a Herbert screw (Fig. 9.7). Immobilisation in a cast is continued for up to 6 weeks, followed by wrist motion exercises.

Most authors [52, 53] recommend this procedure as a primary treatment. The outcomes are generally reported as good, with Mayo wrist scores between 62 and 66 and a DASH score of 24.8. However, some authors have reported no significant differences between an STT fusion and radial shortening, although the outcome was better than a full radiocarpal fusion [53].

With regard to capitate shortening, whilst this does decompress the lunate, the clinical and radiological results so far have been reported as poor.

Scaphocapitate fusion has a similar biomechanical effect as an STT fusion. Sennwald and Uferrast identified satisfactory results in 10 of their 11 cases [54]. Capitohamate fusion also resulted in an excellent outcome in the series of Oishi et al. (45 patients, 42 painfree) [55].

A more sophisticated procedure was proposed by Wilhelm et al. [56]. After resection of the lunate, a transverse osteotomy of the capitate is performed and, by callus distraction, brought into the empty space. They performed this in 14 patients. Lu et al. had a larger series (30 patients) and reported reasonable results [57]. However, we do not see any advantage over a simple proximal row carpectomy.

Salvage Procedures

Once osteoarthritis is present (stage 4), or in stiff wrists (stages 3B), a reliable salvage procedure is preferred. As with other joints, the solutions are either arthrodesis, excision arthroplasty or denervation. The outcome with total wrist fusion is less satisfying, with a high percentage of complications and unsatisfactory results. Our own follow-up studies have demonstrated that preservation of some wrist motion is preferred by patients, although the amount of movement is not crucial. Partial – radiolunate and radioscapholunate fusions – have been proposed to overcome the poor results of full wrist arthrodesis. Kilgus et al. found an acceptable result in five cases of radioscapholunate fusion [58]. Conversely the Wrightington group had 50 % failure of their radio-lunate fusions [59].

Full radiocarpometacarpal arthrodesis resulted in 55 % pain reduction, a DASH of 51.4 and 70 % return to previous occupations in the large series of Sauerbier et al. [60]. This was confirmed in another smaller series by Tambe et al. [53].

Arthroplasty, by using a silicone spacer, was initially proposed by Swanson in 1993 [61]. However, silicone synovitis, with the appearance of intraosseous cysts all over the carpus and distal radius, is a major potential complication. Kaarela et al. had to remove 12 of the spacers in 39 patients [62]. Partial denervation of the wrist joint, by sectioning the posterior interosseous nerve, is a common adjunctive procedure. An isolated full denervation of the wrist joint is, however, also possible and indicated in limited situations i.e. with older patients, those with moderate pain and in patients who wish to retain as much range of motion as possible.

For most clinicians, however, the first choice is proximal row carpectomy (PRC), particularly for stiff wrists with stages 3 and 4 of disease. Despite the theoretical possibility of damage to the lunate fossa, proximal row carpectomy has led to very satisfactory outcomes in most series and also in our hands [63–65].

PRC, however, is not indicated if there is severe damage to the cartilage of the lunate fossa, ulnar translocation of the wrist (seen sometimes following previous radial styloidectomy), damaged capitate head or following previous intracarpal arthrodeses.

Surgical Technique of the Proximal Row Carpectomy

A standard longitudinal dorsal approach is used. The dorsal retinaculum is opened over the third compartment and the dorsal capsule is incised according to Berger by a ligament sparing incision [24]. The carpal bones are then inspected. If the status of the cartilage on the lunate fossa of the radius and on the capitate are found to be satisfactory, the procedure can continue. Usually, the carpal bones are removed piecemeal; "en bloc" resection is rarely possible. Capsule and retinaculum are then repaired anatomically. Postoperatively, the wrist is immobilised in a cast for 4 weeks, although this is not always necessary.

Although most reported series are small, proximal row carpectomy in Kienböck's disease mostly results in a favourable outcome. We assessed the outcomes in 21 patients (mean age 39 years) with advanced Kienbock's disease treated by resection of the proximal carpal row [50]. Thirteen patients had little or no pain, 3 had moderate and 5 had severe pain. Begley and Engber also reported satisfactory results in 14 patients, with decreased wrist pain in all patients, grip strength of 72 % of the contralateral side, unchanged range of motion or slight improvement in 12 of the 14 patients after surgery and a return to former employment for all patients [66].

Conclusion

Kienbock's disease is a progressive and chronic wrist disorder, which occurs as a result of avascular necrosis of the lunate, ultimately leading to osteoarthritis of the wrist. It usually affects young and active adults.

Non-operative treatment is recommended for children and juvenile patients. In older patients with mild symptoms, a conservative approach is also recommended, at least in the first instance.

Otherwise surgical treatment is undertaken. The chosen procedure depends on the stage of the disease, the ROM of the wrist, the length of the ulna, the shape of the sigmoid notch and the presence or absence of a fracture of the lunate.

For stage 1 and 2 we prefer a VBG, with temporary scapho-capitate fixation. An alternative is a radial shortening osteotomy, provided that the ulna is short and that the geometry of the DRUJ can accommodate this.

For stage 3a we currently recommend a VBG with definitive STT arthrodesis, provided that the ROM is acceptable and there is no fracture of the lunate. However, the situation is fluid and we are not convinced that an STT arthrodesis is always required.

For stage 3b and for stage 3a with a fracture of the lunate, with acceptable ROM, we believe a salvage procedure, particularly PRC, gives better results. Similarly for stage 4 and stiff stage 3b, where there is acceptable cartilage on the radius and the head of the capitate, a PRC is an excellent procedure.

For those in which a PRC would be ill advised, due to severe damage to the lunate fossa of the distal radius, either a full wrist arthrodesis, or a denervation for the older patient, is recommended. Finally, prosthetic replacement of the wrist is possible, although experience is limited.

- Stage 1, 2 & 3A preferred treatment is some form of decompression, either radial shortening or an STT fusion with a local vascular bone graft.
- Stage 3B proximal row carpectomy and sometimes formal arthrodesis
- Stage 4 with significant cartilage involvement, wrist arthrodesis
- For patients with stiff wrists, proximal row carpectomy is probably the preferred option.
- Finally, denervation is an option for patients with mild pain who wish to retain full range of motion, although results are unpredictable.

Preiser's Disease: Non-Traumatic Necrosis of the Scaphoid?

In medical literature, the eponym "Preiser" has been used for the idiopathic avascular necrosis of the scaphoid bone [67]. In contrast to Kienbock's disease, the scaphoid is rarely involved in idiopathic bone necrosis, despite the high incidence of necrosis of the proximal pole following scaphoid fractures and the perilous nature of its blood supply [68, 69]. Of the cases reported by Preiser in 1910, all had previously reported an incident of acute trauma and it is not unreasonable to suppose that his cases were scaphoid fractures with post-traumatic pseudarthrosis and bone necrosis. Since then, controversy exists about the actual existence of this disease.

As expected, the aetiology remains unknown. Most authors, however, agree that the common pathway is impaired blood supply to the scaphoid.



Fig. 9.8 Preiser's disease or AVN of the Scaphoid: different aspects

Taleisnik and Kelly [69], Gelberman and Menon [68] and Gelberman and Gross [1] have studied the vascularity of the carpus extensively. They were able to demonstrate a blood supply at risk. Specifically, the proximal pole receives its blood supply from a distal branch of the radial artery entering through the dorsal ridge of the scaphoid and running retrogradely. According to these authors, these scaphoids have a type I intraosseous blood supply, meaning that only one vessel is responsible for supplying the larger part of the scaphoid. This branch traverses an intra-articular membrane. It is proposed therefore, that when intra-articular pressure rises, compression and occlusion of this branch can occur. This is not only the case when there is intra-articular synovitis, but also when the extensor carpi radialis brevis exerts pressure on the scaphoid's surface when the wrist is flexed and ulnarly deviated. Repetitive microtraumas are also held responsible for occlusion of the nutrient branches. The distal pole has supplementary vascularisation and is better protected against bone infarction (Fig. 9.8).

Most publications are case reports or limited series. Since the 1990's, however, the interest in this disease has increased. Vidal et al. in 1991 [70] and Herbert and Lanzetta in 1993 [71] published some larger series (resp. 9 and 8 cases). However, 1 had a mal-united fracture of the scaphoid. Kaleinov et al. distinguished two patterns of the disease: type I (diffuse ischemia and necrosis) and type II (localised necrosis) based on a series of 19 cases [72]. We previously reported a series of 21 cases. On several occasions the condition has been related to systemic disease and/or steroid ingestion, chemotherapy [70], hypoplasia of the scaphoid [70, 73], ulna minus [73] and ulna plus variance [71].

Parkinson et al. suggested that a negative ulnar variance could also be a predisposing factor for avascular necrosis of the scaphoid [73]. This was rejected by Vidal et al. [70] and De Smet et al. [74]. In the larger series of Herbert and Lanzetta [71], 7 of the 8 cases even had an ulnar plus. The distribution of ulnar variance in our series was also not significantly different to a control group. The diagnosis is obvious in most cases, although strict criteria have not been established. We propose the following criteria:

- absence of trauma (even minor) and/or surgical procedures to the wrist
- 2. radiological alterations (condensation, cysts, collapse) involving at least 80 % of the bone
- 3. MRI changes involving the whole scaphoid (with the exception of the distal tubercle)
- 4. histological examination indicating bone necrosis in the distal part of the scaphoid.

For the full diagnosis, the first two criteria would be suggested and the latter two confirmative.

Treatment is still controversial. Conservative measures (NSAID's, rest and splinting) are recommended initially, although the outcome is variable. Scaphoid excisions, partial or total, with or without replacement have been reported. Several so called salvage procedures can be undertaken, including proximal row carpectomy, a full wrist fusion, a four-part bone fusion (lunate-capitate-triquetrum and hamate) with scaphoid resection or a denervation of the wrist. Proximal row carpectomy was reported as giving good results by Alnot et al. [75]. Recently, Moran et al. reported a small series of revascularisation of the scaphoid using a vascularised bone graft taken from the distal radius with promising results [76].

Avascular Necrosis of the Capitate: Jönsson Disease

Despite the fact that the capitate has a similar vascular supply to the lunate and the scaphoid, cases of idiopathic AVN of the capitate are rare and in the literature only single cases are reported. This condition has been named after Jönsson [77] (Fig. 9.9), although there are only 24 cases identified in the literature. AVN of the capitate can be seen after fractures of the capitate, with or without perilunate dislocation (Fenton's Syndrome or trans-scaphoid, trans-capitate perilunate dislocation or capitonavicular Syndrome). Dereudre et al. described three distinct aetiological factors; micro-trauma, hyperlaxity or carpal instability and vascular factors [78]. We also think that metabolic causes should be included. There have also been reports of AVN of the capitate in hyperuricaemia steroid intake and Gaucher's disease. Usually it is found in young adults (athletes), the symptomatology is non-specific and the keystone of diagnosis is clinical suspicion. The diagnosis relies on radiographic appearance and MRI findings. Milliez described 3 types: I. the head of the capitate only, II. the body of the capitate only and III. total involvement [79]. Treatment is still a matter of discussion. Conservative measurements have been proposed in children. Recently, vascularised bone grafts have been prosposed [80]. A partial prosthesis of the head of the capitate (pyrocarbon) seems to be logical, but no long term series has been reported. Finally, an arthrodesis between the capitate, scaphoid and lunate, or a full midcarpal arthrodesis are good salvage options.

Necrosis of the Other Carpal Bones

• Since the other carpal bones do not have an 'at risk' vascular supply, the incidence of avascular necrosis is rare. The diagnosis is



Fig. 9.9 AVN of the Capitate

often made after a long period of complaints and multiple examinations. Since the availability of MRI, the diagnosis has become more reliable. Osteonecrosis, or AVN of the hamate, (Fig. 9.10) is very rare and only a few cases have been reported. The hamate has a type 2 vascular supply, without intraosseous anastomoses. Although the natural evolution of necrotic carpal bones is not known, certainly for those bones involved, pain is the major reason to treat these patients. One can also assume that these carpal bones will ultimately collapse, resulting in articular incongruity and subsequently osteoarthritis. As a consequence, a more aggressive approach has been recommended. Two of the previously reported cases underwent curettage and conventional grafting associated with a capitohamate fusion. Both resulted in good pain relief, although the range of motion was reduced. The recent development of vascularised pedicle bone grafts from the distal radius



Fig. 9.10 AVN of the Hamate



Fig. 9.11 AVN of the scaphoid, lunate and hamate

Table 9.6 Case reports of avascular necrosis in other carpal bones

Triquetrum:	1 case [80]
Pisiform:	2 cases [81, 82]
Trapezium:	1 case [83]
Trapezoid:	1 case [84]
Multiple bones:	3 cases [85]

seem to have had excellent early clinical and radiological success, as described by Moran et al. [76]. We used the fifth extensor compartment VBG in a personal case.

- The case of a 66-year old female patient with hyperlipidaemia, corticosteroid induced osteoporosis and obstructive lung disease, with avascular necrosis of the proximal row of the carpus (Fig. 9.11) and the hamate is described. No other sites of avascular bone necrosis were identified. This patient, however, similar to the previously described one, had several contributing factors for avascular necrosis.
- For other isolated carpal bone AVN's, please see Table 9.6:



Fig. 9.12 AVN of head of the metacarpal

Avascular Necrosis of the Head of the Metacarpal: Dieterich Disease

Avascular necrosis of the metacarpal head, or Dieterich's disease, is also extremely rare and has been associated with trauma, systemic lupus erythematosis (SLE), congenitally short digits and steroid use [86] (Fig. 9.12).

It has been reported in all the metacarpal heads, but appears to involve most frequently the long finger, followed by the index, ring and small fingers. The thumb is least commonly involved [87].

Wright and Dell, who studied the vascularity of the metacarpals, found that in 35 % of the specimens, the main artery to the distal epiphysis was absent, making these metacarpal heads solely dependent on small circumferential peri-capsular vessels [88]. This pattern is even more frequent in the middle finger (60 %). Blunt trauma to the metacarpal head, with joint effusion, may compress the periosteal blood vessels, causing necrosis. In SLE, avascular necrosis occurs as a result of vasculitis involving these vessels.

If the diagnosis is not obvious on standard radiographs and there is only slight flattening of the metacarpal head or disruption of the trabecular pattern, further investigation is necessary. A technetium bone scan will show an area of increased uptake at the involved MCP joint. At the same time, it is useful to screen the other bones in patients using steroids. Recently, MRI has proven to be very useful in establishing the diagnosis of avascular necrosis. It also quantified the extent of the necrotic zone and any involvement of the overlying cartilage. Usually MRI demonstrates a hypointense zone on T1-weighted images and a hyperintense zone on T2-weighted images, both suggestive of avascular necrosis.

Several treatment options, ranging from rest to surgery, exist. The type of surgery depends on the quality of the cartilage layer. If this is still good, subchondral debridement and cancellous bone grafting gives excellent results. If the cartilage is destroyed however, arthroplasty or arthrodesis may be necessary.

References

- 1. Gelberman R, Gross M. The vascularity of the wrist. Clin Orthop. 1986;202:40–9.
- Wagner JP, Chung KC. A historical report on Robert Kienböck (1871–1953) and Kienböck's disease. J Hand Surg. 2005;30A:1117–21.
- Schmitt R, Fellner F, Obletter N, Fiedler E, Bautz W. Diagnosis and staging of lunate necrosis. A current review. Handchir Mikrochir Plast Chir. 1998;30:142–50.
- Lichtman DM, Alexander AH, Mack GR, Gunther SF. Kienböck's diseas–pdate on silicone replacement arthroplasty. J Hand Surg Am. 1982;7A:343–74.
- Baine GI, Begg M. Arthroscopic assessment and classification of Kienbock's disease. Tech Hand Up Extrem Surg. 2006;10:8–13.
- Gelberman RH, Bauman TD, Menon J, Akeson WH. The vascularity of the lunate bone and Kienböck's disease. J Hand Surg Am. 1980;5A:272–8.
- Hulten O. Uber anatomische variationene der Handgelenckknochen. Acta Radiol. 1928;9:155–69.
- Epner R, Bowers W, Guilford W. Ulnar variance the effect of positioning on roentgen filming technique. J Hand Surg Am. 1982;7:298–305.
- Palmer AK, Glisson RR, Werner FW. Ulnar variance determination. J Hand Surg Am. 1982;7:376–9.
- Weiss AP. Negative ulnar variance is not a risk factor for Kienböck's disease. J Hand Surg. 1994;19-A:1057–8.
- Kristensen SS, Thomassen E, Chistensen F. Ulnar variance in Kienböck's disease. J Hand Surg. 1986;11-B: 258–60.
- Goemine S, Degreef I, De Smet L. Negative ulnar variance has a prognostic value in the progression of Kienböck's disease. Acta Orthop Belg. 2010;76:38–41.

- Ledoux P, Lamblin D, Wuilbaut A, Schuind F. A finite-element analysis of Kienbock's disease. J Hand Surg. 2008;33 Eur:286–91.
- Mirabello SC, Rosenthal DI, Smith RJ. Correlation of clinical and radiographic findings in Kienböck's disease. J Hand Surg. 1987;12-A:1049–54.
- Antuna-Zapico JM. Malacia del Semilunar. Doctoral thesis, Valladolid, 1966.
- Stahl F. Lunatomalacia. Acta Chir Scand Suppl. 1947;95:126.
- Kristensen SS, Thomassen E, Christensen F. Kienböck's disease–late results by non-surgical treatment. A follow-up study. J Hand Surg. 1986;11 Br: 422–5.
- Mikkelsen SS, Gelineck J. Poor function after nonoperative treatment of Kienböck's disease. Acta Orthop Scand. 1987;58:241–3.
- Keith PP, Nuttall D, Trail I. Long-term outcome of nonsurgically managed Kienböck's disease. J Hand Surg. 2004;29 Am:63–7.
- Salmon J, Stanley JK, Trail IA. Kienböck's disease: conservative management versus radial shortening. J Bone Joint Surg. 2000;82 Br:820–3.
- Saffar P. Replacement of the semilunar bone by the pisiform. Description of a new technique for the treatment of Kienboeck's disease. Ann Chir Main. 1982;1: 276–9.
- Moneim MS, Duncan GJ. Kienbock's disease: treatment by implantation of vascular pedicle and bone grafting. Iowa Orthop J. 1998;18:67–73.
- 23. Jones NF, Brown EE, Vögelin E, Urist MR. Bone morphogenetic protein as an adjuvant in the treatment of Kienbock's disease by vascular pedicle implantation. J Hand Surg Eur Vol. 2008;33Eur: 317–21.
- 24. Moran SL, Cooney WP, Berger RA, Bishop AT, Shin AY. The use of the 4 + 5 extensor compartmental vascularised bone graft for the treatment of Kienböck's disease. J Hand Surg. 2005;30A:50–8.
- Bengoechea-Beeby MP, Cepeda-Uña J, Abascal-Zuloaga A. Vascularized bone graft from the index metacarpal for Kienböck's disease: a case report. J Hand Surg [Am]. 2001;26:437–43.
- Zafra M, Carrasco-Becerra C, Carpintero P. Vascularised bone graft and osteotomy of the radius in Kienböck's disease. Acta Orthop Belg. 2005;71:163–8.
- Arora R, Lutz M, Deml C, Krappinger D, Zimmermann R, Gabl M. Long-term subjective and radiological outcome after reconstruction of Kienböck's disease stage 3 treated by a free vascularized iliac bone graft. J Hand Surg. 2008;33 Am:175–81.
- 28. Gabl M, Lutz M, Reinhart C, Zimmerman R, Pechlaner S, Hussl H, Rieger M. Stage 3 Kienböck's disease: reconstruction of the fractured lunate using a free vascularized iliac bone graft and external fixation. Hand Surg. 2002;27 Br:369–73.
- Armistead RB, Linscheid RL, Dobyns JH, Beckenbaugh RD. Ulnar lengthening in the treatment of Kienböck's disease. J Bone Joint Surg. 1982;64 Am:170–8.

- Axelson R. Niveau Operationen bei Mondbein nekrose. Handchirurgie. 1973;5:187–96.
- Rosemeyer B, Artmann M, Viernstein K. Kienbock's disease. Follow up studies and therapeutic considerations. Arch Orthop Unfallchir. 1976;85(1):119–27.
- 32. Gomis R, Martin B, Idoux O, Chammas M, Allieu Y. Kienboeck disease: treatment by shortening osteotomy of the radius. Rev Chir Orthop Reparatrice Appar Mot. 1994;80:196–204.
- Nakamura R, Imaeda T, Miura T. Radial shortening for Kienböck's disease: factors affecting the operative result. J Hand Surg. 1990;15 Br:40–5.
- Quenzer DE, Dobyns JH, Linscheid RL, Trail IA, Vidal MA. Radial recession osteotomy for Kienböck's disease. J Hand Surg. 1997;22 Am:386–95.
- 35. Siala A, Ben Ayeche ML, Frikha R, Ghannouchi G, Moula T. Results of diaphyseal shortening of the radius in the treatment of Kienböck's disease: a series of 31 cases. Rev Chir Orthop Reparatrice Appar Mot. 2000;86:151–7.
- Sundberg SB, Linscheid RL. Kienböck's disease. Results of treatment with ulnar lengthening. Clin Orthop Relat Res. 1984;187:43–51.
- Trail IA, Linscheid RL, Quenzer DE, Scherer PA. Ulnar lengthening and radial recession procedures for Kienböck's disease. Long-term clinical and radiographic follow-up. J Hand Surg. 1996;21 Br: 169–76.
- Weiss AP. Radial shortening. Hand Clin. 1993;9: 475–82.
- Schattenkerk ME, Nollen A, van Hussen F. The treatment of lunatomalacia. Radial shortening or ulnar lengthening? Acta Orthop Scand. 1987;58(6):652–4.
- De Smet L, Fabry G. Orientation of the sigmoid notch of the distal radius. Acta Orthop Belg. 1993;59: 269–72.
- Sagerman SD, Zogby RG, Palmer AK, Werner FW, Fortino MD. Relative articular inclination of the distal radioulnar joint: a radiographic study. J Hand Surg [Am]. 1995;20:597–601.
- Horii E, Garcia-Elias M, Bishop AT, Cooney WP, Linscheid RL, Chao EY. Effect on force transmission across the carpus in procedures used to treat Kienböck's disease. J Hand Surg. 1990;15 Am:393–400.
- Werner FW, Palmer AK. Biomechanical evaluation of operative procedures to treat Kienböck's disease. Hand Clin. 1993;9:431–43.
- 44. Wada A, Miura H, Kubota H, Iwamoto Y, Uchida Y, Kojima T. Radial closing wedge osteotomy for Kienböck's disease: an over 10 year clinical and radiographic follow-up. J Hand Surg. 2002;27 Br:175–9.
- 45. Garcia-Elias M, An KN, Cooney WP, Linscheid RL. Lateral closing wedge osteotomy for treatment of Kienböck's diseas. A clinical and biomechanical study of the optimum correcting angle. Chir Main. 1998;17:283–90.
- Watanabe K, Nakamura R, Horii E, Miura T. Biomechanical analysis of radial wedge osteotomy for the treatment of Kienböck's disease. J Hand Surg. 1993;18A:686–90.

- Illarranmendi AA, Schulz C, De Carli P. The surgical treatment of Kienböck's disease by radius and ulna metaphyseal core decompression. J Hand Surg. 2001;26 Am:252–60.
- 48. Schulz C, De Carli P, Anetzberger H, Illarramendi A. Stress osteotomy of the distal radius- and ulna metaphysis (Illarramendi procedure): an alternative treatment method in lunate necrosis. Handchir Mikrochir Plast Chir. 1998;30:188–95.
- Watson HK, Hempton R. Limited wrist arthrodesis. 1 The triscaphoid joint. J Hand Surg Am. 1980;5:320–7.
- 50. Sauerbier M, Tränkle M, Erdmann D, Menke H, Germann G. Functional outcome with scaphotrapeziotrapezoid arthrodesis in the treatment of Kienböck's disease stage III. Ann Plast Surg. 2000;44:618–25.
- Watson HK, Monacelli DM, Milford RS, Ashmead IV D. Treatment of Kienböck's disease with scaphotrapeziotrapezoid arthrodesis. J Hand Surg. 1996;21A:9–15.
- 52. Minami A, Kimura T, Suzuki K. Long-term results of Kienböck's disease treated by triscaphe arthrodesis and excisional arthroplasty with a coiled palmaris longus tendon. J Hand Surg. 1994;19 Am:219–28.
- Tambe AD, Trail IA, Stanley JK. Wrist fusion versus limited carpal fusion in advanced Kienbock's disease. Int Orthop. 2005;29:355–8.
- Sennwald GR, Ufenast H. Scaphocapitate arthrodesis for the treatment of Kienböck's disease. J Hand Surg. 1995;20 Am:506–10.
- Oishi SN, Muzaffar AR, Carter PR. Treatment of Kienbock's disease with capitohamate arthrodesis: pain relief with minimal morbidity. Plast Reconstr Surg. 2002;109:1293–300.
- 56. Wilhelm K, Hierner R, Brehl B. Callus distraction for progressive lengthening of the capitate bone after resection of the lunate bone in stage III lunate malacia. Surgical technique and 1 year results. Handchir Mikrochir Plast Chir. 1997;29:10–9.
- 57. Lu L, Gong X, Liu Z, Zhang Z. Capitate transposition to replace necrotic lunate bone with a pedicle for Kienbock's disease: review of 30 cases. Chin Med J (Engl). 2003;116:1519–22.
- Kilgus M, Weishaupt D, Künzi W, Meyer VE. Radioscapholunate fusion: long-term results. Handchir Mikrochir Plast Chir. 2003;35:317–22.
- Tambe A, Ali F, Trail I, Stanley J. Is radiolunate fusion a viable option in advanced Kienböck disease? Acta Orthop Belg. 2007;73:598–603.
- Sauerbier M, Kluge S, Bickert B, Germann G. Subjective and objective outcomes after total wrist arthrodesis in patients with radiocarpal arthrosis or Kienböck's disease. Chir Main. 2000;19:223–31.
- Swanson AB, de Groot Swanson G. Implant resection arthroplasty in the treatment of Kienböck's disease. Hand Clin. 1993;9:483–91.
- Kaarela OI, Raatikainen TK, Torniainen PJ. Silicone replacement arthroplasty for Kienböck's disease. J Hand Surg. 1998;23 Br:735–40.
- De Smet L, Robijns P, Degreef I. Proximal row carpectomy in advanced Kienbock's disease. J Hand Surg. 2005;30 Br:585–7.

- 64. Iwasaki N, Minami A, Oizumi N, Suenaga N, Kato H, Minami M. Radial osteotomy for late-stage Kienböck's disease. Wedge osteotomy versus radial shortening. J Bone Joint Surg. 2002;84 Br:673–7.
- Nakamura R, Horii E, Watanabe K, Nakao E, Kato H, Tsunoda K. Proximal row carpectomy versus limited wrist arthrodesis for advanced Kienböck's disease. J Hand Surg. 1998;23 Br:741–5.
- Begley B, Engber W. Proximal row carpectomy in advanced Kienböck's disease. J Hand Surg. 1994;19 Am:1016–8.
- Preiser G. Eine typische posttraumatische und zur Spontanfraktur fuhrende Osteitis des Naviculare carpi. Fortschr Geb Roentgenstr. 1910;15:189–97.
- Gelberman R, Menon J. The vascularity of the scaphoid bone. J Hand Surg Am. 1980;5:508–13.
- Taleisnik J, Kelly P. The extraosseous and intraosseous blood supply of the scaphoid bone. J Bone Joint Surg. 1966;48 Am:1125–37.
- Vidal M, Linscheid R, Amadio P, Dobyns J. Preiser's disease. Ann Hand Up Limb Surg. 1991;10:227–36.
- Herbert TJ, Lanzetta M. Idiopathic avascular necrosis of the scaphoid. J Hand Surg. 1994;19 Br:174–82.
- Kaleinov D, Cohen M, Hendrickx RW, Sweet S, Culp R, Osterman A. Preiser's disease: identification of two patterns. JHand Surg. 2003;28 Am:767–8.
- Parkinson R, Noble J, Bale R, Freemont AJ. Rare abnormalities of the scaphoid in association with congenital radial ray defects of the hand. J Hand Surg. 1991;16 Br:208–11.
- 74. De Smet L, Aerts P, Walraevens M, Fabry G. Avascular necrosis of the carpal scaphoid: Preiser's disease. Report of 6 cases and review of the literature. Acta Orthop Belg. 1993;59:139–42.
- Alnot JY, Frayman JM, Bocquet L. Les ostéonécroses aseptiques primitives totales du scaphoïde. A propos

de 3 cas. Ann Chir Main Membre Super. 1990;9: 221–5.

- Moran S, Cooney W, Shin A. The use of vascularized grafts from the distal radius for the treatment of Preiser's disease. J Hand Surg. 2006;31 Am:705–10.
- Jönsson G. Aseptic necrosis of the os capitatum. Acta Radiol. 1942;23:562–4.
- Dereudre G, Kaba A, Pansard E, Mathevon H, Mares O. Avascular necrosis of the capitate: case report and a review of the literature. Chir Main. 2010;29:203–6.
- Milliez PY, Kinh Kha H, Allieu Y, Thomine JM. Idiopathic aseptic osteonecrosis of the capitate bone. Literature review apropos of 3 new cases. Int Orthop. 1991;15:85–94.
- Murakami H, Nishida J, Ehara S, Furumachi K, Shimamura T. Revascularization of avascular necrosis of the capitate bone. Am J Roentgenol. 2002;179: 664–6.
- Por Y, Chew W, Tsou I. Avascular necrosis of the triquetrum. Hand Surg. 2005;10:91–4.
- Match R. Non-specific avascular necrosis of the pisiform bone. J Hand Surg. 1980;5 Am:341–2.
- Olah J. Bilateral aseptsche Nekrose des Os Pisiforme. Z Orthop Ihre Grenzgeb. 1968;140:590.
- Garcia-Lopez A, Cardoso Z, Ortega L. Avascular necrosis of the trapezium bone: a case report. J Hand Surg. 2002;27 Am:704–6.
- Struzegger M, Mencarelli F. Avascular necrosis of the trapezoid bone. J Hand Surg. 1998;23B:550–1.
- De Smet L. Avascular necrosis of multiple carpal bones. Ann Chir Main Memb Super. 1999;18:202–4.
- Dieterich's H. Subchondrale Herderkrankung am Metacarpale III. Arch Klin Chir. 1932;171:555–67.
- Wright T, Dell P. Avascular necrosis and vascular anatomy of the metacarpals. J Hand Surg. 1991; 16-Am:540–4.

Part II

Contractures

Dupuytren's Disease

10

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Keywords

Dupuytren's Disease • Limited fasciectomy • Dermofasciectomy •
Percutaneous needle fasciotomy • Needle aponeurotomy • Collagenase •
Radiotherapy • Dupuytren Diathesis • Demographics • Etiology
• Prevalence • History • Grading and Staging • Anatomy • Recurrence •
Rehabilitation • Splints

Introduction – Brief Overview of the Importance of This Topic

Dupuytren's Disease (DD) is a benign fibromatosis of palmar fascias of the hand, which gives rise to the formation of nodules and cords and often leads to the development of flexion contractures of finger joints and web space contractures. The ring and little finger ray are most commonly affected, but the disease may involve all rays and the way the rays are affected may vary considerably. The same is true for the age at which the first symptoms occur. Generally the disease starts in between 50 and 60 years of age. Males are at younger age more affected than females, but with increasing age the incidence between the genders seems to approach equality in the ninth decade.

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The reported prevalence has by Hindocha et al. been found to vary between 0.2 and 56 %, but systematic metaanalysis of all published work on this topic by Lanting et al. has revealed that not all studies have been executed without bias and that a more realistic range is from 0.6 to 31.6 [1, 2] Incidence figures are scarce. In 1991, and based on referral to hand centres, it was estimated that the incidence of DD in the UK was 32.5/100,000/year and that 20.5/100,000/year were operated [3]. The disease is most prevalent in Caucasians from the North-Western part of Europe and their offspring, but also affects other races [4–6].

Clinical Pearl

- Dupuytren Disease is a chronic disease and can at present not be cured
- Dupuytren Disease has a multifactorial etiology in which genetic load, certain concomitant diseases and environmental factors interact

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- Various more or less invasive treatments are available and each of them has its own short and long term advantages and disadvantages
- Treatment should be tailored to the disease characteristics and the demands of the patient

Associated fibromatoses are Ledderhose Disease (LD), which affects the soles of the feet, causing painful nodules hindering walking but not contractures, and Peyronie Disease (PD), which encompasses the formation of plaques on the dorsum of the penis, leading to penile curvature and sometimes causing problems with sexual intercourse. In addition to, or as part of DD, there may also be fibromatosis at the dorsum of the PIPJ's, leading to the formation of knuckle or Garrod pads [7].

DD generally does not disturb finger flexion, although it may cause or be related to trigger finger [8]. DD may lead to inconveniences when the fingers become bent. Often heard complaints are the inability to put on gloves and to put the hand in a pocket, and patients often say that they have difficulty washing their faces because of the bent fingers. They also report embarrassment during hand shaking. Besides, the contracture may lead to functional impairment and interfere with job specific tasks.

Hueston coined the term Dupuytren Diathesis for patients in whom he found that the disease starts early, affects both hands and more family members, and gives rise to knuckle pads [9]. In the last decade two groups of researchers have suggested modification of the characteristics list. Abe et al., after studying a Japanese population, amended the Hueston criteria with previous surgery for affection of the little finger and radial involvement of the thumb, and Ledderhose Disease, while Hindocha et al. were of the opinion that male sex, age of onset younger than 50 and – of the ectopic lesions – only knuckle pads play a role as diathesis characteristics [10, 11]. Patients that meet these criteria usually experience a more aggressive course of disease, with more recurrences and disability.

At present, the disease cannot be cured and will almost always recur after treatment if time permits, or extend into previously unaffected areas. The purpose of this chapter is to review the current knowledge of the various aspects of this disease together with the advantages and disadvantages of the available treatment modalities.

At this stage there is a paucity of randomized clinical trials with sufficient follow up to warrant definitive conclusions, but at the end of this chapter some evidence based and personal guidelines for treatment are postulated.

History

The origin of Dupuytren's Disease has intrigued many authors. After a very elegant anthropological study on the migration of mankind over Europe, Robert MacFarlane came to the conclusion that a genetic mutation had occurred between 1200 BC, when both Celtic and Germanic tribes lived in northern Europe, and 200 BC when the age of migrations began, most likely created DD [12].

The first written report from the European Continent on what we now know as Dupuytren Disease dates back to 1614, when Felix Plater from Basel in Latin described his observations of the palm in a stone-mason with a contracture, that he falsely thought was caused by the tendons that had been ruptured from their sheaths [13]. However, Whaley and Elliot found earlier reports of four miracle cures set in Orkney and Iceland in the twelth and thirteenth Centuries, recorded in the sagas of the Earls of Orkney and the Bishops of Iceland, in which the condition of the hand which was healed had a resemblance to Dupuytren Disease [14].

Henri Cline of London was in 1777 (the year Baron Guillaume Dupuytren was born) the first to take notes of dissections of hands with the disease and the one who clearly understood that DD is an affection of the palmar fascias and not one of the underlying tendons. He also was the first to suggest palmar fasciotomy as a treatment for DD. Elliot, whom therefore feels that the disease should have been named Cline's Disease, reconstructed that

Astley Cooper in 1822 was probably the first to actually perform this procedure [13]. Dupuytren's name became connected to this disease because of the lecture he gave on this subject on 5 December 1831 and because of the efficient system that existed in Paris in those days and enabled the fast spreading of new findings. In this lecture he stressed that this disease originates from the palmar aponeurosis and he also demonstrated the open palmar fasciotomy [15]. In 1934 Goyrand questioned Dupuytren's opinion that Dupuytren's Disease was caused by contraction of the palmar aponeurosis only, since he had observed fibrous bands that lie superficial from the aponeurosis and which also extended into the fingers and in his opinion were responsible for metacarpophalangeal joint (MCPJ) and proximal interphlangeal joint (PIPJ) contracture [16]. For this reason he advised longitudinal incisions instead of transverse, which allowed for the removal instead of division of pathological tissue while preserving vital structures. This theory proved to be true and this operation was in essence the first description of the technique that many hand surgeons nowadays still employ, and which we know as selective or limited fasciectomy (LF).

In the first half of the twentieth century, surgeons like Lexer in Germany, May in the USA and McIndoe in Great Britain believed that, for the sake of prevention of recurrence, it was prudent not only to remove the diseased tissue, but also all healthy fascia in the palm and fingers [17]. This radical fasciectomy fell into disgrace, since it was hampered by many complications without preventing recurrences, and it drove surgeons back to less extensive procedures like limited faciectomy [18].

At the end of the 1970s rheumatologists in Paris made the pendulum of treatment swing back completely, when they popularised percutaneous fasciotomy using disposable needles [19]. In the 1990s, Badalamente and Hurst from Stony Brook University in New York reported their first experiments on the use of collagenase derived from Clostridium Histolyticum, which in recent years has resulted in the development of an injection therapy for DD [20]. In that same era, a number of studies from Germany appeared on the ability of radiotherapy to slow down disease progression when in an early phase [21].

Etiology

The etiology of DD has not been fully elucidated. However, a combination of genetic and environmental factors, such as smoking and alcohol abuse, seems to be involved. Besides, the disease occurs more frequently when other diseases are present such as diabetes mellitus, liver disease or when drugs for epilepsy are used [22, 23]. Some have found a relation to manual work [24] but others, such as Khan et al. in 2004, were unable to show such a relation, but found that the incidence rates in the elderly were highest in the non-manual social classes [25]. A recent meta-analysis does support the hypothesis of an association between high levels of exposure to manual labour and vibration (Odds Ratio (OR) for manual work: 2.0 [1.6; 2.6] and for vibration exposure: 2.9 [1.4;6.1]) and the development of Dupuytren Disease in certain cases [26].

As far as evidence for a genetic basis for the disease is concerned: a number of population studies has shown that the disease has an autosomal dominant trait with variable penetrance and the group of Bayat has performed a series of candidate genes association studies, however without revealing causal genes [27, 28]. Dolmans et al. have in 2011 published a Genome Wide Association Study using DNA samples of almost 2,500 patients and 11,500 controls, which identified nine regions in the genome that are strongly associated with DD [29]. In six of these regions genes are located that are part of the WNT-signalling pathway, a genetic pathway that also has been indicated to play a role in Dupuytren's Disease by others [30]. In an additional study a correlation between the genetic load and the presence of certain diathesis factors come to light [31].

Presentation and Investigation, Pathogenesis and Histology

The first manifestations of DD may be very subtle subcutaneous irregularities in the palm (Fig. 10.1). These may be difficult to discern, even for the experienced eye and easily confused with other diseases or skin aberrations.

It becomes easier to diagnose the disease when nodules start to appear and become bigger, firstly in the distal palm in the vicinity of the transverse palmar creases, and later in the finger or at the MCPJ area of the thumb. The skin usually



Fig. 10.1 Right hand of a patient from India with early signs of DD in the ring finger ray. There is a cord at the level of the transverse palm crease and an early stage of dimpling half a centimeter distal to it

becomes adherent just distal to these nodules where fibres insert into it, and skin pits are the earliest signs of contraction of some of these affected fibres as is blanching of the skin during extension. Nevertheless, the differential diagnosis of Dupuytren's Disease in that stage still is extensive and should include any hand condition that causes nodules or pits: it encompasses ganglia and inclusion cysts, occupational hyperkeratosis, callous formation, tenosynovitis, giant cell tumours, and epitheloid sarcoma [32]. The natural progression is from nodules and pits to cords. Once these develop and contractures emerge, the diagnosis usually is evident (Fig. 10.2), although there is still a differential diagnosis of dermal contracture as in (burn) scars, congenital conditions such as camptodactyly, stuck trigger fingers, bowstringing following damage to the pulley system as in rock climbers, tendon adhesions following infection or repair and intrinsic joint contractures following trauma. The speed

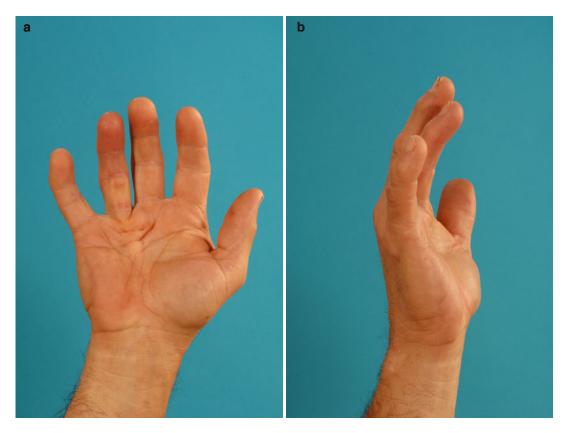


Fig. 10.2 Dupuytren's Disease of ring finger of right hand in 60 year old male. (a) palmar view, (b) ulnar view. Photographs taken in maximal active extension

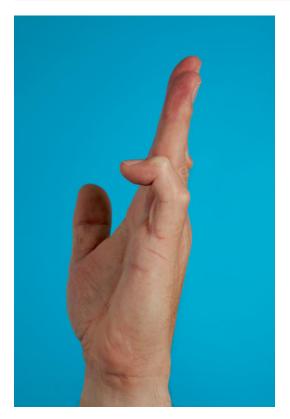


Fig. 10.3 Dupuytren's Disease of little finger with DIPJ-contracture

at which the disease progresses from nodules to cords to contractures has not been researched extensively. However, it is the author's impression that it is more likely to follow an exponential course than a linear one. Cords often overlay tendons in the palm and can be found central or radial or ulnar in the finger and may extend to the distal interphalangeal joint (DIPJ). In most cases cords ultimately cause contractures, most of the time affecting the metacarpophalangeal joints (MCPJ) and/or proximal interphalangeal joints (PIPJ) or the web spaces. The disease only seldom leads to contractures of the DIPJ (Fig. 10.3). More often the DIPJ is forced into hyperextension, giving rise to a Boutonnière deformity of the affected finger (Fig. 10.4). There is usually bilateral involvement, although there is no synchronicity between both hands. The ring finger and little finger ray are most often affected, but DD may cause pathology in any ray (Fig. 10.5).

In the initial phase, the nodules may be painful when compressed, but this sign usually disappears with time. Cords are almost always insensate, and therefore percutaneous needle fasciotomy can be performed using local anaesthesia that is only applied in minimal quantities to numb the skin [33].

Histologically, three phases have already been distinguished by Luck in 1959:

- a proliferative phase, in which nodules prevail and there is abundance of proliferating fibroblast, that are not yet aligned along lines of stress relative to the amount of collagen;
- an involutional phase, in which the number of cells declines but alignment along lines of stress becomes obvious, and;
- a residual phase, when there are hardly any cells left and the tissue is scar-like [34].

The cells found in the pathologic tissue are specialised fibroblasts, and have been found to express α -smooth muscle actin that is known from smooth muscles cells of vascular origin and therefore have been named myofibroblasts [35]. A lot of basic research has been devoted to this cell type in DD, and it is clear now that the key to the development of the disease is the inappropriate behaviour of these myofibroblasts. Contracture formation starts with the contraction of individual myofibroblasts and is followed by the secretion of collagen type I and III and other extracellular matrix (ECM) molecules, which subsequent stabilize the contracture, allowing the myofibroblast to relax again and repeat the cycle [36, 37]. Stiffness of the ECM has been found to be crucial in myofibroblast behaviour and reseachers in the field at present direct all their attention to the identification of modulating factors of this process [38]. Parallel to this research, clinicians like Tyrkko and Viljanto have tried to correlate histopathological findings with the disease pattern and have come to the conclusion that the presence of nodules with abundance of proliferating cells (Type I disease according to Luck) is a predictor for early recurrence and this has recently been confirmed by Balaguer et al. [39, 40] Iqbal et al. have recently found that mesenchymal stem cells from the perinodular fat and the overlying skin are a potential source for myofibroblasts [41].

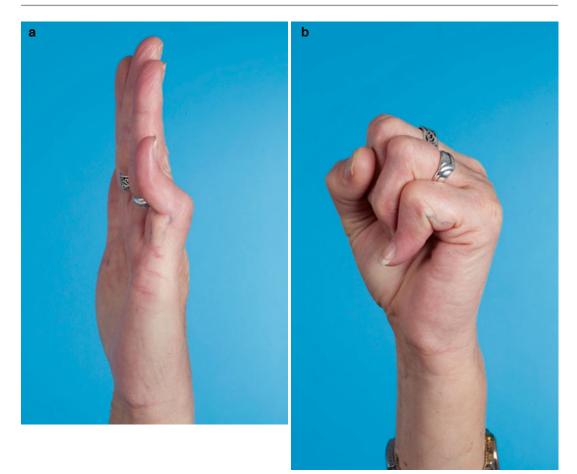


Fig. 10.4 Patient with DD of left little finger with Boutonnière deformity (a) maximal extension, (b) maximal flexion



Fig. 10.5 Bilateral disease with variable phenotype

Grading and Staging

The most practical grading systems for research purposes are those of Iselin, and Tubiana and Michon. Iselin described in 1955 a classification system that distinguishes four stages (Table 10.1): Stage 1: DD nodules and cords without contracture; Stage 2: MCPJ contractures; Stage 3: MCPJ and PIPJ contracture; Stages 4: Boutonnière deformity with MCPJ and PIPJ contracture and DIPJ hyperextension [42]. His staging system is very practical for quick scoring such as during prevalence studies [43]. The clinically most widely used staging system for Dupuytren's Disease was originally described by Tubiana and Michon in 1961 and later refined by Tubiana [44]. This system classifies the findings in each ray in one of six stages, depending on the absence of any disease (Stage 0), presence of nodules without contractures (Stage N) or the total amount of flexion contracture in degrees as measured using a goniometer on the dorsal side of the joints, increased by the amount of hyperextension at DIPJ (Stage I: 0–45°; Stage II: 46–90°; Stage III: 91–135°; Stage IV: 135° or more) (Table 10.1). On top, the letters P (palm), D (digit) and H (hyperextension of DIPJ) may be added to indicate the location of disease as well as the presence of hyperextension. A further sophistication has been suggested by Tubiana in 1986 that makes the system very complex, and therefore this is unfortunately not frequently employed [45]. In the German literature on the use of radiotherapy for DD, one may find a further refinement of Stage N, a Stage N/I, which corresponds with a very early Stage I contracture of only 5°. The downside of this system is that progression into a higher stage may take $0-40^{\circ}$ and is therefore not linear.

In 1998 Woodruff and Waldram published a very practical grading system to plan surgery (Table 10.2). It basically reflects the complexity of the deformity in minutes anticipated to be necessary to correct it and can be used for operating time planning [46].

Table 10.1 Grading systems for Dupuytren Disease:

 every ray is given a stage number

Described by	Stage	Meaning
Iselin 1		DD nodules and cords without contracture
	2	MCPJ contractures
	3	MCPJ and PIPJ contracture
	4	Boutonnière deformity with MCPJ and PIPJ contracture and DIPJ hyperextension
Tubiana	0	No signs of Dupuytren Disease
	Ν	Nodules only. No contracture
	Ι	Total Passive extension deficit (TPED) smaller than 45°
	II	TPED in between 46–90°
	III	TPED in between 91° and 135°
	IV	TPED in between 136° and 180°

Table 10.2 Grading systems for Dupuytren Disease

 based on estimated operating time needed

Woodruff and Waldram	Description	Approximate surgery time
	 Finger contracture only, hyperextends at MCP joint, hand lies flat on table 	None
	2 Single finger pre-tendinous cord, MCP joint contracture only (Possible day case)	30 min
	3 Single finger pre-tendinous band, MCP joint and PIP joint contracture	60 min
	4 As 3 but two-finger contracture	90 min
	5 Finger stuck in palm, suitable only for amputation	30 min

Surgical Anatomy of the Palmar Fascia

The fibromatosis that we know as Dupuytren's Disease takes it effect in or near some fascial structures in the hand. When looking form a historic perspective, Cleland, Weitbrecht, Grapow, Legueu

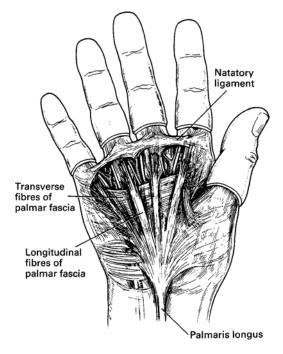


Fig. 10.6 The palmar fascia (Reprinted with permission from Elliott and McGrouther [109], Figure 8.2)

and Juvara, Grayson and his professor Wood Jones, Gosset, Milford, White, Skoog, Stack, McFarlane, McGrouther and Zancolli all have made important contributions to our current understanding of fascial structures of the hand. Both McFarlane and McGrouther have to be credited for placing the structures relevant for DD in perspective and relating them to what we may expect at surgery [47, 48].

In the palm the normal fascial structures run in three different planes and directions: longitudinally just underneath the skin, transversely in various planes, and sagittally, linking superficial structures to deep ones. The longitudinal fibres are organized in a fan shape and together form the palmar aponeurosis (Fig. 10.6). It has its apex over the carpal tunnel, where it may have a connection to the palmaris longus tendon and its base at the base of the fingers and can be divided in pretendinous and prelumbrical bands, depending on their location. The pretendinous bands usually are more condensed than the prelumbrical bands and all pass superficial to the transverse ligament of the palmar aponeurosis (TLPA), which is situated at a line that joins the proximal and distal

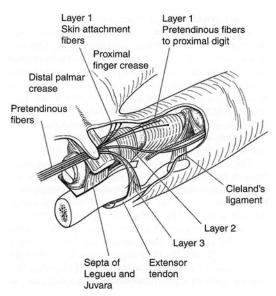


Fig. 10.7 Division of pretendinous band in three layers (Reprinted with permission from Elliott and McGrouther [110])

palmar crease and was described by Skoog [49]. The TLPA ulnarly blends with the hypothenar fascia and is on its radial end is continuous with the proximal commissural band, which runs through the first web space and ends in the thenar fascia at the level of the first metacarpophalangeal joint. Just beyond TLPA, the pretendinous bands divide into three layers (Fig. 10.7): (a) Layer 1 is the most superficial one, which has its insertion in the skin of the distal palm. Some Layer 1 fibres run into the finger and insert into the palmar skin of the proximal phalanx; (b) Layer 2 fibres form the so called "spiral band", since they spiral from their origin at the pretendinous band underneath the neurovascular bundle towards the lateral side of the fingers. This band is present on both sides in all long fingers, except for the ulnar side of the little finger; (c) Layer 3 is the deepest layer of fibres. These fibres dive deep into the hand on both sides of the flexor tendon sheath, to insert on either side of the corresponding metacarpophalangeal joint and may play a role in causing trigger finger symptoms as an early sign of DD.

Another important transverse fascial structure, in addition to TLPA, is the Natatory ligament (NL). This ligament lies just underneath the skin

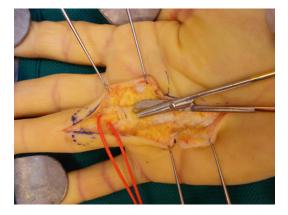


Fig. 10.8 Exposure of DD in ring finger ray. The scissors are placed underneath Natatory ligament. The vessel loop holds the radial neurovascular bundle

in the distal palm and is situated superficial to the neurovascular bundles (Figs. 10.6 and 10.8). On its ulnar end it is continuous with the hypothenar fascia, and on the radial side with the distal commissural band. NL has attachments to the flexor tendon sheath, lines the webs and has extensions along the lateral sides of the digits, where it blends with fibres from the spiral band and is continuous with the lateral digital sheet.

Vertical or sagittal ligaments of importance in the palm are the septa described in the late 1800s by the French couple Legueu and Juvara that are usually just as wide as TLPA and connect the longitudinal fibres at the level of the TLPA with the deep transverse palmar ligament that connects the volar plates of all MCPJ's except the thumb (Fig. 10.7). On cross section the TLPA and the deep transverse ligament, together with the ligaments of Legueu and Juvara form nine boxes, which on alternating basis hold the lumbrical muscles together with the neurovascular bundles, and the flexor tendons of each ray. Much smaller vertical fibres are dispersed all over the palmar aponeurosis and anchor it to the overlying skin. These fibres were discovered and named after Grapow at the end of the nineteenth century.

The digital fascias that are of most importance bear the names of Cleland and Grayson. Cleland published his work in 1878 and Grayson in 1941 [50, 51]. However, their detailed anatomical description underwent significant changes while it passed through history: drawings depicting the exact course of their fibres have been conflicting (Fig. 10.9). The only certainty today is that there is a structure located volar to the neurovascular bundle, which is known as Grayson's ligament, and a ligamentous structure that lies dorsal to these vital structures, which bears the name of Cleland. Further detailed dissection and studies of these structures are necessary to reveal their exact anatomy as well as of what is known as the lateral digital sheet and retro-vascular sheet. Of interest in this respect is the recent work of Guimberteau who investigated and described the fine trabecular network of fibres that is responsible for allowing tissue gliding and might play a yet undisclosed role in the pathogenesis of Dupuytren's Disease [52]. At the place where the hypothenar muscles pass into the little finger, there is a cross roads of fibres that run in all six directions and all these fibres seem to play a role in Dupuytren's Disease [53]. At the thenar and in the first webspace there is also a complex three dimensional network of bands than can become involved (Fig. 10.10).

Already in the days of Dupuytren, there was disagreement about the origin of Dupuytren's Disease. As said before, some authors, including Dupuytren himself, believed that the disease starts within the fascia. Others, like Goyrand in 1833, reported that they felt that it originates in the tissues in between the skin and the fascias [54]. This dispute has never been resolved. Of some of the aforementioned fascial structures it is certain that they play a role in the disease (Table 10.3), but of others it is yet not fully elucidated. If a well defined structure becomes affected by disease, it is no longer called a "band" but becomes a "cord".

Skoog has stated that the transverse ligament of the palmar aponeurosis (TLPA) is never affected by DD [55]. Since this structure lies volar to the neurovascular bundles but dorsal to the pretendinous and prelumbrical cords, it can be left behind and as such protects them during secondary surgery when pretendinous cords are no longer there. It can therefore be used as a reference point during subsequent surgery. Distal to TLPA however, the dissection always needs to be performed with great care, because the neurovascular bundle may be displaced

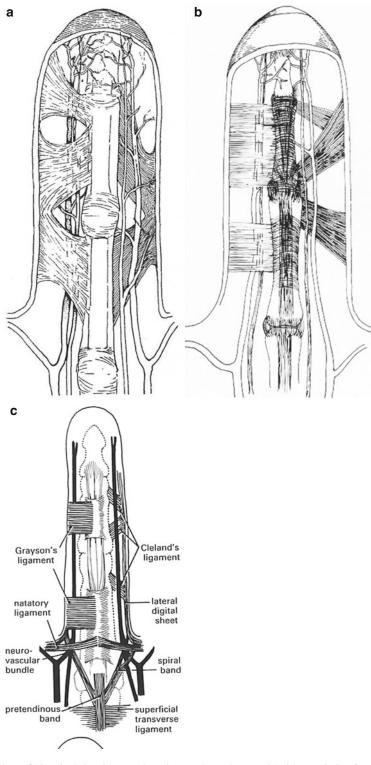


Fig. 10.9 Overview of historical drawings on the orientation of Grayson's and Cleland's ligaments in the finger. Grayson's ligament is shown on the left side in each drawing and situated volar to the neurovascular bundle. Cleland's ligament is shown on the right, dorsal to the neurovascular bundle. (a) Copy of Grayson's findings as

drawn by Wood (With permission from Grayson [51]). (b) Depiction of Milford's findings in 1968 (From Milford [111]), (c) shows the currently most widely used figure based on McFarlane's drawing of 1972 which is an adaptation of Milfords findings (Reprinted with permission from McFarlane [112], Figure 14A)

by the pathology and a sound knowledge of the surgical anatomy is mandatory to prevent damage to it. Especially a spiral band that transforms into a spiral cord can displace the neurovascular bundle medially and volarly and may become so intimately related to it, that only the experienced eye can discern it. Short and Watson have described a warning sign for such a situation: if there is a flexion contracture of the MCPJ and subcutaneous fat is present in between the cord and the skin distal to TLPA, there should rise a high suspicion for a spiral cord [56]. In such a situation, the neurovascular bundle usually does not regain its natural position until just distal to the proximal interphalangeal joint. Recently Tonkin has described a number of cases where a double spiral around the digital nerve had been encountered [57].

McFarlane has in the 1970s tried to simplify the quite often complex phenotype of the disease in the finger and has come up with three patterns of disease: the central cord, the lateral cord and the spiral cord (Fig. 10.11). Further work is needed to be able to explain all findings, including those of Tonkin, during surgery.

Treatment Modalities, Complications of Treatment and Outcome

As said before, the pendulum of surgical treatment has described a complete swing form open fasciotomy in the early 1800s, dictated by the lack of proper anaesthesia, to radical surgery in an – as it appeared fruitless – attempt to prevent extension and recurrence, back to minimally invasive percutaneous needle fasciotomy, this time finding a place because of its limited burden. In between the extremes of the pendulum's swing, treatments of intermediate aggressiveness such as selective or limited fasciectomy of pathology only, and segmental fasciectomy, a procedure in which only small segments of the cords are removed, can be found. In addition, some have advocated injection therapy of painful nodules with steroids [58], or collagenase derived from Clostridium Histolyticum to weaken or dissolve

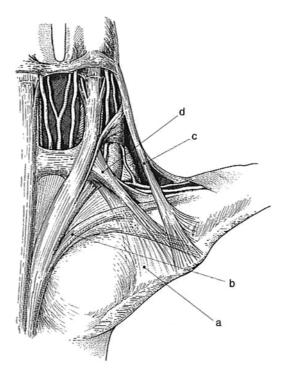


Fig. 10.10 Fascial structures of the thumb and first web space; *a* thenar muscle fascia; *b* pretendinous band; *c* distal commissural band; *d* proximal commissural band (Reprinted with permission from Tubiana [113])

part of a cord [20], and radiotherapy have been developed [59]. Finally researchers have tested whether pharmacological intervention with substances such as fluorouracil on top of surgery may reduce the chances for recurrence. In this section an overview of all treatment modalities will be given.

In the discussion of these treatment strategies, a number of aspects of each treatment will be addressed in order to get a good overview: the way the method manages the affected and adjacent tissues, more specifically: skin, fascia and joints; indication for and best timing of treatment; specific treatment risks as well as complications; immediate outcome and time of disability; late outcome including definition and rate of recurrence and extension; additional treatment options. But first, a short description of the most commonly performed treatment modalities will be given.

Anatomical structure. Full name and (abbreviation)	Name if affected by DD Full name and (abbreviation)	Clinical relevance
Fascial structures in the palm		
Pretendinous band (PTB) Prelumbrical band (PLB)	Pretendinous cord (PTC) Prelumbrical cord (PTC)	Situated immediately beneath the skin in palm. Responsible for earliest signs of disease in most patients. Divides in three layers distal to transverse ligament of palmar aponeurosis
Layer 1 of PTB	Pretendinous cord in palm; Central cord in finger	Situated immediately beneath the skin, distal to line joining proximal and distal palm crease. May cause MCPJ and PIPJ contracture. Does not displace nv-bundle
Layer 2 of PTB (spiral band)	Spiral cord	Contracture causes displacement of nv-bundle medially and palmarly. May cause MCPJ contracture. Warning sign: Short-Watson sign (see text)
Layer 3 of PTB	Vertical cord	Dives deep into the hand on both sides of MCPJ. May cause painful triggering
Transverse Ligament of Palmar Aponeurosis (TLPA)	-	Runs along line joining proximal and distal palm crease, deep to PTB/PLB. No affected by DD. Can be left behind durin fasciectomy. Will facilitate subsequent surgery when left intact
Ligaments of Legeue and Juvara	-	Connect TLPA to Deep transverse ligament
Natatory ligament (NTL)	Natatory cord	Situated immediately beneath the skin an superficial to nv-bundle. May cause web contracture
Proximal/distal commissural band	Proximal/distal commissural cord	Proximal band is extension of TLPA; distal band is ectension of NTL. Situated immediately beneath the skin in the first web; may cause first web contracture
Abductor digiti minimi fascia	Abductor digiti minimi cord (ADMC)	Forms Y-shape together with pretendinou band and ulnar nv bundle can always be found proximal to junction
Deep transverse ligament (DTL)	-	Connects volar plates of MCPJ. Together with Ligaments of Legueu and Juvara and TLPA forms nine boxes through which flexor tendons and lumbrical muscles and nv-bundles pass
Fibres of Grapow	-/nodules in palm?	Dispersed over palmar aponeurosis. Anchor palmar skin to TLPA
Fascial structures in the digits		
Lateral digital sheet	Lateral digital cord	Situated immediately under the skin on each side of each finger; may displace nv-bundle towards midline. May cause MCPJ and PIPJ contracture when involved together with spiral cord and Grayson's ligament inserting to A4 pulley. May also cause DIPJ contracture as lateral cord
Grayson's ligament	Central cord (CC)	Harbours nodules and cord at proximal

 Table 10.3
 Various fascial structures in palm and in fingers of interest in Dupuytren's Disease

Table IU.5 (continued)	Table	10.3	(continued)
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Anatomical structure. Full name and (abbreviation)	Name if affected by DD Full name and (abbreviation)	Clinical relevance
Cleland's ligament	-/Retrovascular cord??	Not easily visualised, since situated behind nv-bundle. May cause PIP and DIPJ contracture
Transverse retinacular ligament	-	Connects mid slip of the extensor apparatus at PIPJ tot the lateral bands and the volar plate. Palmar portion may shorten in severe PIPJ contractures, preventing the lateral bands to slide back after contracture release
Oblique retinacular ligament (Landsmeer)	-	Connects volar plate of PIPJ to dorsal capsule of DIPJ. May shorten and cause Boutonnière deformity

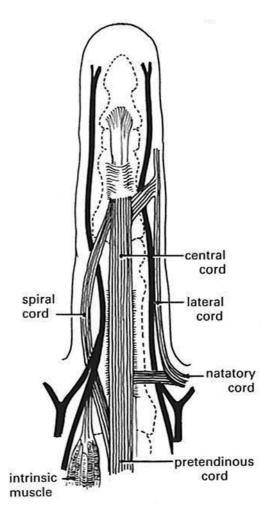


Fig. 10.11 Digital cords (Reprinted with permission from McFarlane [114]; Figure 14.6B, p 159)

Treatment Modalities

To relieve pain in early stage (Stage N) DD, patients may be advised to wear biking gloves, which are fingerless and lined with a silicone pad at the palm that disperses pressure. Although not always practical, this may alleviate the discomfort experienced during power grip, until it disappears, which usually happens over the subsequent 6 months.

Radical fasciectomy (RF) is only listed here for historic reasons. It was employed in the 1950s and early 1960s, and encompassed the complete removal of all fascias in the hand and fingers. Its popularity was short lived, since it was hampered by many more complications than limited fasciectomy, particularly hematoma formation and skin slough [60].

Limited or selective fasciectomy (LF) is still the most commonly performed surgical procedure for DD: only the diseased tissue is removed, at the palm usually with a small margin of normal fascia (Fig. 10.12). The generally accepted indication for treatment is a progressive flexion contracture of $20-30^{\circ}$ of any joint or a painful nodule that does not respond to conservative measures. This situation corresponds with what most hand surgeons advise patients; to seek treatment when one can no longer put the affected hand flat on the table (Hueston table top test) [61]. If a PIPJ is contracted more than 60° ,



Fig. 10.12 Example of limited fasciectomy of the little finger. Same patient as in Fig. 10.4. A pretendinous cord, in the finger running as central cord was excised

it has been found difficult to surgically redress it completely.

Segmental fasciectomy (SF) is a variation of LF in which only small, 1 cm long pieces of diseased tissue at strategic places are removed. This method was described by Moermans of Louvain in the 1990s and surgeons from this group still are strong advocates of this technique [62, 63]. As with all surgical techniques for DD, MCPJ contractures can be very well corrected, but the technique is less successful for PIPJ contractures and the amount of complications is at least as high as of LF [64].

In the three aforementioned methods, the surgeon needs to manage the skin too. McGrouther has reported on the almost endless number of surgical approaches that have been published using incisions in almost any direction [17]. When there is no or only limited shortage of skin as in contractures of limited extent, its management usually is straightforward: A longitudinal incision from palm to finger, a Bruner type incision, or a transverse incision at the level of TLPA and at the joint creases or a combination of incisions, all allow for the removal of the pathology. For SF, small C-shaped incisions are used.

McCash has shown that following the management of fascia using transverse incisions, skin shortage in the palm can be left alone, as long as bare tendons are not exposed [65]. For the fingers, one or more Z-plasties or YV-plasties can be employed in the longitudinal or zig-zag incisions to gain extra length for closure. Z-plasties can best be planned between the proximal finger crease and the PIPJ crease, since at that level there is usually the greatest abundance of skin due to skin expansion by the underlying nodules.

If skin transposition is not enough, or if skin is so affected that it is decided to be prudent to be removed (dermofasciectomy), it can be replaced using skin grafts. Skin grafts have also been advocated as firebreaks and in some surgeon's hands have proved very successful in the prevention of recurrence [66]. Dermofascietomy (DF) is otherwise used by most surgeons for the treatment of recurrences, especially when the skin is affected, and for primary cases with diathesis, since some researchers have shown that recurrences are more delayed following skin replacement [67]. Skin grafts are usually taken from the ipsilateral extremity or the groin and have full dermal thickness. Donor sites are closed primarily but leave scars, one of the down sides of this treatment. Others are: increased duration of the procedure, prolonged rehabilitation period and increased risk for complications: a graft may not take, extending the recovery period.

Apart from grafts, a great variety of local (homo- or heterodigital) or regional flaps can be used to manage skin shortage. As an example, Jacobsen has introduced a modification of the McCash method for fasciectomy of the little finger, which – in addition to the transverse palmar incision – employs a midlateral ulnar incision for access to the little finger [68]. Once the pathological tissue has been removed and the finger joints can be extended, the flap is allowed to slide distally, leaving a palmar defect only that will heal secondarily as in the McCash technique.

With LF an average reduction of Total Passive Extension Deficit (TPED) of 79 % at 6 weeks post-operatively has been achieved. Results were best at the MCPJ (87 % reduction of Passive Extension Deficit (PED)) and worst at the PIPJ (49 % reduction of PED) [69]. Barton reported that the surgical prognosis of Dupuytren's disease of the PIPJ's is worst in the little finger [70]. The extent of the contracture has been found to be a clear predictor of ultimate outcome [71]. Abe et al. found in a Japanese series a direct correlation between the amount of PIPJ contracture and the ultimate outcome: in contractures of $0-30^{\circ}$ the mean percentage of improvement was 76 %, in contractures of $30-60^{\circ}$ 48 %, and if a flexion contracture of the PIPJ had been more than 60° , the percentage of improvement was only 25 [10].

Specific complications of LF, SF and DF include skin slough, that usually can be treated conservatively, hematoma formation, that needs to be evacuated before settling and leading to fibrosis, and Chronic Regional Pain Syndrome (CRPS), which is the most difficult to treat and may result in a stiff hand in extension with a severe flexion lag, that is functionally sometimes worse than the flexion deformity the patient seeked help for. Nerve injury occurs in approximately 1 % of primary and up to 7 % in recurrent cases and should be handled by direct repair, if feasible and will then lead to good results [72]. Surgery may also cause injury to the digital arteries, the incidence of which is most likely underreported. In a worst case scenario, especially after repeated surgery, vascular injury may result in gangrene of the finger, necessitating partial or complete amputation of a finger. The cumulative complication rate for LF has been found to be 19 % and even higher in DF and RF and the mean recovery period to normal hand use after surgery is on average 6–8 weeks [73]. During this period, the achieved result at operation will gradually improve [69].

For persistent PIPJ-contractures at the end of LF, some authors advise the sequential transverse division of the flexor tendon sheath, the check-rein ligaments of the volar plate and the release of the accessory collateral ligaments, until full release of the PIPJ contracture is accomplished. Breed and Smith however have observed best and sustainable results by diligent manual manipulation towards extension of the PIPJ [74]. Attenuation of the central slip of the extensor may also be a significant factor in the inability to

sustain correction of PIPJ contracture [75]. Messina and Messina and co-workers in a series of papers showed that continuous traction on a cord by means of an external fixator was able to reduce Tubiana III and IV contractures to such an extent that fasciectomy became easier [76]. This reduction appeared to be caused by a change in collagen cross-link profile as the result of a degradation of collagen and increase in newly synthesized collagen under influence of MMP, gelatinase and acidic cathepsins [77]. In a more sophisticated way distraction can now be delivered using the Digit Widget[™] [78]. In a very recent study Craft et al. have in PIPJ contractures compared the effect of preoperative soft tissue distraction using the Digit Widget[™] and subsequent LF with LF combined with check-rein ligament release [79]. All PIPJ contractures pre-treated by the Digit Widget[™] achieved significantly better results, and, this result was most pronounced in the contractures of over 60°.

Following LF, SF and DF, the treated hand is usually put in a soft bulky dressing for a week, sometimes reinforced by a plaster of Paris. Following graft take (if applicable) and suture removal, patients are usually stimulated to start practising flexion and extension and this process is often supervised by hand therapists, who may also fit a night splint.

All above mentioned procedures need to be performed under regional or general anaesthesia. This is one of the eye catching differences with the following less invasive treatment techniques. Needle aponeurotomy (NA), also named percutaneous needle fasciotomy (PNF), is one of the least invasive procedures available for DD treatment. During this procedure, that can be performed using local anesthesia, cords are percutaneously divided by the use of a needle or a narrow scalpel, in French known as "bistory" [33]. This treatment modality is akin to that employed at the beginning of the nineteenth century in the era of Cooper and Dupuytren, although nowadays the skin is not cut, but only punctured. Its initial variant remained popular until the end of the nineteenth century, then disappeared to be reinvented by a Parisian group of Rheumatologists at the end of the 1970s,

when disposable needles became available that were sharp enough to be used as a fasciotome. PNF may be performed at multiple levels along the cords during the same session [33]. Since cords are insensate, only very small amounts of local anaesthesia need to be injected and only intra-dermally at the site of needling to allow the procedure. By doing this, nerve conduction can be maintained during the procedure enabling the patient to warn the surgeon whenever he accidently hits a nerve. The treatment is very effective for MCPJ, but less so for PIPJ contractures. Outcome of PNF is similar as for LF in the lower Tubiana Stages (I and II), but significantly less successful in the higher Stages (III and IV) [69]. Complications are limited to skin tears and the risk to damage nerves or tendons is very small. The cumulative risk of serious complication of PNF is much less than that of the more invasive treatments and this is one of the reasons for its popularity [80]. Skin fissures may occur and with adherent skin, as in recurrent cases, are almost the rule. Since they heal without leaving a trace, they usually do not bother the patient. PNF is at present one of the most popular treatment modalities amongst patients on internet forums because of its limited invasiveness, impressive results and since most patients experience a very fast return of hand function and are usually back to normal activity much quicker than after LF [69].

Steroid injection is also advocated by some to relieve pain and discomfort that may be an early sign of the disease, when only nodules in the palm are present [58]. It is also a treatment method often used for patients with Ledderhose's Disease [81]. However, the injections may be very painful and regional or even general anaesthesia is often necessary to be able to inject enough medicine to obtain a good result. Some authors advocate to combine PNF with steroid injection, but the evidence that supports this is weak [82, 83].

The newest injection treatment modality employs two types of collagenase derived from the microorganism Clostridium Histolyticum, which have together been found to be a powerful tool to dissolve pathological DD tissue. 0.58 mg of the drug (Xiaflex in the USA, Canada and Australia or Xiapex in Europe, Auxillium Pharmaceuticals, Malvern, PA, and SoBi, Stockholm, Sweden) is injected into the palpable cord that is responsible for a joint contracture of at least 20°, and 24 h later the treated joint is extended using light force to rupture it. The rupturing can be painful and may be performed under local anaesthesia. If the obtained result is not satisfactory, the treatment may be repeated after 30 days. A disadvantage of collagenase treatment when using it according to the label is that only one cord may be treated at a time, although recently the successful results of multiple injections have been published [84].

This treatment has been carefully developed by Dr's Hurst and Badalamente in a series of both in vitro and in vivo experiments over the last 20 years. Clinically, since the beginning of this century, a number of Phase II and III studies has been performed and published. Injection with collagenase was in double blinded studies found to be significantly more effective than placebo to release contractures [20, 85]. In 77 % of the MCPJ and in 40 % of the PIPJ collagenase released the contracture to 0-5°. General serious adverse events (SAE's) have not been reported, but local SAE's have in 97 % of cases. In this Phase III study in 1 % of cases tendon ruptures occurred. A modification of the technique has been able to significantly reduce and almost eliminate this complication. The most frequently occurring local SAE is swelling of the hand, which may be so profound that it mimics infection. This is one of the main reasons for the application of a soft and bulky dressing. Digital nerves and arteries are not at risk for being dissolved, since their collagen constitution differs from that of the pathological cords (and tendons). Since usually more than one injection is necessary per ray (or even joint) and since the drug is expensive, additional studies are needed to prove its cost effectiveness.

Early in the twenty-first century, radiotherapy (RTX) emerged as a treatment modality for DD and for some time, results have been conflicting [59, 86]. Subsequently more cohort studies have appeared in the German and English literature, but all from the same institute at Erlangen of from other centres but in all occasions by radiotherapists that where trained there, with follow up at time points with increasing duration. The current protocol is as follows: the affected area, including a margin is irradiated in two sessions of five times in a week, 6–8 weeks apart with a total of 30 Gy. After 3 months, the authors found that disease was stable in 92 % of Tubiana N, N/I and I cases, had regressed in 7 % and progressed in 1 %. In 75 % a non-significant reduction in size and consistency was found [87]. Complications related to the treatment are only minor late toxicity (skin atrophy, dry desquamation) in 32 % of the patients. In the publications on radiotherapy it is stated that post-radiotherapy recurrences have been surgically treated without a higher incidence of complications [21]. However, in order to be able to properly position radiotherapy as treatment for early DD, randomized clinical trials are needed in which radiotherapy in randomized against no treatment.

Long Term Outcome: Recurrence and Extension

There is a wide disparity in the used definitions for recurrence on one hand and only a very limited number of randomized studies that compare the various treatment modalities on the other, let alone with sufficient follow up [88]. The basic issue regarding the definition of recurrence is that the treatment modalities are so different. As explained before, some remove diseased tissue only (LF and DF), and some all substrate for DD (RF), some just segments of it (SF), while others only divide (PNF), dissolve (collagenase) or try to arrest progression (RTX). In some studies a recurrence is defined as any sign of new tissue in a previously operated spot [89], while others, like the group of Hurst and Badalamente and ourselves, have a more indirect but more quantitative way of expressing recurrence: we measure the extension deficit of each joint (PED) and add those together to get the total passive extension deficit (TPED) and define recurrence as an increase of TPED to a certain number of degrees (20° in the collagenase studies and 30° in most other studies) [20, 69]. In a review on this topic, in which recurrence was defined as reappearance of deformity necessitating additional surgery (i.e. a positive table top test), it was summarized that the mean recurrence rate after LF was 15 % after a mean of 27 months [90]. Recently the results of our RCT comparing LF and PNF with a follow up duration of 5 years have become available. Following LF the recurrence rate was 21 % [91]. Others, using the LeClercq definition, have published recurrence figures of up to 73 % after 7 years [92].

DF has been advocated by most for the treatment of recurrences in which the skin is also severely affected, and by some as a principal method to prevent recurrence in patients with DD diathesis by installing fire breaks. In this respect the work of Ketchum is interesting [93]. He applied DF in 68 such patients and could provide follow-up of 36 hands of 24 patients with an average duration of almost 4 years. He found no recurrent disease in the palms and digits that were covered with the full-thickness grafts. The incidence of extension outside the grafts was 8 %. Armstrong et al. have reported similar positive results: in cases in which there was clear skin involvement, they replaced this by a full thickness graft and found a recurrence rate of only 8.4 % of the treated rays (n=143) after almost 6 years follow up [67]. Other authors however have not been able to achieve similar results, and in a very well executed RCT on the benefit of dermofasciectomy and skin grafting, no difference in recurrence rate was found at 3 years post surgery. In both groups, which were otherwise comparable, the recurrence rate at the PIPJ was 12.2 % [94]. The recurrence rates of radical fasciectomy appear similar to those of LF and will not be discussed in detail since this treatment is hampered by so many complications [18]

PNF recurrence rates are relatively high compared to more aggressive treatment modalities. After three years, Foucher, just as we have, found recurrence rates in approximately 60 % of cases and after 5 years our recurrence rates, defined as an increase of TPED as compared to the 6 weeks result of more than 30°, had increased to 85 % [91, 95, 96]. Hovius et al. are in the process of testing if this high recurrence rate can be reduced by adding grafted fat by means of lipofilling to the cord that has been extensively divided. Long term results are unfortunately not available yet [97]. The recurrence rate (increase of PED in completely redressed joints of more than 20°) in the collagenase studies is 19 % after 2 years and 37 % after 3 years [98]. At present there is only one very small study with long term follow up (8 years) data of only eight patients. In 67 % of the MCPJs and 100 % of the PIPJ recurrence was found [99].

The progression rates of radiotherapy is as follows: After a mean of 10 years, 87 % of the patients that had been treated with Tubiana Stage N and 70 % of the patients treated while in Tubiana Stage N/I remained stable or regressed. In more advanced stages, the rate of disease progression increased to 62 % (Stage I) or 86 % (Stage II). Sixty-six percent of the patients showed a long-term relief of initially reported side effects (i.e., burning sensations, itching and scratching, pressure and tension) [21]. The basic issue that needs to be elucidated, however, is the time versus progression relation in Dupuytren's Disease. In my clinical experience this relation is more likely exponential than linear and since radiotherapy has only been found to be effective in early stages of the disease, it is unclear what it is actually being accomplished. There is one study available in which a control group is introduced with long follow up that shows favourable results for radiotherapy. From that study it cannot be deduced what were the criteria for each treatment arm [100]. Besides, hand surgical follow up studies are needed to report on the difficulties encountered at subsequent surgery and on the occurrence of complications.

Some patients experience recurrence earlier than others. From our randomised clinical trial in which we compared PNF and LF it became clear that there is a strong correlation between age at the moment of treatment and chance for recurrence [91]. This is probably the most important diathesis characteristic that influences recurrence. In an effort to prevent or postpone recurrence, especially in such cases, researchers have found that drugs such as tamoxifen and 5-fluorouracil in vitro were able to delay fibroblast proliferation [101, 102]. Subsequently some added these measures to their treatment regimen. Systemic treatment with 5-Fluorouracil in an RCT unfortunately proved ineffective [103]. In a similar design, Degreef et al. found that highly dosed neo-adjuvant tamoxifen improved the surgical outcome of segmental fasciectomy by achieving a better finger extension and a high patients' satisfaction. The same group has successfully attempted to delay recurrence by placing cellulose implants in the gap created by segmental fasciectomie [104].

Treatment of Recurrence

Evidence for the best treatment for recurrence is scarce and there is a need for comparative studies in this respect. What is known is that there is a greater risk of complications during limited fasciectomy: Coert et al. found an incidence of accidental nerve injury of 7 % in recurrent cases as compared to 1 % primary cases [72]. PNF can also be used for the treatment of recurrences [105]. With skin involvement and skin scarring the likelihood for a skin fissure or rupture becomes greater and more effort needs to be directed to the release of the pathology from the skin [33].No studies have yet been reported on the use of collagenase in recurrences and the same is true for radiotherapy.

Rehabilitation

As stated before, splinting and hand therapy are commonly advised in the treatment of Dupuytren's Disease after surgery. However, until a few years ago there was no evidence as to the benefit of this regimen and a great variety of protocols are being used [106]. Herweijer et al. studied if hand therapy following surgery led to better results and could not prove this [107]. Recently the results of an RCT designed to test the benefit of splinting in addition to hand therapy in patients with a PIP contracture of at least 30° have been published [108]. The authors found no benefit of routine additional night splint therapy. They are of the opinion that only patients with rapidly reoccurring extension deficits benefit form night splints.

Conclusions/Personal View

Dupuytren's Disease is a chronic disease for which at present a cure is lacking. Since the turn of the century the body of knowledge on the basics of the disease, especially on a genetic and molecular level, has increased substantially. Besides, new treatment modalities have emerged and gradually high quality trials are emerging in the literature that compare treatment regimen. At present no clear cut evidence based treatment algorithm can be drawn. However, in the development of a personal view, the application of an evidence based approach is necessary and for some subjects possible [22]. No treatment at present is universally the best for every patient with DD. Following counselling the patient about all surgical and non-surgical treatment modalities, I advise him or her primarily based on (1) age, and (2) progressiveness of disease. In the most extreme forms a young (<40 years) patient with aggressive disease with PIP contractures will be warned for early recurrence and offered limited fasciectomy or dermofasciectomy and skin grafting (based on the work of Armstrong and Ketchum), while an old patient (>75 years) with contracture and mild progression will be advised percutaneous needle fasciotomy. Early stage (Tubiana N and I) patients are hopefully in the near future offered radiotherapy in randomized clinical trial setting in an attempt to elucidate the true role of this treatment modality. All others are offered either PNF or limited fasciectomy, stating that PNF is less invasive and thus gives less trouble after treatment on the short run, but is hampered by earlier recurrence, with a very clear age-recurrence relation.

Collagenase has only been available in my clinic for 1.5 years, since we acted as a training centre. Early results were comparable to those of others, but I do not have long term results [20]. Nevertheless, I believe it is an interesting addition to the treatment armamentarium and its place is especially dependent on its long term recurrence rate, and on its costs which have yet to be determined.

For recurrence, the choice for the best treatment is even more difficult. We have recently shown that recurrences after PNF can effectively be treated again by PNF [105]. Only if the fibromatosis is very adherent to the skin, it may become difficult to achieve an acceptable result without skin rupture. If the patients accept skin rupture and a small flap or graft to close the defect, it can be offered. When PNF is no longer possible or desired, LF is the next step for me. Given the increasing body of evidence that splinting cannot prevent recurrence, I advise hand therapy only for all those with extensive disease, primarily to help regain flexion as soon as possible.

References

- Hindocha S, McGrouther DA, Bayat A. Epidemiological evaluation of Dupuytren's disease incidence and prevalence rates in relation to etiology. Hand (N Y). 2009;4(3):256–69.
- Lanting R, Broekstra DC, Werker PM, van den Heuvel ER. A systematic review and meta-analysis on the prevalence of Dupuytren Disease in the general population of western countries. Plast Reconstr Surg. 2014;133(3):593–603.
- Burke FD, Dias JJ, Lunn PG, Bradley M. Providing care for hand disorders: trauma and elective. The Derby Hand Unit experience (1989–1990). J Hand Surg Br. 1991;16(1):13–8.
- Abe Y, Rokkaku T, Ofuchi S, Tokunaga S, Takahashi K, Moriya H. Dupuytren's disease on the radial aspect of the hand: report on 135 hands in Japanese patients. J Hand Surg Br. 2004;29(4):359–62.
- Slattery D. Review: Dupuytren's disease in Asia and the migration theory of Dupuytren's disease. ANZ J Surg. 2010;80(7–8):495–9.
- Sladicka MS, Benfanti P, Raab M, Becton J. Dupuytren's contracture in the black population: a case report and review of the literature. J Hand Surg. 1996;21(5):898–9.
- Garrod AE. Concerning pads upon the finger joints and their clinical relationships. Br Med J. 1904; 2(2270):8.
- Kuehlein B. The influence of Dupuytren's Disease on trigger fingers and vice versa. In: Eaton C, Seegenschmiedt MH, Bayat A, Gabbiani G, Werker PMN, Wach W, editors. Dupuytren's Disease and related hyperproliferative disorders. 1st ed. Berlin/ Heidelberg: Springer; 2012. p. 249–54.
- 9. Hueston JT. Recurrent Dupuytren's contracture. Plast Reconstr Surg. 1963;31:66–9.
- Abe Y, Rokkaku T, Ofuchi S, Tokunaga S, Takahashi K, Moriya H. Surgery for Dupuytren's disease in Japanese patients and a new preoperative classification. J Hand Surg Br. 2004;29(3):235–9.
- Hindocha S, Stanley JK, Watson S, Bayat A. Dupuytren's diathesis revisited: evaluation of prognostic indicators for risk of disease recurrence. J Hand Surg Am. 2006;31(10):1626–34.
- McFarlane RM. On the origin and spread of Dupuytren's disease. J Hand Surg Am. 2002;27(3):385–90.
- Elliot D. The early history of contracture of the palmar fascia. Part 1: the origin of the disease: the curse of the MacCrimmons: the hand of benediction: Cline's contracture. J Hand Surg Br. 1988;13(3):246–53.
- Whaley DC, Elliot D. Dupuytren's disease: a legacy of the north? J Hand Surg Br. 1993;18(3):363–7.

- Elliot D. The early history of contracture of the palmar fascia. Part 2: the revolution in Paris: Guillaume Dupuytren: Dupuytren's disease. J Hand Surg Br. 1988;13(4):371–8.
- Elliot D. The early history of contracture of the palmar fascia. Part 3: the controversy in Paris and the spread of surgical treatment of the disease throughout Europe. J Hand Surg Br. 1989;14(1):25–31.
- McGrouther DA. Treatment. In: McFarlane RM, McGrouther DA, Flint MH, editors. Dupuytren's Disease, biology and treatment. Edinburgh/London/ Melbourne/New York: Churchill Livingstone; 1990. p. 295–310.
- Dickie WR, Hughes NC. Dupuytren's contracture: a review of the late results of radical fasciectomy. Br J Plast Surg. 1967;20(3):311–4.
- Lermusiaux JL, Debeyre N. Le traitement médical de la maladie de Dupuytren. Rev Rhum Mal Osteoartic. 1977;44(11):633–8.
- Hurst LC, Badalamente MA, Hentz VR, Hotchkiss RN, Kaplan FT, Meals RA, et al. Injectable collagenase clostridium histolyticum for Dupuytren's contracture. N Engl J Med. 2009;361(10):968–79.
- Betz N, Ott OJ, Adamietz B, Sauer R, Fietkau R, Keilholz L. Radiotherapy in early-stage Dupuytren's contracture. Long-term results after 13 years. Strahlenther Onkol. 2010;186(2):82–90.
- Brandt KE. An evidence-based approach to Dupuytren's contracture. Plast Reconstr Surg. 2010; 126(6):2210–5.
- Tripoli M, Cordova A, Moschella F. Dupuytren's contracture as result of prolonged administration of phenobarbital. Eur Rev Med Pharmacol Sci. 2011; 15(3):299–302.
- Mikkelsen OA. Dupuytren's disease the influence of occupation and previous hand injuries. Hand. 1978;10(1):1–8.
- Khan AA, Rider OJ, Jayadev CU, Heras-Palou C, Giele H, Goldacre M. The role of manual occupation in the aetiology of Dupuytren's disease in men in England and Wales. J Hand Surg Br. 2004;29(1):12–4.
- Descatha A, Jauffret P, Chastang JF, Roquelaure Y, Leclerc A. Should we consider Dupuytren's contracture as work-related? A review and meta-analysis of an old debate. BMC Musculoskelet Disord. 2011;12:96.
- 27. Bayat A, Stanley JK, Watson JS, Ferguson MW, Ollier WE. Genetic susceptibility to Dupuytren's disease: transforming growth factor beta receptor (TGFbetaR) gene polymorphisms and Dupuytren's disease. Br J Plast Surg. 2003;56(4):328–33.
- Bayat A, Watson JS, Stanley JK, Ferguson MW, Ollier WE. Genetic susceptibility to Dupuytren disease: association of Zf9 transcription factor gene. Plast Reconstr Surg. 2003;111(7):2133–9.
- Dolmans GH, Werker PM, Hennies HC, Furniss D, Festen EA, Franke L, et al. Wnt signaling and Dupuytren's disease. N Engl J Med. 2011;365:307–17.
- Degreef I, De Smet L, Sciot R, Cassiman JJ, Tejpar S. Immunohistochemical evidence for Zic1 coexpression with beta-catenin in the myofibroblast of

Dupuytren disease. Scand J Plast Reconstr Surg Hand Surg. 2009;43(1):36–40.

- Dolmans GH, de Bock GH, Werker PM. Dupuytren diathesis and genetic risk. J Hand Surg Am. 2012; 37(10):2106–11.
- Rayan GM. Clinical presentation and types of Dupuytren's disease. Hand Clin. 1999;15(1):87–96, vii.
- Eaton C. Percutaneous fasciotomy for Dupuytren's contracture. J Hand Surg Am. 2011;36(5):910–5.
- Luck JV. Dupuytren's contracture; a new concept of the pathogenesis correlated with surgical management. J Bone Joint Surg Am. 1959;41-A(4): 635–64.
- Gabbiani G, Majno G. Dupuytren's contracture: fibroblast contraction? An ultrastructural study. Am J Pathol. 1972;66(1):131–46.
- Follonier Castella L, Gabbiani G, McCulloch CA, Hinz B. Regulation of myofibroblast activities: calcium pulls some strings behind the scene. Exp Cell Res. 2010;316(15):2390–401.
- Tomasek JJ, Gabbiani G, Hinz B, Chaponnier C, Brown RA. Myofibroblasts and mechano-regulation of connective tissue remodelling. Nat Rev Mol Cell Biol. 2002;3(5):349–63.
- Hinz B, Gabbiani G. Fibrosis: recent advances in myofibroblast biology and new therapeutic perspectives. F1000 Biol Rep. 2010;2:78.
- Tyrkko J, Viljanto J. Significance of histopathological findings in Dupuytren's contracture. Ann Chir Gynaecol Fenn. 1975;64(5):288–91.
- 40. Balaguer T, David S, Ihrai T, Cardot N, Daideri G, Lebreton E. Histological staging and Dupuytren's disease recurrence or extension after surgical treatment: a retrospective study of 124 patients. J Hand Surg Eur Vol. 2009;34(4):493–6.
- 41. Iqbal SA, Manning C, Syed F, Kolluru V, Hayton M, Watson S, et al. Identification of mesenchymal stem cells in perinodular fat and skin in Dupuytren's disease: a potential source of myofibroblasts with implications for pathogenesis and therapy. Stem Cells Dev. 2012;21(4):609–22.
- 42. Iselin M. The forms of Dupuytren's disease. Concours Med. 1955;77(51):4769–71.
- Degreef I, De Smet L. A high prevalence of Dupuytren's disease in Flanders. Acta Orthop Belg. 2010;76(3):316–20.
- Tubiana R, Michon J, Thomine JM. Scheme for the assessment of deformities in Dupuytren's disease. Surg Clin North Am. 1968;48(5):979–84.
- Tubiana R. Evaluation of deformities in Dupuytren's disease. Ann Chir Main. 1986;5(1):5–11.
- Woodruff MJ, Waldram MA. A clinical grading system for Dupuytren's contracture. J Hand Surg Br. 1998;23(3):303–5.
- McGrouther DA. The microanatomy of Dupuytren's contracture. Hand. 1982;14(3):215–36.
- McFarlane RM. Patterns of the diseased fascia in the fingers in Dupuytren's contracture. Displacement of the neurovascular bundle. Plast Reconstr Surg. 1974; 54(1):31–44.

- Skoog T. Dupuytren's contracture: pathogenesis and surgical treatment. Surg Clin North Am. 1967;47(2): 433–44.
- Cleland J. On the cutaneous ligaments of the phalanges. J Anat Phys. 1878;12:526.
- Grayson J. The cutaneous ligaments of the digits. J Anat. 1941;75(2):164–5.
- Guimberteau JC, Delage JP, McGrouther DA, Wong JK. The microvacuolar system: how connective tissue sliding works. J Hand Surg Eur Vol. 2010;35(8): 614–22.
- White S. Anatomy of the palmar fascia on the ulnar border of the hand. J Hand Surg Br. 1984;9B:51.
- Goyrand G. Nouvelles recherches sur la re'traction permanente des doigts. Mem Acad R Med. 1833;3:489.
- Skoog T. Transverse elements of the palmar aponeurosis in Dupuytren's contracture. Scand J Plast Reconstr Surg. 1967;1:51.
- Short WH, Watson HK. Prediction of the spiral nerve in Dupuytren's contracture. J Hand Surg Am. 1982;7(1):84–6.
- Hettiaratchy S, Tonkin MA, Edmunds IA. Spiralling of the neurovascular bundle in Dupuytren's disease. J Hand Surg Eur Vol. 2010;35(2):103–8.
- Ketchum LD, Donahue TK. The injection of nodules of Dupuytren's disease with triamcinolone acetonide. J Hand Surg Am. 2000;25(6):1157–62.
- Herbst M, Regler G. Dupuytren's contracture. Radiotherapy in the early stages. Strahlentherapie. 1985;161(3):143–7.
- Orlando JC, Smith JW, Goulian D. Dupuytren's contracture: a review of 100 patients. Br J Plast Surg. 1974;27(3):211–7.
- Hueston JT. Table top test. Med J Aust. 1976;2(5): 189–90.
- Moermans JP. Segmental aponeurectomy in Dupuytren's disease. J Hand Surg Br. 1991;16(3):243–54.
- Moermans JP. Long-term results after segmental aponeurectomy for Dupuytren's disease. J Hand Surg Br. 1996;21(6):797–800.
- Clibbon JJ, Logan AM. Palmar segmental aponeurectomy for Dupuytren's disease with metacarpophalangeal flexion contracture. J Hand Surg Br. 2001;26(4):360–1.
- McCash CR. The open palm technique in Dupuytren's contracture. Br J Plast Surg. 1964;17:271–80.
- Ketchum LD. The use of the full thickness skin graft in Dupuytren's contracture. Hand Clin. 1991;7(4):731–41; discussion 743.
- Armstrong JR, Hurren JS, Logan AM. Dermofasciectomy in the management of Dupuytren's disease. J Bone Joint Surg Br. 2000;82(1):90–4.
- Jacobsen K, Holst-Nielsen F. A modified McCash operation for Dupuytren's contracture. Scand J Plast Reconstr Surg. 1977;11(3):231–3.
- 69. van Rijssen AL, Gerbrandy FS, Ter Linden H, Klip H, Werker PM. A comparison of the direct outcomes of percutaneous needle fasciotomy and limited fasciectomy for Dupuytren's disease: a 6-week follow-up study. J Hand Surg Am. 2006;31(5):717–25.

- Barton NJ. Dupuytren's disease arising from the abductor digiti minimi. J Hand Surg Br. 1984;9(3): 265–70.
- Donaldson OW, Pearson D, Reynolds R, Bhatia RK. The association between intraoperative correction of Dupuytren's disease and residual postoperative contracture. J Hand Surg Eur Vol. 2010;35(3): 220–3.
- Coert JH, Nerin JP, Meek MF. Results of partial fasciectomy for Dupuytren disease in 261 consecutive patients. Ann Plast Surg. 2006;57(1):13–7.
- McFarlane RM, McGrouther DA. Complications and their management. In: McFarlane RM, McGrouther DA, Flint MH, editors. Dupuytren's disease: biology and treatment. Edinburgh: Churchill Livingstone; 1990. p. 377–82.
- Breed CM, Smith PJ. A comparison of methods of treatment of pip joint contractures in Dupuytren's disease. J Hand Surg Br. 1996;21(2):246–51.
- Smith P, Breed C. Central slip attenuation in Dupuytren's contracture: a cause of persistent flexion of the proximal interphalangeal joint. J Hand Surg Am. 1994;19(5):840–3.
- Citron N, Messina JC. The use of skeletal traction in the treatment of severe primary Dupuytren's disease. J Bone Joint Surg Br. 1998;80(1):126–9.
- Bailey AJ, Tarlton JF, Van der Stappen J, Sims TJ, Messina A. The continuous elongation technique for severe Dupuytren's disease. A biochemical mechanism. J Hand Surg Br. 1994;19(4):522–7.
- Goss BC. Summary of literature on skeletal fixation devices for flexion contractures of the PIP joint. 2002. http://www.handbiolab.com/wp-content/themes/handbiolab/files/digitwidget/DW%20Lit%20Summary. pdf.
- Craft RO, Smith AA, Coakley B, Casey 3rd WJ, Rebecca AM, Duncan SF. Preliminary soft-tissue distraction vs checkrein ligament release after fasciectomy in the treatment of Dupuytren PIP joint contractures. Plast Reconstr Surg. 2011;128: 1107–13.
- Badois FJ, Lermusiaux JL, Masse C, Kuntz D. Nonsurgical treatment of Dupuytren disease using needle fasciotomy. Rev Rhum Ed Fr. 1993;60(11): 808–13.
- Pentland AP, Anderson TF. Plantar fibromatosis responds to intralesional steroids. J Am Acad Dermatol. 1985;12(1 Pt 2):212–4.
- McMillan C, Binhammer P. Steroid injection and needle aponeurotomy for Dupuytren contracture: a randomized, controlled study. J Hand Surg Am. 2012;37(7):1307–12.
- Broekstra DC, Werker PM. Steroid injections in combination with needle aponeurotomy as a treatment method for Dupuytren disease: suggestions for increasing the research evidence. J Hand Surg Am. 2012;37(11):2429–30; author reply 2430–1.
- Coleman S, Gilpin D, Tursi J, Kaufman G, Jones N, Cohen B. Multiple concurrent collagenase clostridium histolyticum injections to Dupuytren's cords: an

exploratory study. BMC Musculoskelet Disord. 2012;13:61. 2474-13-61.

- Gilpin D, Coleman S, Hall S, Houston A, Karrasch J, Jones N. Injectable collagenase Clostridium histolyticum: a new nonsurgical treatment for Dupuytren's disease. J Hand Surg Am. 2010;35(12):2027–38.e1.
- Weinzierl G, Flugel M, Geldmacher J. Lack of effectiveness of alternative non-surgical treatment procedures of Dupuytren contracture. Chirurg. 1993;64(6):492–4.
- Keilholz L, Seegenschmiedt MH, Sauer R. Radiotherapy for prevention of disease progression in early-stage Dupuytren's contracture: initial and long-term results. Int J Radiat Oncol Biol Phys. 1996;36(4):891–7.
- Werker PM, Pess GM, van Rijssen AL, Denkler K. Correction of contracture and recurrence rates of dupuytren contracture following invasive treatment: the importance of clear definitions. J Hand Surg Am. 2012;37(10):2095–2105.e7.
- Leclercq C, Tubiana R. Long-term results of aponeurectomy for Dupuytren's disease. Chirurgie. 1986;112(3):194–7.
- Dias JJ, Braybrooke J. Dupuytren's contracture: an audit of the outcomes of surgery. J Hand Surg Br. 2006;31(5):514–21.
- van Rijssen AL, ter Linden H, Werker PM. Five-year results of a randomized clinical trial on treatment in Dupuytren's disease: percutaneous needle fasciotomy versus limited fasciectomy. Plast Reconstr Surg. 2012;129(2):469–77.
- Jurisic D, Kovic I, Lulic I, Stanec Z, Kapovic M, Uravic M. Dupuytren's disease characteristics in Primorsko-goranska County, Croatia. Coll Antropol. 2008;32(4):1209–13.
- Ketchum LD, Hixson FP. Dermofasciectomy and fullthickness grafts in the treatment of Dupuytren's contracture. J Hand Surg Am. 1987;12(5 Pt 1):659–64.
- 94. Ullah AS, Dias JJ, Bhowal B. Does a 'firebreak' fullthickness skin graft prevent recurrence after surgery for Dupuytren's contracture?: a prospective, randomised trial. J Bone Joint Surg Br. 2009;91(3):374–8.
- Foucher G, Medina J, Navarro R. Percutaneous needle aponeurotomy: complications and results. J Hand Surg Br. 2003;28(5):427–31.
- van Rijssen AL, Werker PM. Percutaneous needle fasciotomy in Dupuytren's disease. J Hand Surg Br. 2006;31(5):498–501.
- Hovius SE, Kan HJ, Smit X, Selles RW, Cardoso E, Khouri RK. Extensive percutaneous aponeurotomy and lipografting: a new treatment for Dupuytren disease. Plast Reconstr Surg. 2011;128(1):221–8.
- Peimer CA, Blazar P, Coleman S, Kaplan FT, Smith T, Tursi JP, et al. Dupuytren contracture recurrence following treatment with collagenase clostridium histolyticum (CORDLESS study): 3-year data. J Hand Surg Am. 2013;38(1):12–22.
- Watt AJ, Curtin CM, Hentz VR. Collagenase injection as nonsurgical treatment of Dupuytren's disease: 8-year follow-up. J Hand Surg Am. 2010;35(4):534–9. 539.e1.
- 100. Seegenschmiedt MH. Long term outcome of radiotherapy for early stage Dupuytren's disease: a phase

III Clinical study. In: Eaton C, Seegenschmiedt MH, Bayat A, Gabbiani G, Werker PMN, Wach W, editors. Dupuytren's Disease and related hyperproliferative disorders. 1st ed. Berlin/Heidelberg: Springer; 2012. p. 349–72.

- 101. Kuhn MA, Wang X, Payne WG, Ko F, Robson MC. Tamoxifen decreases fibroblast function and downregulates TGF(beta2) in dupuytren's affected palmar fascia. J Surg Res. 2002;103(2):146–52.
- 102. Jemec B, Linge C, Grobbelaar AO, Smith PJ, Sanders R, McGrouther DA. The effect of 5-fluorouracil on Dupuytren fibroblast proliferation and differentiation. Chir Main. 2000;19(1): 15–22.
- 103. Bulstrode NW, Bisson M, Jemec B, Pratt AL, McGrouther DA, Grobbelaar AO. A prospective randomised clinical trial of the intra-operative use of 5-fluorouracil on the outcome of Dupuytren's disease. J Hand Surg Br. 2004;29(1):18–21.
- Degreef I, Tejpar S, De Smet L. Improved postoperative outcome of segmental fasciectomy in Dupuytren disease by insertion of an absorbable cellulose implant. J Plast Surg Hand Surg. 2011;45(3):157–64.
- 105. van Rijssen AL, Werker PM. Percutaneous needle fasciotomy for recurrent Dupuytren disease. J Hand Surg Am. 2012;37(9):1820–3.
- 106. Larson D, Jerosch-Herold C. Clinical effectiveness of post-operative splinting after surgical release of Dupuytren's contracture: a systematic review. BMC Musculoskelet Disord. 2008;9:104.
- Herweijer H, Dijkstra PU, Nicolai JP, Van der Sluis CK. Postoperative hand therapy in Dupuytren's disease. Disabil Rehabil. 2007;29(22):1736–41.
- 108. Jerosch-Herold C, Shepstone L, Chojnowski AJ, Larson D, Barrett E, Vaughan SP. Night-time splinting after fasciectomy or dermo-fasciectomy for Dupuytren's contracture: a pragmatic, multi-centre, randomised controlled trial. BMC Musculoskelet Disord. 2011;12(1):136.
- 109. Elliott PM, McGrouther DA. Dupuytren's disease. In: Smith P, editor. Lister's hand surgery: diagnosis and indication. 4th ed. London: Churchill Livingstone; 2002. p. 527.
- 110. Elliott PM, McGrouther DA. The Palm. In: McFarlane RM, MCGrouther DA, Flint MH, editors. Dupuytren's disease. Biology and treatment, The hand and upper limb series, vol. 5. Edinburgh: Churchill Livingstone; 1990. p. 127–35.
- 111. Milford LWJ. Retaining ligaments of the digits of the hand: gross and microscopic anatomical study. Philadelphia: W.B. Saunders Company; 1968.
- McFarlane RM. The anatomy of dupuytren's disease.
 In: Dupuytren's disease: biology and treatment. Edinburgh: Churchill Livingstone; 1990. p. 159.
- 113. Tubiana R. The hand. Philadelphia: WB Saunders; 1999. p. 436.
- 114. McFarlane RM. The finger. In: McFarlane RM, McGrouther DA, Flint MH, editors. Dupuytren's disease. Edinburgh/London/Melbourne/New York: Churchill Livingstone; 1990.

The Burned Hand

Juan P. Barret

Keywords

Burn hand • Frostbite • Hypertrophic scar • Burn blisters • Escharotomy • Silver sulfadiazine • Tangential excision • Dermal template • Flaps • Skin graft • Rehabilitation • Splinting • Silicone • Pressure garment

Introduction

Small burns and superficial burns are the most common injuries admitted to burn centres around around the world. In most centres, they account for more than 80–90 % of all admissions [1]. Thanks to prevention programs and the increasing awareness of society regarding burn injuries, the incidence of massive, life threatening burns is declining in the developed countries. However, they continue to be a public health concern throughout the world, especially in middle and low-income countries [2]. Many of them, however, represent major burns by the American Burn Association criteria because they usually are deep burns of hands, face, feet, perineum, and major joints [3] (Table 11.1). Deep minor burns, either deep partial thickness or full thickness burns, have significant morbidity in terms of time to healing, infective complications and subsequent

scarring (Fig. 11.1). Conservative management leading to spontaneous healing usually involves prolonged and painful dressing changes and the resultant scar is invariably hypertrophic leading to cosmetic and functional debility. Thus an early surgical approach that tries to preserve dermis and achieve wound healing is preferred.

Quality of life and improved outcomes are now more than ever an issue in modern societies.

Table 11.1 Criteria for transfer of a burn patient to a burn centre

econd-degree burns greater than 10 % total body urface area (TBSA)	
hird degree burns	
Burns that involve the face, hands, feet, genitalia, erineum, and major joints	
Chemical burns	
Electrical burns including lightning injuries	
ny burn with concomitant trauma in which the burn ijury poses the greatest risk to the patient	n
nhalation injury	
atients with pre-existing medical disorders that cou omplicate management, prolong recovery, or affect nortality	
Iospitals without qualified personnel or equipment and care of critically burned patient	for

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Fig. 11.1 Hand burns are categorised as major injuries by both the American Burn Association (ABA) and the European Burns Association (EBA). Typical aspect of a third degree contact burn with hot melted plastic. This injury resulted in the amputation of 3 digits

These can only be achieved by excellence in burn care [4]. Although surgery is the central treatment of minor deep burns, all members of the burn team are necessary to provide the best outcome and reintegration into society. The hands and the face are frequently involved in burn accidents. The functional repercussions of severe burns to the hands are obvious, and for this reason, we believe correct treatment is very important. It is considered that thermal damage to the hands, age, and percentage of burned body area requiring grafts are the determining factors of the patient's capability to return to his/her normal occupation [5]. Factors that increase the severity of burns of the upper limbs include bilateral involvement and aetiologies such as burns caused by flames, chemicals, or high-voltage electricity, deep burns involving the dorsum of the fingers, and burns that are circumferential and/or cross joints [6].

Initial Assessment and Topical Treatment

Initial assessment and stabilization of the burn victim follows the standard ABC of trauma assessment and a complete work-up as with any other burn victim. The reader is referred to other textbooks for the treatment and assessment of major burns. It should be remembered that the main foundation of burn treatment is cooling, prevention of hypothermia, early and aggressive resuscitation and definitive treatment in a burn care facility [7].

Topical treatment of burned hands is similar to that to the rest of the body. The first step is to eliminate the aetiological agent, dissipating the heat and reducing the temperature of the tissues in the first moments after the accident (cooling also reduces inflammation and relieves pain), profusely flushing the burns (which is especially important with chemical burns), or removing the patient from contact with an electrical source.

Local treatment of burns continues with the elimination of devitalized superficial tissue, such as blisters on the hands that have ruptured. Although there is controversy on this subject, we remove all blisters even if not ruptured. Once the patient is under analgesia, the wounds are profusely washed with normal saline. When there is a loss of epithelium, they are covered with a primary and secondary dressing. It is important to apply dressings that allow elevation and early movement of the hand and a complete range of motion. We prefer hydrocolloid or silver coated foam dressings that control pain and allow for early mobilization. In deep burns or major burns involving the hands, we tend to use 1 % silver sulfadiazine ointment dressings that prevent secondary infection. Silver sulfadiazine can be also be used with sealed plastic bags; they keep the hands free, moist and able to move during the healing process. Shallow or intermediate burns may also be treated by means of temporary artificial skin devices such as Biobrane[®] or Suprathel[®].

Full-thickness circumferential burns, especially those located in the upper limbs, can cause compartment syndrome, which should be actively watched for in the initial hours following the accident with every change of dressing, and when it is suspected, a decompression escharotomy should be performed.

We emphasize to the patient the importance of postural drainage using early elevation and active mobilization of the affected extremity. If the patient is unable to assist in their care due to their clinical condition, we place elastic traction holding the injured upper limb upwards in an upright position to minimize edema [8].

Correct diagnosis of the wound depth is mandatory in order to undertake the correct treatment. For decades, clinical observation by senior burn surgeons has been the most utilised tool for diagnosis, which dictates the subsequent route for definitive treatment. However, clinical diagnosis has only been proved incorrect in up to 35 % of situations. This error in diagnosis may produce an overestimation of the burn depth and a significant number of unnecessary operations. Currently, Laser Scanning with the Moor® technology is the equipment of choice for diagnosis [9]. Burns of indeterminate depth (those that are not superficial and full thickness in nature) are routinely scanned. Results are followed by clinicians and wounds that have poor vascularization and a time course of more than 21 days for complete healing are excised and grafted [10].

Therefore, the modern trend in the assessment and treatment of hand burns is as follows:

- Superficial burns (partial thickness or superficial second degree burns): Clinical inspection and diagnosis suffices. Treatment with hydrocolloids, alginates, or silver impregnated foams with light dressings that allow early rehabilitation are the treatment of choice.
- Deep burns (full thickness or third degree burns): Clinical diagnosis is easy and straightforward. These wounds are best treated with 1% silver sulfadiazine dressings and early excision and grafting with or without dermal substitutes.
- 3. Indeterminate burns (deep partial thickness burns): Diagnosis by means of Laser Scanning technology. Wounds with good healing potential (less than 3 weeks): same treatment as superficial wounds with close observation. External Pulse Acoustic Ultrasound Treatment (EPAUT) should be considered [11]. EPAUT involves the application of a number of pulses of shock waves to the injured skin. It penetrates some millimetres into the skin and promotes the liberation of growth factors, increases vascularisation, promotes debridement and helps in the healing process; converting deep wounds to superficial ones that

will heal uneventfully. Most wounds will heal with minimal scarring. For those wounds with minimal or poor healing potential (up to or beyond 21 days), early excision and grafting should be contemplated.

Surgical Intervention

Full thickness hand burns and burns to the hands with poor or minimal wound healing potential are best treated with formal excision of non-vital tissues and grafting [12].

Burns to the hands should be considered as semi-urgent operations, delaying excision and grafting may result in prolonged healing time, inflammatory response, scarring and poor outcomes. Minor or non-life threatening burn injuries that involve the hands should be excised as soon as possible and definitive treatment offered to the patient.

On the other hand, life threatening burn injuries should be considered as a whole, and correct but not emergent treatment to hand injuries installed. Large areas of deep burns (anterior trunk, back, lower limbs) are excised first. This allows better control of infection, inflammatory and hyper-catabolic response. Attention is then turned to important functional areas, such as the hands, which requires a longer operative time and large amounts of skin or skin substitutes.

Generally speaking, however, the surgical treatment of hand burns does not differ either in minor or in life threatening burns.

The operation should be performed under tourniquet control to minimise blood loss. If this manoeuvre is not anticipated, up to 2–4 packed red cells units may be lost during the excision and grafting of hand burns. All anticipated blood products should be ready and available before the surgery is started [12].

The Watson or Goulian knife may be used for tangential excision. However, new technologies, such as the "water knife" (hidrosurgery technology, Versajet[®] Smith and Nephew) are equally efficient for this type of excision and allows a precise depth of excision of deep partial burn wounds

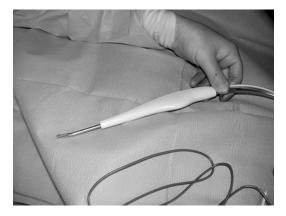


Fig. 11.2 The Versajet[®] water knife. It allows precise excision of dead tissue using the effect of the Venturi phenomenon

[13]. Tangential excision or Hydrosurgery excision (Fig. 11.2) must be carried out until punctate bleeding is observed. Wiping out the haemorrhage will present a white coloured dermis. If fat tissue is reached a similar appearance to that observed during hand surgery under tourniquet will be observed. Full thickness burns that extend to the fat tissue may require fascial excision. If this type of surgical treatment is required, all thrombosed vessels are excised en- bloc with the non-vital tissue. The superficial fascia and paratenon should be preserved if undamaged and will produce a much better functional outcome. If paratenon and deep tissues are excised, flap coverage or dermal regeneration templates such as Integra® will be necessary.

Active bleeders are controlled with careful haemostasis. Small and punctate bleeding points are controlled with epinephrine/adrenaline soaks (1:50.000). They are changed every 7–10 min until a dry wound is achieved. When epinephrine is used, the wound will get a bluish discoloration and clinical inspection of vital tissues will be not possible any more. Therefore, burn surgeons must be confident that all non-vital tissues have been excised before the epinephrine soaks are applied to the wound surface.

The ideal wound coverage continues to be the autologous skin graft. Hands are functional areas, therefore thick split thickness skin grafts are utilised. These grafts are placed unmeshed and follow aesthetic units (Fig. 11.3). Grafts seams are

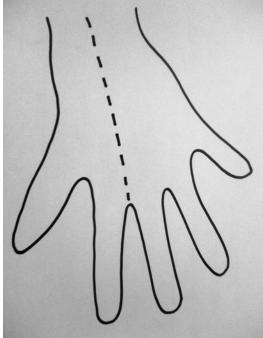


Fig. 11.3 The hand aesthetic units. Grafts should be positioned as two large sheets of skin avoiding seams over the knuckles

never placed over the MCP joints. If a complete hand is to be grafted, two separate skin grafts are used, with a graft seam following the longitudinal axis of the limb. The junction is normally placed between the second and the third metacarpal, which produces the best aesthetic effect. It has to be anticipated that skin coverage of two complete hands may require a donor site that includes a whole thigh. This is of particular relevance in major burns. If this is the case, dermal regeneration templates are indicated. They allow for timely skin coverage with a thin autograft. This allows for skin coverage of large surfaces to provide the best survival.

Full thickness burns to the hand require a different approach. The loss of the complete depth of skin mandates good coverage for satisfactory rehabilitation. Dermal regeneration templates have an absolute indication in these types of burns. Dermal regeneration templates, such as Integra[®] or Matriderm[®], are bioengineered permanent skin substitutes that integrate into the

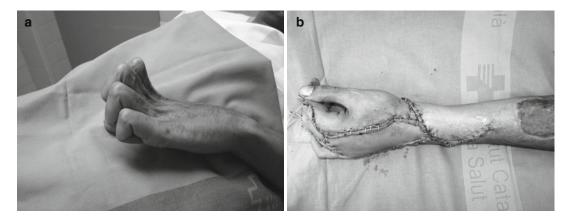


Fig. 11.4 (a) Extension deformity with destruction of the extensor tendons. (b) Reconstruction with the free dorsalis pedis flap with vascularised tendons

wound and promote healing as well as and the organisation of scars in a dermis-like structure. Templates are applied to the burn surface in the same fashion skin grafts are utilised. Fibrin glue sprays are useful to prevent seroma or haematoma formation. Two currently available approaches can be used either a single layer dermal template with a super-thin skin graft as one stage (prolonged wound healing of the skin graft is to be anticipated) or a bi-laminar dermal regeneration template in a two stage protocol, the second stage usually performed at 3–4 weeks. We prefer the latter since it allows for continuous rehabilitation during the whole period.

Deep burns affecting paratenon-denuded tendons may be treated either with dermal templates or flap coverage. If the area of tendon exposure is small, dermal templates suffice; however, in most cases flap coverage is better. In this situation, microvascular free transfer of either the temporo-parietal fascia or the antero-lateral thigh adipo-fascial flap obtains the best results. These flaps require coverage with skin grafts or dermal templates. They produce a thin coverage, good gliding surfaces and which allows for early rehabilitation and excellent outcome. Vascularised nerves, bone or tendons may also be required in cases where some of these structures have been burned/lost (Fig. 11.4a, b).

Local flaps in hand burn surgery are primarily indicated for the treatment of burned digits (Chapter A4, Watson & Pickford). Finger-tips burns in long digits, when bone is exposed, are best treated with the Y-Y advancement palmar flap (Tranquilli-Leali/Atasoy) based on the ascending arteries that branch off the distal central artery of the pulp. However, if the injury is bevelled obliquely on the palmar surface, the V-Y advancement lateral flap (Kutler) or the advancement-rotation quadrangular flap produce better results. If a more sensate reconstruction is preferred, though more complex, the neurovascular island flap (Littler), modified triangular flap (Venkataswami) or the dorsolateral island flap may be employed. Patients that present with injuries in such locations that prevent the utilization of local flaps from the same digit can be reconstructed by means of heterodigital reverse-flow neurovascular island flaps or boomerang flaps.

Injuries that involve the thumb pulp involve a different approach. These injuries can be treated with a neurovascular palmar advancement flap (Moberg), a dorsolateral island flap or the first metacarpal artery island flap (Kite or Foucher). Deep injuries to the palmar or dorsal surface of digits also require flap coverage. Advancement-rotation quadrangular flaps, first or second dorsal metacarpal artery island flaps are good choices. Or the dorsum, the adipofascial turn over flap (Lai) may be considered. Large defects of the dorsum of the hand can be treated with rotation flaps, bipedicle flaps or rhomboidal flaps. We have to remember too the new perforator based flaps based over the web spaces. However, in many situations the defect is too large for closure with local flaps, and distant flaps are necessary. Reverse flow flaps based on the radial, ulnar, or posterior interosseous arteries are good options if available, although such injuries often require free tissue transfers (these have reduced the utilization of the groin flap in modern burn hand surgery).

Rehabilitation

Current outcome measures concentrate on functional results, quality of life, and psychosocial issues. Survival and successful closure of the burn wound are, pre se, are no longer the main clinical factors that determine outcome. Rehabilitation plays a central pivot point in burn care.

Hand burns can result in a major functional impairment. As such the restoration of function and return to society as a productive individual are the main goals of the multidisciplinary approach in burn centres throughout the world. Rehabilitation physicians, physiotherapists, and occupational therapists are key team members. Rehabilitation services are involved from day one in the care of burn patients, and, together with the rest of the burn team members, a comprehensive, individualised rehabilitation program must be instituted for each patient [14, 15].

This program should be initiated with a rehabilitation and social services visit and workout that includes all social, psychosocial, and functional problems of the patients. All likely problems encountered must be addressed, and a discharge program instituted, aimed at complete restoration of function and the prevention of all possible complications drawbacks foreseen during this visit.

Initial intervention includes elevation of the affected limb, splinting if necessary, early mobilization of the burned hand and application of light dressing that allow such intervention. It should be emphasized that rehabilitation interventions are not only instituted on the admission of the patient to the burn centre, but continue during surgical intervention if necessary (with intra-operative splinting) and continue during the whole outpatient care of the patient until the patient returns to his/her normal daily living activities and work/school integration. Splinting, pressure therapy, polarized light therapy (lamps with polarised light, which help in reducing oedema and produce better scars) occupational therapy, silicone inserts and massage are all part of the rehab program. Interim pressure garments are beneficial and should be considered at the time of surgical intervention. Traditionally preventive splints position the hand in the intrinsicplus position during the acute phase. However, individualised splints are also advocated. The burn team should assess the potential functional restrictions and possible future contractures and fashion personalised splints accordingly.

Reconstruction

Function should be the main priority. Correct and intensive rehabilitation is the most important part of reconstruction for function in acute burns and those patients in the initial (usually the first year) post-burn period. If the program progresses with good patient compliance, late reconstruction might not be necessary.

Those patients that present with early restrictions in range of motion should follow an intense rehabilitation program aimed to correct the impending deformity. It is reasonable to apply the program for an 8–12 week period. Patients that do not improve should then be assessed anew and surgical intervention considered. Table 11.2 summarises the most common deformities encountered in the burned hand.

All plastic surgery techniques apply in burn hand reconstruction. It is necessary to take a good medical history and physical examination to understand the process and problems resulting in the functional restrictions. It is imperative to do so to avoid recurrence of the deformity. Full thickness skin grafts and dermal regeneration templates are very useful in large flat restricted scarred areas, especially over the dorsum of the hand. However, flaps are also very useful in overcoming burn hand deformities. Table 11.3 lists the most common procedures performed for different deformities.
 Table 11.2
 Deformities commonly encountered in hand
 reconstruction

	Incisional release
1. Flexion deformities	Excisional release
2. Boutoniere deformity	Primary closure
3. Mallet finger deformity	Skin grafting
4. Webspace contractures	Z- plasties and modific
5. Syndactyly	Local and regional flap
6. Adduction deformity of the thumb	Distant/free flaps
7. MCP hyperextension	Dermal regeneration te
8. Wrist hyperextension	Tendon grafting and re
9. Tendon and nerve destruction	Nerve grafting and rec
10. Palmar contracture	Capsulotomies, arthroo
11. Wrist flexion contracture	Amputations
12. Amputation	Pollicisation, toe to ha

Table 11.3 Useful techniques in hand reconstruction

Incisional release	
Excisional release	
Primary closure	
Skin grafting	
Z- plasties and modifications	
Local and regional flaps	
Distant/free flaps	
Dermal regeneration templates	
Tendon grafting and reconstruction	
Nerve grafting and reconstruction	
Capsulotomies, arthrodesis, joint replaceme	ent
Amputations	
Pollicisation, toe to hand transfers	
Hand and upper extremity allotransplantation	on

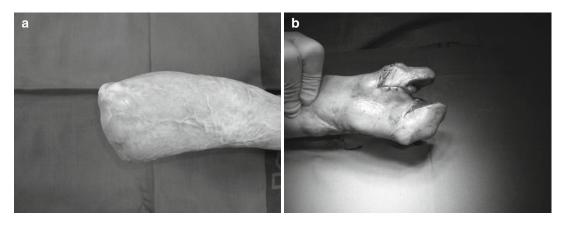


Fig. 11.5 (a) Metacarpal burned hand. Function is severely impaired in this type of deformity. (b) Reconstruction of pinch function by second to first metacarpal pollicisation

Phalangeal deformities are reconstructed using either local flaps, Z-plasty and modifications, dermal-regeneration templates or full thickness skin grafts. Tendon repair is necessary with boutoniere deformities. In this situation, the reconstruction proceeds in the standard fashion. All anatomical structures are located and reconstructed, provided that the extensor mechanism has not been damaged/destroyed in the initial injury.

Webspace contracture and syndactyly is a common deformity in burned hands. When approaching this type of reconstruction, the surgeon must be aware that they recur very frequently, and the patients should be warned regarding possible future reconstructive needs. Reconstruction proceeds by using modified or multiple Z-plasties. In some severe cases, skin grafting is also necessary.

Hyper-extension and flexion deformities of the metacarpo-phalangeal and palmar surfaces are managed by incisional/excisional release (depending on the degree of the contracture and hypertrophic scars) and reconstruction by either skin grafts, dermal regeneration templates or regional/distant/free flaps. Thumb reconstruction is normally managed by pollicisation (especially in the metacarpal hand) (Fig. 11.5a, b) or with toe to hand transfers. The most severe cases in burn hand reconstruction require a complete holistic approach. Many deformities can be present in these

patients and a full reconstructive plan, including the rehabilitation requirements, should be set in place.

Frostbite Injury

Frostbite injury is one of the possible injuries caused by cold temperatures, normally as a result of caused by prolonged exposure at temperatures below freezing. It involves the formation of ice crystals within living tissues, leading to different degrees of tissue necrosis and irreversible neurovascular changes.

Two different events occur after prolonged cold exposure. The most visible one is the direct injury to the cells. The second event is vascular impairment, leading to vessel thrombosis and more indirect injury to the tissues.

The main steps of treatment of frostbite injuries include:

- 1. Restoration of normal core body temperature
- Trauma management, as in any other traumatic injuries
- 3. Sedation and analgesics
- 4. Assessment of vessel circulation and antithrombotic agents if indicated
- 5. Antibiotic prophylaxis
- Light dressings and/or hand type silver sulfadiazine dressings

Rapid rewarning of the affected hand is often performed by a warm water bath with temperatures ranging 40–44 °C. Next, light bulky dressings including of plastic bags with silver sulfadiazine are employed. If vascularisation is impeded, antithrombotic agents or/and drugs affecting the sympathetic nervous system should be considered.

Thereafter, it is recommended to undertake a conservative approach to the affected areas, since many areas may heal primarily and others may benefit from a conservative/delayed debridement. At this stage, it is easy to debride the necrotic areas and perform reconstruction. The management of these sequelae often includes management of mummified digits and necrosis of skin and soft tissues (most of the cases occurring at the tip of the fingers). Late sequelae may involve intrinsic muscle atrophy and vasospastic syndromes.

References

- Peck MD. Epidemiology of burns throughout the world. Part I: distribution and risk factors. Burns. 2011;37: 1087–100.
- Pruitt BA, Wolf SE, Mason AD. Epidemiological, demographic, and outcome characteristics of burn injury. In: Herndon DN, editor. Total burns care. 3rd ed. Philadelphia: Elsevier; 2007;14–32.
- Stander M, Wallis LA. The emergency management and treatment of severe burns. Emerg Med Int. 2011; 2011:161375.
- Williams N, Stiller K, Greenwood J, Calvert P, Masters M, Kavanagh S. Physical and quality of life outcomes of patients with isolated hand burns – a prospective audit. J Burn Care Res. 2012;33:188–98.
- Wrigley M, Trofman K, Dimick A, et al. Factors relating to the return to work alter burn injury. J Burn Care Rehabil. 1995;16:445–50.
- Robson MC, Smith DJ, VanderZee AJ. Making the burned hand functional. Clin Plast Surg. 1992;19: 663–771.
- Warden GD. Fluid resuscitation and early management. In: Herndon DN, editor. Total burn care. Philadelphia: Elsevier, 2007; pp. 107–118.
- McCauley RL. Reconstruction of the pediatric burned hand. Hand Clin. 2000;16:249–59.
- Monstrey S, Hoeksema H, Verbelen J, et al. Assessment of burn depth and burn wound healing potential. Burns. 2008;34:761–9.
- Deitch EA, Wheelaham TM, Rose MP, et al. Hypertrophic burn scars: analysis of variables. J Trauma. 1983;23:895–8.
- Arno A, Garcia O, Hernan I, et al. Extracorporeal shock waves, a new non-surgical method to treat severe burns. Burns. 2010;36:844–9.
- Heimbach D. Early burn excision and grafting. Surg Clin North Am. 1987;67:93–107.
- Gravante G, Delogu D, Esposito G, et al. Versajet hydrosurgery versus classic escharectomy for burn debridement: a prospective randomised trial. J Burn Care Res. 2007;28:720–4.
- Dewey WS, Richard RL, Parry IS. Positioning, splinting, and contracture management. Phys Med Rehabil Clin N Am. 2011;22:229–49.
- Kowalske KJ. Hand burns. Phys Med Rehabil Clin N Am. 2011;22:249–59.

Post-Traumatic Contracture

Sarah Mee and Ian A. Trail

Keywords

Actiology • Classification • Presentation and assessment • Investigations • Role of therapy • Role of surgery • Outcomes

Introduction

Contracture after injuries to the hand and wrist is not uncommon, affecting particularly the wrist, metacarpophalangeal (MCP) and proximal interphalangeal (PIP) joints. Fortunately, in most cases the contracture either resolves or is not clinically significant and can be ignored. In others, however, treatment is required. The nature of a contracture depends very much on the severity of the initial injury, the treatment received and perhaps most importantly the patient's attitude. For the latter, a patients understanding of the nature of the injury, the treatment received and what is expected of them is paramount. Even if treatment is optimal, an unresponsive patient will

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more than likely have a poor outcome, including an ongoing contracture. With regard to the injury itself, the more severe the trauma; the more likely it is that a patient will develop a contracture. For this reason it is important that the clinician appreciates what particular structures have been damaged; is there any bony injury, has the articular cartilage been damaged, or is there malalignment? For the soft tissues again it is important to ascertain which have been permanently damaged; the dorsal or volar capsule (volar plate), or the collaterals. In addition, have any of the surrounding structures been injured, particularly the tendons including the central slip, but also the neurovascular structures as well as the skin and underlying soft tissues. All of these may need attention if the contracture is to be addressed.

Finally, with regard to treatment, the role of the surgeon can be contentious. Whilst obviously addressing severity and over-seeing treatment, the role of surgical intervention is not always clear cut. It is often too easy to recommend intervention when this is not in a patient's best interest. Surgery itself will inflict further trauma to the injured area and can result in otherwise normal tissues being damaged. In a study by Weeks, et al on 453 PIP joint flexion contractures treated with

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an intensive programme of active, passive and resisted exercises, static and dynamic splinting, 87 % responded to treatment to the extent that surgery was not indicated [1].

The key for the surgeon therefore is to balance the relative benefits and risks of intervention.

Perhaps of greater importance, however, is the pivotal role of the therapist. Any rehabilitation regime must ensure maximum pre-operative improvement, as well as firstly demonstrating to the patient the commitment necessary after any surgical intervention, and secondly indicating to the therapist the likely level of compliance of that patient.

The most important task for the hand therapist is to establish which of the underlying structures is causing the stiffness and the limitation of joint range of motion. A regime can then be planned to target those specific structures; for example, a programme concentrating on tendon glide will have little impact on a stiff joint. In addition, the longer the period of stiffness, the more extensive and intensive the rehabilitation is likely to be. Part of the role of the hand therapist is to continue to motivate the patient to maintain the range of modalities and the time commitment required.

All the modalities discussed in this chapter are relevant either pre-operatively or post-operativelythe differences being the application, duration and specific modalities chosen for each patient and joint. In-depth assessment and clinical reasoning to set up the programme is vital.

Background/Aetiology

Contraction, or at least the potential for contraction, is normal after any injury. In most cases, however, this does not occur as the injury is mild and patients are soon able to mobilize and stretch to prevent any permanent deformity. In more severe cases, however, particularly in cases where surgery has been undertaken and early mobilization impractical, stiffness and contracture may ensue. It is therefore key to identify which structures have been injured, either bony or soft tissue.

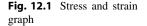
Bony injuries can obviously be either extra or intra-articular. Undisplaced or well fixed extraarticular fractures often do well; in so much that
 Table 12.1
 Classification of injuries

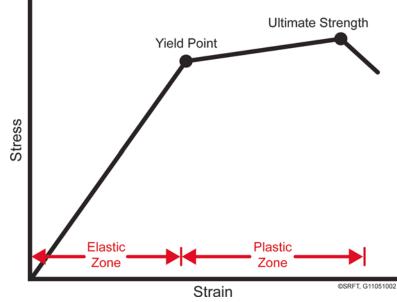
Bony injuries	Intra-articular disruption	
	Extra-articular disruption	
Soft tissue injuries	Intrinsic	Collateral ligament injury
		Capsular injuries
		Volar plate injuries
	Extrinsic	Tendon/flexor sheath
		Neurovascular structure
		Skin/subcutaneous tissue

patient's can begin mobilizing sooner rather than later. However, displaced intra-articular fractures managed either surgically or conservatively are more likely to result in stiffness and contracture. The basic patho-physiology being the damage to or altered position/alignment of the surrounding soft tissues results in healing with contracture or altered mechanics, which in turn leads to stiffness. Hence the importance of splinting the joint and surrounding soft tissue on full stretch (the MCP joint's at 90° and the PIP's in extension). Intra-articular incongruity can lead to both maltracking of the joint, or act as a direct block to movement. Added to that, extra-articular fractures can also result in adhesions to the overlying flexor and extensor tendons, which again will result in contracture. The more displaced the fracture, the more likely it is to develop adhesions. A comminuted fracture or indeed fractures that undergo surgery also have a higher risk of adhesion formation.

Of the soft tissue injuries, as can be seen from Table 12.1, these can be either classified as intrinsic or extrinsic, depending on whether the injury is to the tissues immediately surrounding the joint or external to it.

Whilst it is unusual for an individual structure to be injured in isolation, usually one structure is damaged predominantly. For example, a collateral or other restraining ligament, when put under load, will initially elongate. When this occurs during the "elastic phase" (Fig.12.1), if the load is removed the ligament returns to its pre-injury length. Later, however, in the "plastic phase," if the load is removed the ligament will not return





to its previous length, but will heal in an elongated fashion. Clinically this will result in instability, rather than contracture.

Finally, the end result is rupture, either as an intra-ligament tear, or as an avulsion from bone. During this process, other structures, specifically the adjacent capsule, will also be affected, although the principle injury is to the collateral. When the healing process begins collagen is laid down, which eventually leads to scar formation and contracture. If this affects the collateral ligaments predominantly this will lead to a loss of movement, particularly at the extremes, e.g., full extension at the PIP joint or flexion at the MCP joint [2].

At other sites, particularly if the ligament is avulsed from bone, direct healing may not necessarily occur, e.g., ulnar collateral ligament injuries at the metacarpophalangeal joint of the thumb. Again this will result in instability, rather than contracture. Optimal treatment involves allowing the ligament to heal in as normal position and length as possible, yet maintaining motion.

Capsular injuries, particularly to the volar plate, again often result in contracture, usually as a result of the volar plate healing in a contracted fashion. At the PIP joint, this would result in a flexion deformity. Less often, detachment or elongation can occur, which will result in hyper-extension.

Extrinsic injuries to tendon or skin, which in their own right can lead to contracture, can also result in stiffness in associated normal joints. For example, a flexor tendon injury that dehisces after repair, or becomes stuck within the flexor sheath, will result in a flexion deformity of the finger, which will ultimately result in a secondary contracture of the associated joints, either the MCP or PIP, despite them essentially being normal. Similarly, rupture of the extensor central slip off the base of the middle phalanx will result in weakness extending the PIP joint and, again, a tendency to develop a flexion contracture.

Why, therefore, does an erstwhile normal joint develop such a contracture? The current rationale is that normal cartilage nutrition depends on joint loading and motion [3]. If the latter is absent, cartilage degeneration occurs and adhesions develop. These result in the contracture of the joint capsule, volar plate and collateral ligaments, leading to more permanent loss of movement.

Brand and Hollister stated that joint stiffness is on average caused by 90 % elastic factors (collagen and elastin), 9 % viscous oedema and 1 % friction in the joint [4]. The hand therapist, therefore, needs to concentrate on modalities that allow, in particular, elongation of contracted elastic tissues. Collagen fibres Type III (scar) need to be stimulated to alter to Type I (normal extensile tissue). Prolonged stretch with gentle consistent force encourages this. Flowers and LaStayo concluded from a study of total end range time that, if a joint is held at its moderately lengthened position for a significant time, the tissues will lengthen [5].

It is also important to remember that a patient may present with decreased range of movement due to pain. This is not true stiffness and management of the pain will produce a full range of motion.

With regard to management, obviously both areas have to be addressed if the patient is to achieve better movement; that is not only has the tendon to be addressed, but also the accompanying joint contracture. Fortunately, it is often possible to treat the latter conservatively prior to releasing or reconstructing the tendon.

In addition, injuries to the flexor sheath in their own right can produce adhesions and consequent restriction of movement of the flexor tendon. When deficient it can result in "bow-stringing" of tendons, particularly at the level of the proximal interphalangeal joint. Again, this has the effect of producing a contracture with associated secondary stiffness of the PIP joint. Obviously, again both issues have to be addressed for treatment to be successful.

Finally, if there is significant damage to skin and the subcutaneous tissue, for example as a result of major trauma, burn or a degloving injury, this can also lead to contracture in an underlying joint. Again, any treatment will require release of this contracture and the application of new skin and subcutaneous tissue, ideally using flap cover.

Presentation, Investigation and Treatment Options

History

The clinical presentation of a contracture is usually obvious. Whilst patients may complain of pain, the deformity is often readily seen. Functionally, loss of movement or fixed deformity can lead to specific disability. Examples at the wrist would be an inability to manipulate the hand into a particular position, e.g., handwriting, toileting etc. The lack of finger extension can diminish span, making gripping difficult. As such, taking tops off jars and other twisting actions may be difficult. In addition, patients often make complaint of an inability to open the hand to take change. Finally, contracted fingers can be caught, for example in pockets.

Added to this, grip or pinch strength will normally be diminished, as the wrist or hand cannot be placed in the optimum position for strength. Finally and by no means least, patients often complain about the appearance.

Establishing the Cause of Stiffness

On examination any contracture is usually obvious. It is, however, important firstly to determine whether this deformity is fixed, passively correctable or a combination of both. A fixed contracture usually means that there is a problem with the joint itself, whereas if the contracture is passively correctable, there is usually an extrinsic tendon or muscle component. (The degrees of contracture are quantified by direct measurement using a goniometer.) This is important, as it will provide objective evidence of any improvement or not. Clinical photographs can also be useful.

Assessment of Stiffness (Table 12.2)

 Assess for chronic oedema that may be restricting range of motion, particularly at the PIPJ. Dorsal oedema will restrict flexion, as the skin has to stretch further. Shin and Amadio showed that 12 mm of skin stretch was necessary for 90° flexion at the PIP joint, although this was increased to 19 mm for an oedematous joint [6]. Oedema may be assessed using various methods e.g. tape measures for individual digits or a volumeter if oedema is present throughout the hand. Be aware that circumferential measurements using tapes show variable inter-rater reliability, though intra-rater reliability is higher [7, 8].

DIPJ sits in ext	DIPJ sits in flex	PIPJ sits in flex	PIPJ sits in ext
Limited in flex	Limited in ext	Limited in ext	Limited in flex
\downarrow	Ļ	\downarrow	\downarrow
Active limitation caused by	Active limitation caused by	Active limitation caused by	Active limitation caused by
Flexor Digitorum Profundus	Terminal extensor tendon	Central slip	Flexor Digitorum Superficialis
		Trigger locking	Early swan neck deformity
		Lateral bands	
Passive limitation caused by	Passive limitation caused by	Passive limitation caused by	Passive limitation caused by
Arthrodesis	Arthrodesis	Volar plate	Intra-articular tightness
Intra-articular tightness	Intra-articular tightness	Dupuytrens	Dorsal capsule
Extrinsic extensor tightness	OA	Trigger finger	Established swan neck
ORL	Extrinsic flex tightness	Intra-articular	Extrinsic extensor tightness
Intrinsic mm tightness		Collateral ligaments	Intrinsic mm tightness
Lateral bands/TRL		Extrinsic flex tightness	Central slip contracture
		Volar joint capsule	
		Lateral bands	
		Transverse retinacular ligament	

 Table 12.2
 Algorithm for assessment of structures causing stiffness

When assessing the PIP joint – the following algorithm may be helpful

- Measure the range of motion isolated and composite as well as active and passive. This will differentiate between capsular tightness, tendon tethering and extrinsic or intrinsic tightness. Using an appropriate goniometer and a standardised method of measuring increases the reliability of joint measurement in experienced therapists to between 5 and 10° [9].
- Always look at what is restricting the ROM tightness or pain. Is it true tissue tightness or muscle guarding due to anticipation of pain? This is not true stiffness and the pain response would need to be managed initially.
- 4. Manual Muscle testing assess the strength and action of individual muscles to isolate tethering, shortening or rupture of particular tendon/muscle units. The Medical Research Council (MRC) Scale for Muscle Strength is the most commonly used (1981), with Grade III (movement through range against gravity) being the only objective grading [10].
- Differentiate between intrinsic and extrinsic tightness of the muscles and possible tendon tethering – for digital intrinsic tightness, the therapist passively extends the MCP joint to

neutral and then passively flexes the interphalangeal joints into maximum flexion [11]. If it is not possible to fully flex the IP joints and there is pain or stretch obliquely along the lateral borders of the proximal phalanx, this denotes tightness of the intrinsic muscles (Fig. 12.2a).

This position places the intrinsic muscle/ tendon unit on full stretch, while composite flexion of the digit relaxes the intrinsics (Fig. 12.2b, c).

For digital extrinsic tightness, the therapist passively flexes the digit at all joints to maximum range of motion. If this is limited, the long extensors are tight or scarred (Fig. 12.2d).

Extrinsic extensor or flexor muscle/tendon tethering or tightness is tested with passive composite wrist and finger extension or flexion.

6. Assess for intra-capsular tightness or shortening- Joint palpation with ballottement or accessory mobilisation aims to assess for tightness of ligaments or joint capsule and to differentiate between joint tightness or laxity (Table 12.2). Always compare with the contra-lateral side, as there is a wide

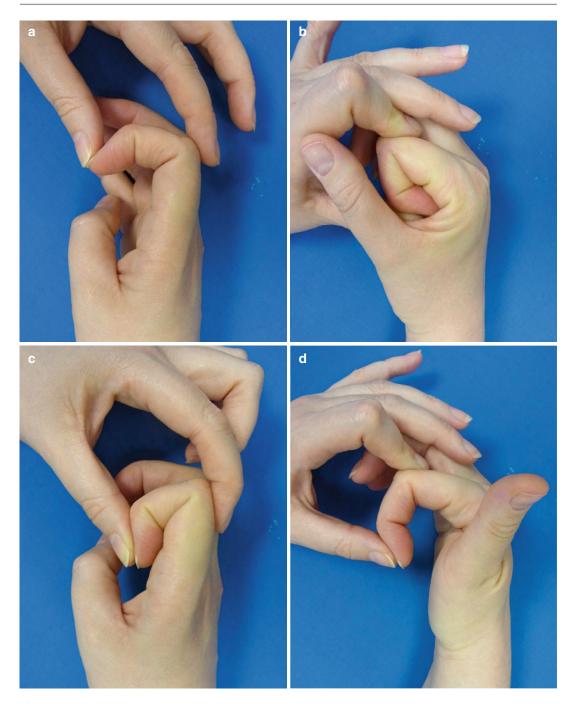


Fig. 12.2 Demonstration of intrinsic and extrinsic tightness. (a) Intrinsic tightness. (b) Full composite flexion. (c) Normal intrinsic stretch. (d) Extrinsic tightness

range of 'normal'. Remember that ligaments may be tight in different positions of movement; for example collateral ligaments at the PIP joint are tight in flexion beyond 60° and accessory collaterals tight between 0 and 20° . Similarly, wrist dorsal and volar radioulnar ligaments tend to be tighter in supination or pronation respectively.

 Assess grip strength, sensation and function, as these may well alter the patient's motivation to improve and their expectation of the outcome of rehabilitation or surgery.

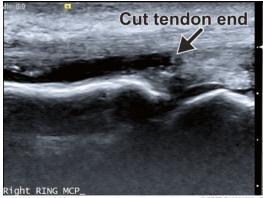
Clinical Pearl

Assessing the stiff hand requires knowledge of the detailed anatomy, mechanics and specific tests to differentiate between capsular intraarticular or extra-articular tightness.

Investigations

All patients who present with a contracture after an injury should have two views of a **plain x-ray** of the affected area. This is to identify any previous bony injury either malunion or malangulation, or indeed if secondary osteoarthritis has supervened. All of these will have an effect on any treatment proposed.

Dynamic Ultra sound scans can also be useful, particularly in identifying tendon damage; either rupture or dehiscence following repair, acute or chronic inflammation or tethering of soft tissue. This would also apply to the flexor sheath where the tendons will be seen to have separated (Fig. 12.3). Other modalities of scanning MRI/ CT are currently less useful, but will undoubtedly improve with time. CT may be most relevant when assessing for altered articular surfaces producing a bony block to movement.



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Fig. 12.3 Ultrasound image of divided flexor tendon

Prevention of Stiffness

The optimum treatment for all contractures is prevention. There is a general tendency after injury for joints to assume a position of comfort. The best example of this is at the proximal interphalangeal joint, which is often held at between 30° and 40° of flexion. It is in this position that the capsule has its maximum volume and as a consequence is a position of comfort. Unfortunately, however, if left at this angle any soft tissue injuries will heal in a contracted position resulting in permanent stiffness. As a consequence, it is essential that the joints are splinted with the soft tissues at full stretch. For the wrist this would be neutral, for the MCP joint 90° of flexion and the PIP joint at full extension.

Surgical Treatment Options

Treatment options for contractures in the hand and wrist are numerous and will tax even the most skilled and experienced hand surgeon or hand therapist. Whatever is undertaken, however, will require a team approach. Firstly, the patient should be fully aware of what is proposed, together with the relative advantages and disadvantages combined with the complications. Their participation particularly in any therapy is crucial. It is therefore strongly recommended that the patient is seen by the therapist pre-operatively to allow the post-operative therapy programme to be defined and explained. Indeed in our opinion post-operative therapy input is more important than the surgery itself. If the input of an appropriately qualified hand therapist is not available then this type of surgery should not be attempted.

Table 12.3 shows the treatment options available:

Of the bony operations, **osteotomy** is probably the most rewarding. However, it should be carefully planned, particularly with regard to the site of the osteotomy, the need for bone graft and the placement of any internal fixation. The latter must not be placed over joint capsules or collaterals and preferably give rigid fixation such that early mobilization can begin. With this in mind, plate

Cause of stiffness	ness Surgical treatment options	
Bony	Osteotomy	
	Replacement	
	Excision	
	Arthroplasty	
	Arthrodesis	
Soft tissue		
Intra-articular	Capsular release	
	Collateral and accessory collateral ligament release	
	Volar plate release	
Extra-articular	Tendon realignment	
	Tendon release, repair, reconstruction	
	Pulley reconstruction	
	Flap cover	

Table 12.3 Surgical treatment options

and screws seem preferable to k wires. On the down side there is obviously a risk of non-union or infection. Added to that range of motion may not increase significantly, although it may well be in a more functional plane. Extra-articular osteotomies typically do better than intra-articular; the latter only being required for severe intra-articular incongruity, particularly in younger patients. Results, however, can be unpredictable.

Joint replacement can also be undertaken if there is particularly severe joint disruption, or if secondary arthritis has developed. Indeed, in selected cases this can be the treatment of choice giving more predictable results, including correction of contracture.

Finally, **arthrodesis** should be considered if reconstruction is not possible, but the deformity is causing significant impairment. Effectively the joint is arthrodesed in a more functional position.

Releases around the joint, either of the capsule, collaterals or volar plate, form the basis of most procedures. It is important, however, that the operation is undertaken in a systemic yet limited manner. That only the contracted tissues are released and normal tissues are left untouched. Gentle handling of the tissues is therefore essential, as significant post-operative bruising and swelling will result in difficult mobilization and ultimately little improvement. Arthroscopic release of a joint, particularly of the wrist capsule, has obvious advantages in this regard.
 Table 12.4
 Rehabilitation treatment options

	1	
Cause of stiffness	Rehabilitation options	
Bony	Acceptance of	
-	limitation of movement	
	and maximising	
	function	
	Splinting for pain and	
	support in function	
	Muscle strengthening	
	Activity modification	
Intra-articular tightness or	Heat modalities	
tendon tethering/extrinsic	Oedema modalities	
<u>tightness</u>	Splinting	
	Static, serial, static progressive, dynamic	
	Joint mobilisations	
	Passive stretches	
	Active and resisted	
	exercises	
	Tendon glide exercises	
	Activity rehabilitation	
	Scar, graft or flap	
	management	
	Blocking splints to	
	isolate joints or tendon	
	function	
	Functional activities	

External to the joint, **tendon realignment**, **tendon release**, **repair or reconstruction** are all crucial, as without normal or near normal tendon excursion full active mobilization cannot be undertaken. It is essential, therefore, to obtain at least maximal or full passive joint motion prior to tendon reconstruction. A similar situation would apply to any **sheath/pulley reconstruction**.

With regard to skin and subcutaneous tissues, none of the above procedures will be successful if there is a skin contracture. This has to be addressed by the application, preferably of flap cover, or at least a full thickness skin graft. This is usually undertaken prior to any other reconstruction.

Rehabilitation Treatment Options

Management of the stiff digit or wrist relies on the initial assessment and then intensive therapy, using a core of the most appropriate modalities for those structures most affected (Table 12.4). Clinical reasoning is vital to ensure that treatment targets the appropriate cause of the stiffness. Rehabilitation will take a different form for the stiff joint caused by capsular contracture, rather than that as a result of tendon or other soft tissue tethering. In a chronicly stiff hand there are likely to be elements of both and the therapist will need to prioritise these in relation to the patient expectations and function. Generally, gaining flexion is more beneficial functionally, but patients will often prioritise extension for cosmetic reasons.

In order for stiff joints or tight soft tissues to improve, it is vital that rehabilitation is carried out regularly every day and overnight with splinting. A clear, concise and achievable home rehabilitation programme needs to be taught to the patient as well as attendance at therapy. The therapist should concentrate, in treatment sessions, on modalities that are not possible for the patient to achieve themselves at home, whilst also checking home programme activities and exercises to ensure the patient is using the correct technique.

The hand therapist needs to be realistic about time scales, to use a range of modalities specific to the patient and the problem and evaluate their effectiveness regularly. If a modality is not making a change within a few weeks, a different method or application needs to be tried. The hand therapist needs to know when to advise the patient that further active therapy is unlikely to change the stiffness and that they either need surgery or to learn to adapt to the remaining disability. In choosing the modalities most likely to be effective for an individual patient, aspects such as inflammation and pain must always be considered. Slow prolonged periods of tissue stretch have been shown to produce consistently improved results in elongation and glide of tissue, without micro tissue trauma [5].

Many patients with stiff hands will need to accept a functional range of motion rather than full range and adapt grips to enable maximum function. A study by Hulme et al. (1990) [12] found that flexion postures averaged 61° (range $33^{\circ}-73^{\circ}$) at MCPJ, 60° (range $36^{\circ}-86^{\circ}$) at PIPJ and 39° (range $20^{\circ}-61^{\circ}$) at DIPJ in a range of ADL tasks [12]. A further study by Woodworth et al. (2006) [13] showed that PIPJ's held at 40° (the most common fixed flexion position for post trauma contracture) showed an increase in MCPJ flexion required and an increase in the MCPJ hyperextension used [13]. They also noted that precision tasks, which would normally rely on dynamic PIPJ movement, showed the most significant change in MCPJ movement. Wrist functional range requires 40° of extension and flexion, a total range of 40° of ulnar and radial deviation with 75 % rotation range [14].

It is not in the remit of this chapter to give details of how to carry out each of these modalites, but to encourage clinical reasoning in choosing the most appropriate therapy for each patient. In order to improve clinical reasoning, the discussion will be split into common modalities and then those for intra-articular tightness and soft tissue glide.

Common Rehabilitation Modalities

Heat Treatment

Mild heating of tissues increases blood flow and metabolic rate and allows more tissue extensibility as well as reducing pain [15, 16]. Soaking the hand in warm water, or immersing in wax prior to passive stretching can be of great benefit. Heat packs can be used if immersion in water is contraindicated e.g. in the presence of K-wires. Hot paraffin wax gives a well insulated, low temperature method of heating the tissues [17]. Therapists should incorporate heat and stretch by applying tape (e.g. micropore) to maintain the digits in flexion and then dipping the hand in the paraffin wax.

Oedema Management

Oedema is a normal part of the inflammatory healing process, but the majority of this oedema should be eliminated within 6 weeks to reduce developing stiffness [18]. Oedema remaining after this time is likely to disperse very slowly and residual swelling may remain for many months. The following modalities will have decreasing effectiveness, as the oedema becomes more chronic.

Elevation

Elevation is most effective in acute oedema [19]. Gravity will aid the flow of the lymphatics. Active exercises carried out in elevation will further aid reduction of oedema by creating muscle pumping and movement of soft tissues [20, 21].

External Compression

Compression should not be used during the first 3-5 days of the inflammatory stage, as oedema is necessary for healing [22]. Gentle external compression increases the capillary volume and speeds vessel filling, oedema therefore reduces as the blood flow and lymphatic drainage improves. Compression needs to be only sufficient to stimulate the lymphatic drainage and not to overload the system [18]. Compression is provided by using some form of elastic wrapping, such as elasticated wrap, lycra digisleeves, compression gloves or string wrapping. External compression is contraindicated in the presence of infection. Always wrap from distal to proximal, ideally leaving the finger tips free to assess capillary return. The patient must be warned to watch for signs of the wrapping being too tight.

Cooling Temperatures

Cold induced vasoconstriction may help to minimise inflammatory swelling, by decreasing blood flow to the area [23]. Cryotherapy has also been shown to be effective in pain management [24, 25]. Chronic oedema is less likely to benefit from cryotherapy. Cryotherapy may be provided by ice or cool packs, or contrast bathing. Ice packs have been shown to be more effective than contrast bathing, but be aware that in the early stages local vasodilatation does not inhibit lymphatic drainage [26]. Contrast bathing may also be an effective method of reducing oedema with alternating hot and cold producing a pumping action and aiding lymphatic drainage. Combining cold, compression and elevation has been found to be most effective for decreasing oedema [16].

Active Exercise

Active exercise increases blood flow, facilitating venous return and pushing the fluid along more quickly [27]. Exercises may improve lymph flow

up to ten times [28]. Active movement encourages tendon excursion through the oedema and maintains joint mobility and increases range of motion as the oedema reduces. To be most effective for oedema control, active exercise must also include full range of arm and shoulder motion to keep the fluid moving through the lymphatic system at elbow and axilla.

Electrotherapy

Pulsed shortwave diathermy (PSWD) has been advocated to decrease oedema. There are clinical claims as to the effectiveness of PSWD, with the suggestion that it helps to increase the number of white cells, histocytes & fibroblasts in a wound, which in turn improves the rate of oedema dispersion with a reduction in the inflammatory process [29, 30]. However, there is no conclusive evidence to date.

Lymphatic Massage

MEM (Manual Edema Mobilization) is a method of gently stimulating the lymphatics to facilitate the flow of excessive tissue fluid and proteins from an oedematous area. It involves light proximal to distal then distal to proximal massage of the skin carried out in segments [18]. It is applicable to people suffering persistent high protein oedema, with intact, but overwhelmed, nodes and lymphatic system. It is important that following massage any improvement is maintained by elevation, pressure garments and exercise [25–27].

Elastic Taping

Kase et al. (2003) [31] proposed that Kinesiotape, placed in a direction that may lift the skin and increase the interstitial spaces, encourages natural lymphatic drainage and reducing pressure on underlying tissues. The drainage must always start proximally to clear those nodes and allow the fluid to move freely from the distal hand and arm region.

Kinesio-Taping is most commonly done as a series of fan shapes, with tape stretch of 15 % only, started at proximal axilla lymph nodes, elbow nodes and then into wrist, hand and fingers. Patients and relatives may be taught to do this at home. Most evidence is anecdotal at



Fig. 12.4 Example of kinesiotaping for lymphatic drainage

present, but is supported by the theory of lymphatic drainage (Fig. 12.4) [31].

Scar Management

Scar Massage

Massage techniques can help eliminate tethering and invigorate the blood supply to the area of the scar, increasing range of motion, but having less effect on skin mobility [32]. The massage should be firm and distract the skin from other soft tissue. A greasy cream should be used to ensure that many minutes of massage can be done before the cream is absorbed. The patient must understand that it is the massage that is therapeutic, not the cream.

Ultrasound

Used in the stiff hand and wrist to help with the softening of thick scar tissue, but with limited evidence base for effectiveness in musculoskeletal injuries [33]. Continuous ultrasound used for at least 6 min may encourage scar tissue to become more elastic and extensible and may be beneficial when used prior to scar massage. (www.electrotherapy.org).

Silicone Gel

There are many silicone gel sheets and topical gel treatments available for scar care. There is still controversy about how they work, but they may be effective in flattening raised scars, hastening the maturation process, decreasing colour and some gels keep the scars moist [34]. The selection of the gel is important, as they have differing abilities to contour or adhere to the skin and have different life spans. A study by Chernoff et al. (2007) [35] showed most effective management of scars with topical gel (Kelocote) in the day and a silicon gel sheet overnight or twice daily application of the topical gel [35]. Patients preferred the ease of application of the topical gel.

Pressure Garments

Pressure garments are used most regularly with scars following burn, extensive soft tissue injuries, skin grafts or in the case of web creep [36]. They form a 'second skin' by exerting the pressures on the deep skin layers as the original outer layers of skin would have done. This helps to prevent the new skin from being formed in spiral bundles (producing hypertrophic scars).

Elastomers

Elastomers can be moulded to produce an imprint on any skin which has an awkward or concave shape. This can then be worn to increase pressure on the skin. Some elastomers are infused with silicone. They should be replaced every few months in order to maintain a good imprint. Increased compression can be gained with a splint or pressure garments.

Specific Rehabilitation for Capsular Tightness or Tendon Tethering

The following factors will help the therapist to prioritise the most appropriate modality: the stage of tissue repair, the degree of pre treatment stiffness or tethering, the time since diagnosis or



Fig. 12.5 Passive composite finger flexion

injury, the patient's age, work, lifestyle and compliance and the therapist's knowledge and resources.

Rehabilitation to Gain Capsular Tissue Elongation for Intra-Articular Tightness

All rehabilitation for capsular tightness should concentrate on achieving end of range stretch or placing a prolonged stretch on the tight tissues.

Gaining Range of Motion Passive Exercises/Stretches

Passive stretches should be done for a prolonged period of time, with a low to moderate amount of tension [16]. The patient needs to understand the difference between the sensations of stretch and pain. Forced manipulations are contraindicated because they result in pain, micro trauma to tissues and increase swelling (Fig. 12.5) [37].

Following a low load prolonged stretch into flexion carried out by the therapist, it is possible to tape the finger into the improved flexed position. This way, the patient can experience an effective and safe stretch for longer (without having to hold on to the finger). Begin with suggesting 10–15 min of taping and gradually build it up. This stretch can also be achieved using a variety of splints or straps (often using velstretch, Lycra or neoprene) (Fig. 12.6a–c).

Joint Mobilisations

Specific manual joint accessory movements are very effective for capsular tightness [8]. These may be passive oscillatory movements, two or three per second of small or large amplitude, applied anywhere in the range; or sustained stretch with or without tiny amplitude oscillations at the limit of range [38]. When stretching a stiff joint to regain range, treatment movements include spin, roll and slide which are normal for that particular joint. They may be used for assessment or for treatment of stiffness. These techniques require knowledge of surface anatomy, training and practice.

Exercises

These are less effective for capsular tightness than for tendon glide, but will build muscle power and maintain soft tissue glide. Isotonic exercises will work on core muscle strength without requiring active or full ROM. Active exercises may be most effective following heat and taping and then targeted exercises.

Activity

Activity rehabilitation chooses games, activities or functional tasks that will target the joints involved. These joints can be further isolated by using blocking splints that only allow the stiffer joints to move, or using a sustained grip on an object which will improve the elastic stretch on tissues.

Lycra or Neoprene Sleeves

Cylindrical sleeves with reinforcement dorsally may be effective in reducing fixed flexion deformities in the digit, by producing a prolonged stretch with compression [39, 40] (Fig. 12.7a, b).

Splintage

Flowers and LaStayo (1994) [5] concluded, from a study of total end range time, that, if a joint is held at its moderately lengthened position for a significant time, the tissues will lengthen [5]. They also proved that the amount of lengthening is directly proportional to the time spent at total end range (TERT). Such growth of the dense

12 Post-Traumatic Contracture

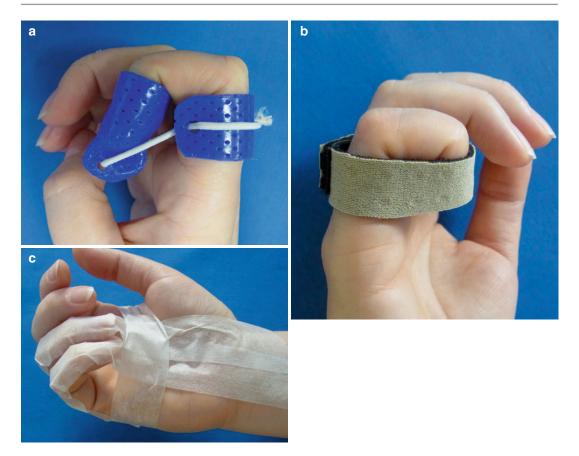


Fig. 12.6 Techniques of improving finger flexion. (a) Dynamic IPJ flexion splint. (b) Elastic IPJ flexion strap. (c) Micropore taping into flexion

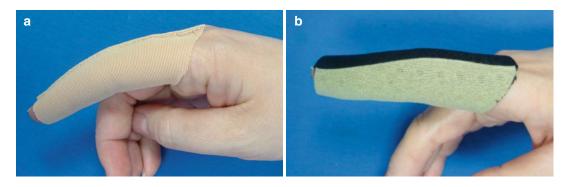


Fig. 12.7 Lycra finger digisleeve and neoprene tube. (a) Lycra digisleeve. (b) Neoprene finger tube

connective tissue around the joint allows for increased movement. Splints must be removed regularly to allow range of movement exercises and must always work within the clinically safe range of force [41]. The duration of splinting must be considered. Prosser (1996) reports the average splint wearing time for 20 patients with PIP joint contractures to be 6–14 h a day for 3–5 months [42]. This is because collagen turnover

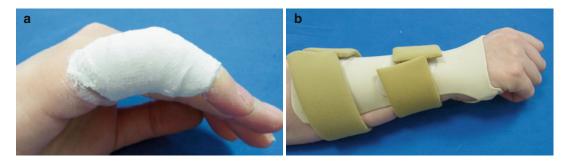


Fig. 12.8 Thermoplastic wrist serial static splint and POP PIP joint cast. (a) Plaster of Paris PIPJ serial cast. (b) Dorsal leverage extension wrist splint

is approximately 6 weeks and an acceptable clinical improvement may take several cycles of collagen turnover. Clinically, 3–6 months of splinting is recommended.

Static Splintage

A hand which is oedematous or being rested for a prolonged period can be rested in a thermoplastic splint. The position of safe immobilisation is the intrinsic plus position (wrist extension, MCPJ flexion and IPJ extension), as this prevents the collateral ligaments tightening. In the presence of oedema, strapping on the splint should be in a figure of eight pattern or a wrap, so that pressure is distributed over a large area. Individual joints can be splinted to prevent contractures forming, or where contractures have already formed, serial splinting can be used, periodically altering to gradually reduce the contracture.

Serial Static Splinting

Serial static splints are those that hold a specific position with prolonged gentle stretch for a length of time (3–5 days) and are then exchanged for another identical splint as tissue changes but held at a slightly improved angle. They are particularly effective in joint or tissue contractures as tissue is encouraged to "grow" and rearrange patterns of collagen rather than stretch, producing a long term effect and improved tissue length. Plaster of Paris is often used in this manner to gain composite extension in the hand and wrist (Tribuzi 1995) [43] or specifically at the PIPJ [43, 44]. Circumferential, dorsal leverage and volar thermoplastic splints may also be effective (Fig. 12.8a, b)

Static Progressive Splinting

This differs from static splinting, as a force is applied to the joint, rather than in static splinting where a joint is held in a particular position. The torque can be applied to the joint using a variety of materials including Velcro straps or an inelastic static line [45].

Glasgow et al. (2008) [46] proposed that static progressive splinting may not be the most effective splinting type for elongation of collagen in stiff joints. They suggest that this may be due to reliance on the patient to make the adjustments which may not be continually made in accordance to the viscous response and no adjustments over night are possible.

Dynamic Splinting

Dynamic splinting is an effective way of applying low to moderate amounts of tension to a stiff joint over a prolonged period of time. Dynamic splints use elastic methods to place torque on to the joint. Glasgow et al. (2008) [46] concluded that dynamic splints are able to respond to increases and decreases of viscous resistance and, therefore, may be most appropriate in contracture [46]. Menzes and Buck Willis (2011) confirmed substantial gains in ROM, with prolonged end range time in treating wrist and elbow contractures (Fig. 12.9) [47].

Casting Motion to Mobilise Stiffness (CMMS)

Judy Colditz (2011) suggests that the stiff hand develops poor habits of movement and substitutes patterns of movement promoting the loosest joints [8]. For example, if there is restricted movement



Fig. 12.9 Dynamic supination splint

at the IP joints, the MCPJ will often flex using the lumbrical muscles more than the long finger flexors. This encourages imbalance. The exact opposite is also seen. If there is tightness into flexion at the MCP joint, patients are often seen using increased long finger flexor movement to produce IP flexion and therefore reduce the amount of use of the lumbricals. CMMS promotes movement in the stiffest joints, by immobilising joints that have good range. This forces the patient to use their power to move stiff joints and encourages the motor patterning of this movement. Midgley (2010) [48] confirmed that CMMS was beneficial in managing patients post Dupuytren's release contracture [48].

Clinical Pearl

Most capsular contractures will need a combination of many modalities over a prolonged time. A systematic review of treatment for upper limb contractures showed that active exercises, joint mobilisations and splinting were most effective [49].

Rehabilitation to Gain Tendon Glide

Tissue glide occurs within structures themselves, such as ligaments and tendons, but also between these structures at fascial interfaces, with tendon or synovial sheaths or between bone and tendon. Disruption and scarring of any aspect will reduce the available glide. Fractures with scarring of the periosteum may tether to all surrounding tissue; for example, a proximal phalanx fracture may tether all circumferential tissue involving flexors, extensors, ligaments, intrinsic and extrinsic tendons and fascial gliding layers. Tendon glide of >5 mm or PIPJ ROM of 0–40° gained in the first 4 weeks post injury or surgery has been shown to be a positive prognostic sign for reduced tethering [50].

Gaining Tendon Glide Active Exercises

Active exercises can be aided by heating the tissues prior to movement to improve the potential for elongation. Methods for this have been discussed earlier in the chapter.

Tendon gliding exercises, which encourage full tendon excursion, should be performed. Consider methods of gaining maximum glide for the finger and thumb flexors and extensors in isolation from each other, as well as differential glide of the intrinsic and extrinsic muscles. For example, holding the interphalangeal joints in flexion and slowly extending the metacarpophalangeal joints to hyperextension will maximise active glide of Flexor Digitorum Profundus with passive glide of the interossei and lumbricals (Fig. 12.10a, b).

Maintaining a power grip and moving the wrist through flexion and extension will maximise extrinsic tendon glide.

Exercises must be carried out regularly throughout the day and need to be repeated slowly. Graded exercises, such as foam sponges or putty, can be started, as long as they do not cause an increase in pain or swelling. Active exercise must also include full range of motion of all upper limb joints. Muscle strength must not be forgotten. Joint movement cannot be maintained if muscles cannot put them through the available range. A graded exercise programme is recommended, incorporating resisted activity and function. Consider using the position of the wrist and/or MCP joints to increase tendon glide, to alter glide to inner or outer range of pull. Exercises such as "place and hold" will produce a strong contraction in inner range. FDP glide with the wrist in full extension is likely to gain a further 2-3 cm of glide [51]. Exercises may be composite or isolated to work individual muscles in isolation or a combination of muscles to produce movement. Muscles may be worked in a concentric manner to

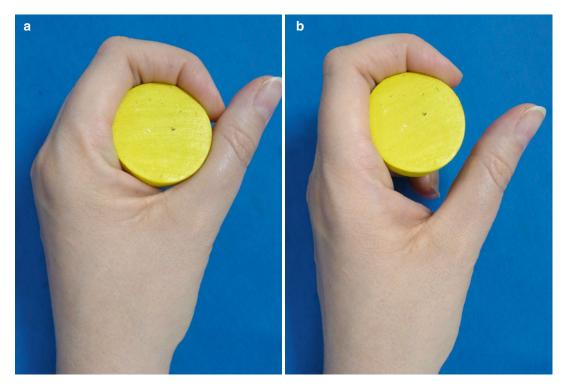


Fig. 12.10 Techniques of gaining tendon glide. (a) Composite flexion around an object. (b) Intrinsic minus increasing intrinsic passive stretch

produce a shortening of the muscle tendon unit in contraction or in eccentric to produce a lengthening of the unit, as the muscle controls the position of the wrist, hand or finger.

Resisted exercises will increase the force of the muscle pull and therefore the pull of the tendon on adhesions. These may include exercises such as Velcro rollers, thera-band and elastic resistance, thera-putty and splints. Repetitive movements will again produce a shearing force of the tendon or gliding tissues on adhesions.

Activity

Using the hand in a wide range of functional and/or rehabilitation activities will gain soft tissue glide, often with decreased pain, as the patient is distracted by the activity. Activities may be chosen and graded specifically for individual or composite tissue glide. These may be rehabilitation activities or incorporated in functional or leisure activities. Blocking splints again may be necessary to isolate the movements required (Fig. 12.11a–c).

Surgical Techniques

- 1. Arthroscopic release at the wrist.
- 2. Dorsal release of the metacarpophalangeal joint (MCP).
- 3. Volar plate release of the proximal interphalangeal joint (PIP).
- 4. Tenolysis flexors and extensors

Whilst these procedures can be undertaken as described in isolation, it is important to remember that they are often undertaken in conjunction with some form of reconstruction, e.g. extensor tendon realignment at the metacarpophalangeal joint and central slip reconstruction at the proximal interphalangeal joint.

Arthroscopic Release of Capsular Contracture at the Wrist

With informed consent and usually under general anaesthetic, a patient's arm is suspended in finger traps with 5 lb (2kg) of counter traction. A tourniquet is also in situ. Under strict asepsis, using a

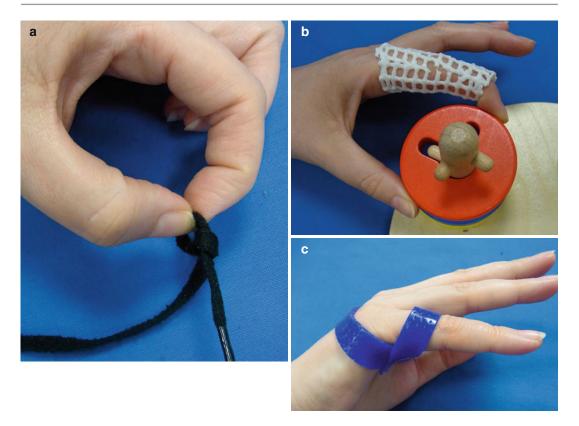


Fig. 12.11 Active techniques of regaining finger flexion. (a) Undoing a knot encourages FDP function. (b) A splint to block the PIPJ isolates FDP action. (c) A splint to prevent MCPJ hyperextension isolates IPJ extension

2.7 scope, both radio carpal and mid-carpal arthroscopies are undertaken. Generally these are undertaken through 3/4 and 6R portals plus a standard mid-carpal portal. A complete intraarticular examination of this joint is performed. Once this is complete, it is possible to proceed to the capsular release. Generally this will involve a capsular release at the radio-carpal joint. Again the two portals utilized are the 3/4 and 6R. In many incidences, particularly with a tight contracture, we also will use an additional 6U portal. With the arthroscope in the 3,4 portal, a soft tissue resector (2.9 mm full radius blade) can be inserted in the 6R or 6U and an ulnar capsule release undertaken. This can be undertaken on both the volar and dorsal surfaces. Switching the resector and the scope allows the release to continue on the radial side (Fig. 12.12). The author would also recommend the use of a fluid management system set at low pressure. To aid the release,



Fig. 12.12 Arthroscopic view of anterior capsular release

manipulations of the wrist can be undertaken at the pertinent stages, the counterweight having been removed to allow this to be undertaken. On the dorsal side, the release tends to be somewhat easier and less hazardous. Specifically as only the extensor tendons are at risk. On the volar side, however, there are more important neurovascular structures which could theoretically be damaged. Generally, however, using small resectors and occasionally arthroscopic scissors, it should be possible to protect any extrinsic tendons, nerves or blood vessels. It is important, however, that the procedure should not continue for too long, and certainly no more than 60 min, to prevent excessive saline extravasation.

Hand Therapy Post Capsular Release

Following the procedure, local anaesthetic is injected into the wrist joint and active and passive mobilisation begun immediately to maintain the range gained in the operation. A static, serial static or dynamic wrist/forearm splint may be necessary to maximise range, particularly if the contracture is long term. Light function may be begun immediately and return to all activities graded as pain and range of motion allows. All other modalities for capsular tightness management need to be considered.

Outcome

In 1988 Hanson et al. reported a number of cases of adhesive capsulitis affecting the wrist joint [52]. He also described various clinical and radiological features. Treatment took the form of closed manipulation under general anaesthetic in four patients with some improvement in range of motion. In one case there was a fracture of the ulna.

Reports on the outcome after arthroscopic capsular release at the wrist are few and far between. Dorsal capsular release was described by Bain et al. (2000) and, subsequently, in a further report in 2008 with results in 12 patients [53, 54]. In 9 of the 12 patients there was a 75 % increase in range of motion and grip strength. The other 3 patients reported between 25 and 50 % improvement. It is of note that rehabilitation that is active early mobilization began in the immediate post-operative period. In this study the authors reported no significant complications. Hattori et al. (2006) [55] reported their results in 11 patients and reported a significant

improvement in all cases with a 22° increase in arc of motion [55].

With regard to the distal radio-ulna joint, while incongruity after distal radial fracture remains the commonest cause of loss of forearm rotation, scarring and contracture of the distal radio-ulna joint capsule can also lead to a reduction in this movement. Kleiman and Graham (1998) recommended, in patients who had restored osseous-anatomy after trauma, a DRUJ capsulotomy to improve forearm rotation [56].

Dorsal Release of the Metacarpophalangeal Joint (MCP)

Again with informed consent, either under a general anaesthetic or regional block, the patient's arm is prepped and draped. The operation is undertaken with a tourniquet in situ. Through a longitudinal incision overlying the MCP joint, the long extensor tendon is identified. This is then released along both its radial and ulna aspects and freed from the underlying capsule. However, the tendon is often adherent and may have to be released by sharp dissection. Once free, the tendon is retracted and the dorsal capsule exposed. This is then released in a transverse fashion; the release being undertaken whilst the joint is manipulated into flexion (Fig. 12.13). At this stage, the articular surfaces of the MCP joint can be inspected. If there is significant damage, then joint replacement should be considered. The capsular release then continues down the radial and ulna aspects of the metacarpal head, including the collateral ligaments, until full flexion of the joint is obtained. At this stage it is important that the articular surfaces glide into flexion rather than hinges. For the latter, it may be that an additional volar plate release is required. Finally, if there is intrinsic tightness, the intrinsics should also be released.

For closure, the extensor tendon is realigned over the centre of the metacarpophalangeal joint and the capsule is left open. The skin is closed and the hand splinted in the safe position that is with the metacarpophalangeal joints flexed to $70-90^{\circ}$. Local anaesthetic infiltration into the wound and operative site is recommended.

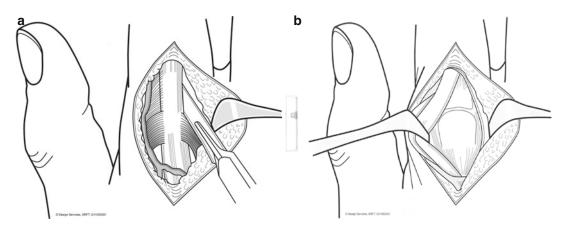


Fig. 12.13 Dorsal capsular release of the metacarpophalangeal joint

Hand Therapy Following MCP Dorsal Capsular Release

The patient should then begin gentle therapy at 1-2 days, with increased intensity after 5 days once the inflammatory reaction has settled. Tendon glide is vital, particularly for the extensors and intrinsic muscles, with differential glide of each tendon/muscle. The splint should be worn 24 h for the first 2-3 weeks with removal every 1-2 h for active and passive exercises, tendon glide and activity rehabilitation. Exercises carried out 10-15 min every 2 h are effective [57]. Intensive oedema and scar management is important. Early gains in ROM and tendon glide often reduce after 3–4 weeks as the scar tissue thickens and intensive rehabilitation in the hand therapy department and at home is needed. Resisted activities and exercises are incorporated as the wound heals and inflammation reduces after 3-4 weeks. Functional activities need to also be part of rehabilitation with blocking splints maximising the tissue and joint movement. A balance between maintaining flexion at the MCP joints and decreasing extensor lag needs to managed with stretch overnight and active tendon and tissue glide in the day.

Outcome

In 1974 Buch reported his results of MCP capsulotomy and on occasions supplemented by a skin graft [58]. Following a course of therapy he noted that a significant number of patients gained between 30 and 80° of flexion. Leroy Young et al., in a large series published in 1978, found that in the majority of patients the stiffness was due to collateral ligament tightness [59]. Following release and an intense course of therapy, they reported long term improvement with an average flexion of 48°; the best results being obtained in patients who sustained a closed fracture or a laceration.

More recently Rozmaryn and Wei (1999) [60] advocated that these releases could be undertaken arthroscopically [60].

Volar Release of the Proximal Interphalangeal Joint (PIP)

Again under either general or regional anaesthesia and with strict asepsis and a tourniquet in situ, the PIP joint is approached from the volar side. The usual skin incision would be a Brunner or zig-zag incorporating the flexor crease. Flaps are elevated and the flexor sheath identified. Both neurovascular bundles are identified and retracted. The flexor sheath is then incised transversally at the level of the PIP joint. This is between the A3 and A4 pulleys. The flexor tendons are then identified and inspected. If they are found to be in a satisfactory condition they are retracted, allowing the volar plate to be visualised. This is then released from the proximal phalanx at its insertion. This allows the plate to slide distally (Fig. 12.14).

If this is insufficient to correct the contracture then collateral ligament releases can be undertaken. Again these structures are released from

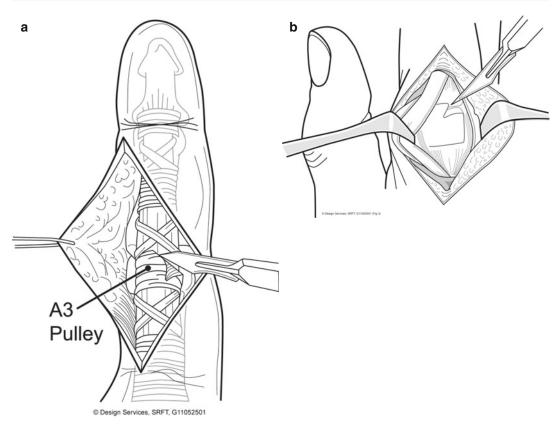


Fig. 12.14 Release of volar plate of proximal interphalangeal joint

their attachment to bone, usually at the proximal phalanx. The releases are aided by gentle manipulation. At this time it is usually possible to inspect the articular surfaces. Again, and as at the MCP joint, if they are badly damaged arthroplasty should be considered. Returning to the flexor tendons, if they are adherent they should also be released. Indeed, the flexor digitorum superificalis tendon can, if necessary, be excised. In addition it may be that following the correction the distal interphalangeal joint assumes a hyper-extended position, due to tightness of the lateral bands. If this is the case, these bands can be released through a separate dorsal incision over the middle phalanx.

At the end of this procedure, once haemostasis has been obtained, the wounds are closed and then infiltrated with a local anaesthetic. Usually it is possible to directly close the skin, however, if not, then an advancement flap can be utilized. For small defects however, it is perfectly acceptable to allow them to heal by primary intention. With the dressing in place, the hand is splinted with the PIP joint in extension.

Hand Therapy Following Volar Release of the PIP Joint

Therapy starts between 3 and 5 days, unless flexor tenolysis has been performed at the same time. Evans et al. (2002) [61] showed that wound tension and overly aggressive early therapy following PIPJ release post Dupuytren's faciectomy produced an increase in scar tissue and inflammatory response [61].

As for any capsular or joint release, splintage needs to be 24 h initially to maintain ROM gains, but removed 1–2 h for active and passive exercises, tendon glide and activity rehabilitation [57]. Saar and Grothaus (2000) [62] and Crowley and Tonkin (1999) [63] showed that longer periods of splinting PIPJ was more beneficial and that 3–6 months of serial or dynamic PIPJ

splinting could gain extension beyond surgical gains [62, 63]. Clinical reasoning needs to balance flexion and tendon glide with joint extension gains and all modalities considered for capsular tightness.

Outcome

The results of surgery to release contractures of the proximal interphalangeal joint can be quite variable. It is for this reason that more often than not conservative treatment is recommended at least in the first instance. Published results also vary, both with regard to the exact technique undertaken but also the range of pathology. As a consequence, the scope of recommended procedures ranges from isolated percutaneous release of the accessory collateral ligament, (Stanley et al. 1986) [64], flexor tenolysis, extensive open excision of all supporting soft tissues and proximal skin release with grafting or flap cover [64]. Generally, however, a sequential approach, preferably under local anaesthetic, is advised, allowing the patient to actively move the finger during the procedure to assess progress. In most series a volar approach is utilized, although many authors have used a mid-lateral. Post-operatively, many surgeons also use a K-wire to fix a joint in extension for a short period of perhaps 2–3 weeks.

In 1979 Watson et al. reported successful results in a large series of 115 fingers with contractures of various aetiologies [65]. Around the same time, Young et al. (1978) [59], again in a large series, reported an average 42° improvement in movement following a sequential release of structures [59]. Gould et al. at the same time in a similar study also showed some improvement although not as marked [66]. More recently Ghidella et al. (2002) [67], in a longer term follow up, showed a more modest improvement, added to that 30 % of patients actually lost motion [67]. Finally, Manset and Delprat in 1992, in a series of 135 post-traumatic causes of PIP contracture, reported no significant improvement, indeed 14 % again had worsened [68].

In 1993 Diao and Eaton reported their results following the complete excision of scarred collateral ligaments for the treatment of contracture [69]. Added to this they also undertook distal palmar plate releases, extensor tenolysis and flexor sheath release. Following a course of therapy the average range of motion increased from 38 to 78°, with no instances of instability. They felt this more radical approach was justified. Brüser et al. in (1999) [70] compared mid-lateral to palmar approaches. Results were better in the former [70].

Finally Inoue (1991) [71] reported his results of lateral band release for an extension contracture of the proximal interphalangeal joint [71] Following surgical release of the lateral band from the central slip, flexion improved significantly.

In summary, whilst open release can lead to improved extension of the proximal interphalangeal joint, this can be at the cost of flexion. If, however, this shift in flexion/extension arc results in a more functional range, then obviously this will be beneficial to the patient.

External Fixators

As with splinting and serial casting, external fixation can be used to correct contractures across the proximal interphalangeal joint. At this time, there are a number of commercial devices in use, although unfortunately there are few independent published reports on their efficacy. A number of case series have reported favourable results although numbers are small.

Conclusions/Personal View

Normal motion of any joint requires normal bony alignment, intact articular surfaces, unimpeded tendon gliding and integrity of the surrounding soft tissues, particularly the collateral ligaments and the volar plate. A deficiency in any one or all of these can lead to contracture and/or deformity. In most cases this can be prevented by appropriate splintage, surgery and early mobilization. In established cases, however, initial treatment should be non-operative, using intensive therapy incorporating a wide range of modalities related to the specific tissues involved, often over many months. Many patients will gain an acceptance of functional range and return to maximal functional activities. Only if this is unsuccessful should surgery be considered.

For those who do not gain sufficient functional range, therapy will include careful pre-operative patient assessment and education regarding prognosis and benefits to ensure realistic patient expectations. Patient satisfaction often relates more to achieving an expectation than to a specific ROM or outcome measure. An intensive therapy and home programme, over 4–6 months, will maximise outcomes, with ongoing motivation being vital. In most instances results indicate that it is possible to improve range of motion or at the very least make the hand more functional.

Clinical Pearl

While all patients with contracture benefit from the attention of therapy, the role of surgical intervention is not always clear. The surgery itself may inflict further trauma to an injured area and can result in otherwise normal tissues being damaged. As a consequence, therefore, the surgeon must balance the relative benefits and risks of intervention.

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References

- Weeks PM, Wray C, Kuxhaus M. The results of nonoperative management of stiff joints in the hand. Plast Reconstr Surg. 1978;61(1):58–63.
- Kuczynski K. The proximal interphalangeal joint, anatomy and causes of stiffness in the fingers. J Bone Joint Surg. 1968;50(3):656–63.
- Akeson WH, Amiel D, Abel MF, Garfin SR, Woo SL. Effects of immobilization on joints. Clin Orthop Relat Res. 1987;219:28–37.
- Brand PW, Hollister A. Clinical mechanics of the hand. 2nd ed. St. Louis: Mosby; 1992.
- Flowers K, LaStayo P. Effect of total end range time on improving passive range of motion. J Hand Ther. 1994;7:150–7.
- Shin AY, Amadio PC. Stiff finger joint's. In: Green DP, Hotchkiss RN, Pederson WC, Wolfe SW, editors.

Green's operative hand surgery. 5th ed. Philadelphia: Elsevier Churchill Livingstone; 2005. p. 417–59.

- Lewis ES. Finger circumference measurements: inter-and intra-rater reliability. J Hand Ther. 2010;15(3):69–76.
- Colditz JC. Therapists management of the stiff hand: chapter 75. In: Skirven TM, Osterman AL, Fedorczyk JM, Amadio PC, editors. Rehabilitation of the hand and upper extremity. Philadelphia: Elsevier Mosby; 2011. p. 894–921.
- Marx RG, Bombardier C, Wright J. What do we know about the reliability and validity of physical examination tests used to examine the upper extremity? J Hand Surg. 1999;24A:185–93.
- Medical Research Council. Aids to the examination of the peripheral nervous system, memorandum, no. 45. London: Her Majesty's Stationery Office; 1981.
- Van Veldhoven G. Intrinsic and extrinsic tightness the importance of the 'pre-splint' test to determine MCP inclusion and position in orthotics. Br J Hand Ther. 2000;5(3):75–6.
- Hulme MC, Gellerman H, McKellop H, Brumfield Jr RH. Functional range of motion of the joints of the hand. J Hand Surg. 1990;15A:240–3.
- Woodworth JA, McCullogh MB, Grosland NM, Adams BD. Impactof simulated proximal interphalangeal arthrodeses of all fingers on hand function. J Hand Surg. 2006;31A(6):940–7.
- Ryu JY, Cooney 3rd WP, Askew LJ, An KN, Chao EY. Functional ranges of motion of the wrist joint. J Hand Surg. 1991;16A(3):409–19.
- Usuba M, Miyanaga Y, Miyakawa S, et al. Effect of heat in increasing the range of knee motion after development of a joint contracture. Arch Phys Med Rehabil. 2006;87:247–53.
- Hardy M, Woodall W. Therapeutic effects of heat, cold, and tissue stretch on connective tissue. J Hand Ther. 1998;11:148–56.
- Allen RJ. Physical agents used in the management of chronic pain by physical therapists. Phys Med Rehabil Clin N Am. 2006;17:315–45.
- Artzberger SM, Priganc VW. Manual edema Mobilisation: an edmea reduction technique for the orthopaedic patient: chapter 65. In: Skirven TM, Lee Osterman A, Fedorczyk JM, Amadio PC, editors. Rehabilitation of the hand and upper extremity. Philadelphia: Elsevier Mosby; 2011. p. 868–81.
- Villeco JP. Edema: therapist's management: chapter 63. In: Skirven TM, Lee Osterman A, Fedorczyk JM, Amadio PC, editors. Rehabilitation of the hand and upper extremity. Philadelphia: Elsevier Mosby; 2011. p. 845–57.
- Brennan MJ, Miller LT. Overview of treatment options and review of the current role and use of compression garments, intermittent pumps, and exercise management of lymphedema. Cancer. 1998;83 (12 suppl Am):2821–7.
- Hunter J, Mackin E. Edema techniques of evaluation and management. In: Hunter J, Mackin E, Callahan A, editors. Rehabilitation of the hand: surgery and therapy, vol. 1. 4th ed. St. Louis: Mosby; 1995.

- Prentice WE. Guidelines for using therapeutic modalities in rehabilitation. In: Prentice WE, editor. Therapeutic modalities in sports medicine. 3rd ed. St. Louis: Mosby; 1994.
- Low J, Reed A. Electrotherapy explained: principles and practice. Oxford: Butterworth Heinemann; 1994.
- Hubbard TJ, Aronson SL, Denegar CR. Does cryotherapy hasten return to participation? A systematic review. J Athl Train. 2004;39(1):88–94.
- Palmada M, Shah S, O'Hare K. Hand oedema: pathophysiology and treatment. Br J Hand Ther. 1999; 4(1):26–32.
- Cote DJ, Prentice Jr WE, Hooker DN, Shields EW. Comparison of three treatment procedures for minimizing ankle sprain swelling. Phys Ther. 1988;68(7): 1072–6.
- Sorenson MK. The edematous hand. Phys Ther. 1989; 69:1059.
- Weissleder H, Schuchhardt C. Lymphedema diagnosis and therapy. 2nd ed. Bonn: Kagerer Kommunikation; 1997.
- Goldin J, et al. The effects of Diapulse on the healing of wounds: a double blind randomised controlled trial in man. Br J Plast Surg. 1981;34:267–70.
- Pennington GM, Danley DL, Sumko MH. Pulsed, non-thermal high-frequency electromagnetic energy (Diapulse) in the treatment of grade I and grade II ankle sprains. Mil Med. 1993;158(2):101–4.
- Kase K, Wallis J, Kase T. Clinical therapeutic applications of the kinesiotaping method. 2nd ed. Tokyo: Kenzo Kase; 2003.
- Donnelly CJ, Wilton J. The effect of massage to scars on active range of motion and skin mobility. Br J Hand Ther. 2002;7(1):5–11.
- Rodger J. The role of ultrasound in the treatment of surgically repaired tendon injuries of the hand: a literature review. Br J Hand Ther. 2000;5(2):43–5.
- Farquhar K. Silicone gel and hypertrophic scar formation: a literature review. Can J Occup Ther. 1992;59(2): 78–86.
- Chernoff WG, Cramer H, Su-Huang S. The efficacy of topical silicone gel elastomers in the treatment of hypertrophic scars, keloid scars, and post-laser exfoliation erythema. Aesthetic Plast Surg. 2007;31: 495–500.
- Carr Collins JA. Pressure technique for the prevention of hypertrophic scars. Clin Plast Surg. 1992;19: 733–43.
- Brand PW, Hollister AM. Clinical mechanics of the hand. 3rd ed. St. Louis: Mosby; 1999.
- Maitland GD. Peripheral manipulation. 3rd ed. Oxford: Butterworth-Heinmann; 1991.
- Clark EN. A preliminary investigation of the neoprene tube finger extension splint. J Hand Ther. 1997;10(3):213–21.
- Kennedy S, Peck F, Stone J. The treatment of interphalangeal joint flexion contractures with reinforced lycra finger sleeves. Br J Hand Ther. 2000;5(2):46–8.
- 41. Van Lede P, Van Lede G. Therapeutic hand splinting: a rational approach. Antwerp: Provan; 1998.

- Prosser R. Splinting in the management of proximal interphalangeal joint flexion contractures. J Hand Ther. 1996;9:378–86.
- 43. Tribuzi SM. Serial plaster splinting: chapter 96. In: Hunter JM, Mackin EJ, Callahan AD, editors. Rehabilitation of the hand: surgery and therapy. 4th ed. St. Louis: CV Mosby Co; 1995. p. 599–1608.
- 44. Bell-Krotoski JA. Plaster cylinder casting for contractures of the interphalangeal joints: chapter 97. In: Hunter JM, Mackin EJ, Callahan AD, editors. Rehabilitation of the hand: surgery and therapy. 4th ed. St. Louis: CV Mosby Co; 1995. p. 1609–16.
- Schultz-Johnson K. Static progressive splinting. J Hand Ther. 2002;15(2):163–78.
- 46. Glasgow C, Tooth L, Fleming J. Which splint? Dynamic versus static progressive splinting to mobilize stiff joints in the hand. Br J Hand Ther. 2008;13(4):104–10.
- Menzes M, Willis Buck F. Dynamic splinting for paediatric contracture reduction of the upper limb. J Hand Ther. 2011;16(4):107–10.
- Midgley R. Use of casting motion to mobilize stiffness to regain digital flexion following Dupuytren's fasciectomy. J Hand Ther. 2010;15(2):45–51.
- Michlovitz SL, Harris BA, Watkins MP. Interventions for loss of range of motion of the upper extremity: a systematic review. J Hand Ther. 2004;17(2):118–31.
- Freeland AE, Hardy MA, Singletary S. Rehabilitation for proximal phalanx fractures. J Hand Ther. 2003; 16:129–42.
- Amadio PC. Friction of the gliding surface. Implications for tendon surgery and rehabilitation. J Hand Ther. 2005;18:112–9.
- Hanson EC, Wood VE, Thiel AE, Maloney MD, Sauser DD. Adhesive capsulitis of the wrist. Clin Orthop Relat Res. 1988;234:51–5.
- Verhellen R, Bain GI. Arthroscopic capsular release for contracture of the wrist: a new technique. Arthroscopy. 2000;16(1):106–10.
- Bain GI. Arthroscopic dorsal capsular release in the wrist: a new technique. Tech Hand Up Extrem Surg. 2008;12(3):191–4.
- Hattori T, Tsunoda K, Watanabe K, Nakao E, Hirata H, Nakamura R. Arthroscopic mobilization for contracture of the wrist. Arthr J Arthr Relat Surg. 2006; 22(8):850–4.
- Kleinman WB, Graham TJ. The distal radioulnar joint capsule: clinical anatomy and role in posttraumatic limitation of forearm rotation. J Hand Surg. 1998;23A:588–99.
- 57. Cannon N. Post operative management of metacarpophalangeal joint and proximal interphalangeal joint capsulectomies: chapter 68. In: Skirven TM, Lee Osterman A, Fedorczyk JM, Amadio PC, editors. Rehabilitation of the hand and upper extremity. Philadelphia: Elsevier Mosby; 2011. p. 922–38.
- Buch VI. Clinical and functional assessment of the hand after metacarpophalangeal capsulotomy. Plast Reconstr Surg. 1974;53(4):452–7.
- Young VL, Jr Wray RC, Weeks PM. The surgical management of stiff joints in the hand. Plast Reconstr Surg. 1978;62(6):835–41.

- Rozmaryn LM, Wei N. Technical note: metacarpophalangeal arthroscopy. Arthr J Arthr Relat Surg. 1999;15(3):333–7.
- Evans R, Dell P, Fiolkowski P. A clinical report of the effect of mechanical stress on functional results after fasciectomy for Dupuytren's contracture. J Hand Ther. 2002;15(4):331–9.
- Saar J, Grothaus P. Dupuytrens: an overview. Plast Reconstr Surg. 2000;106(1):125–34.
- Crowley B, Tonkin MA. The proximal interphalangeal joint in Dupuytren's disease. Hand Clin. 1999;15(1):137–47.
- 64. Stanley JK, Jones WA, Lynch MC. Percutaneous accessory collateral ligament release in the treatment of proximal interphalangeal joint flexion contracture. J Hand Surg Br. 1986;11(3):360–3.
- Watson HK, Light TR, Johnson TR. Checkrein resection for flexion contracture of the middle joint. J Hand Surg Am. 1979;4:67–71.

- Gould JS, Nicholson BG. Capsulectomy of the metacarpophalangeal and proximal interphalangeal joints. J Hand Surg Am. 1979;4:482–6.
- Ghidella SD, Segalman KA, Schuler Murphey M. Long-term results of surgical management of proximal interphalangeal joint contracture. J Hand Surg Am. 2002;27:799–805.
- Mansat M, Delprat J. Contractures of the proximal interphalangeal joint. Hand Clin. 1992;8:777–86.
- Diao E, Eaton RG. Total collateral ligament excision for contractures of the proximal interphalangeal joint. J Hand Surg Am. 1993;18A:395–402.
- Brüser P, Poss T, Larkin G. Results of proximal interphalangeal joint release for flexion contractures: midlateral versus palmar incision. J Hand Surg Am. 1999;24A:288–94.
- Inoue G. Lateral band release for post-traumatic extension contracture of the proximal interphalangeal joint. Arch Orthop Trauma Surg. 1991;110:298–300.

Neurological Contractures: The Spastic Upper Limb

13

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Keywords

- Cerebal palsy Tetraplegia Assessment Functional deficit Investigations
- Surgery
 Rehabilitation
 Results

Introduction

Spasticity is characterized by muscle hypertonia, caused by a hyperactive stretch reflex mechanism. It is linked to a central neurological impairment involving the pyramidal tract.

It may occur in several circumstances:

- in children, *cerebral palsy* is usually secondary to foetal or perinatal encephalopathy, and occurs less frequently than brain damage during childhood.
- in adults, it is usually related to *hemiplegia*, whether vascular (stroke) or traumatic (head injury)
- tetraplegia is often associated with spasticity of the lower limbs. Spastic involvement of the upper limbs is rare and is most frequently associated with incomplete tetraplegia.

Spasticity is rarely an isolated feature. The clinical picture generally includes other neurological and orthopaedic impairments which need to be carefully assessed together with the spasticity.

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Cerebral Palsy

Cerebral palsy is a general term that includes all the sequelae of infantile encephalopathies occurring during the perinatal period, or during infancy.

In 2007, a group of experts in the field of CP gathered for an International Workshop on Definition and Classification of CP, and published the following definition of CP [1]: "Cerebral palsy describes a group of permanent disorders of the development of movement and posture, causing activity limitation, that are attributed to non-progressive disturbances that occurred in the developing foetal or infant brain. The motor disorders of cerebral palsy are often accompanied by disturbances of sensation, perception, cognition, communication and behaviour, by epilepsy, and by secondary musculoskeletal problems"

Since Little [2], it was thought to be mostly linked to perinatal asphyxia, although recent studies indicate that this factor is responsible for only 5–10 % cases of cerebral palsy. Other causes may be related to the foetus itself (gestational age at birth, birth weight, growth restriction), or to the mother (neurologic disorders, infertility treatment, antepartum infection, thyroid disease) [3].

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Postnatal acquired cerebral palsy accounts for 15 % cases, and is mostly due to meningoencephalitis, head injury, and cerebro-vascular accidents.

Infantile cerebral hemiplegia is the main type of cerebral palsy. It is characterized by unilateral cortical and subcortical involvement, particularly in the pyramidal tract, which causes various degrees of spasticity and motor deficit in the contralateral limbs. It can be associated with epilepsy; mental retardation; and speech, vision or hearing deficits. It manifests progressively during growth, but once established, follows a non progressive course, which makes it amenable to surgical treatment in selected cases.

Clinical Pearl

Cerebal palsy encompasses a group of conditions which have resulted in the permanent disorder of the development of movement and posture, resulting in a diminution of function. They are non-progressive, but can also be associated with disturbances of sensation, perception, cognition and communication, as well as behaviour.

Clinical Examination

The clinical picture may vary greatly from one individual patient to the other, depending on the amount and location of the initial brain insult.

Clinical examination is a critical part of the assessment. Its goal is fourfold:

- Evaluate spasticity.
- Evaluate possible muscle contracture and joint deformity
- Evaluate motor and sensory impairment in the upper limb.
- Evaluate existing function, and functional needs of the upper limb.

It is completed by a general examination in order to seek associated neurological disorders, and potential contraindications to surgery.

The data are recorded on standardised charts, which will allow intra- and inter-comparisons of the results of surgical treatment. This examination is lengthy, and requires detailed knowledge of neurology, paediatrics, and orthopaedics.

It is best performed as a team, including all the specialists involved in the child's care (physical therapist, occupational therapist, paediatrician and surgeon). This should ideally be done in a warm, quiet, and friendly environment, ensuring that the child is comfortable and confident. If painful procedures (i.e. injections) are necessary, they should be performed last. This is of paramount importance since the child's cooperation is essential for sensory and motor evaluation, and because spasticity may increase considerably if the child is frightened or recalcitrant.

Generally speaking, the clinical picture may vary greatly with the child's emotional state and fatigue level. Some of these children also have limited concentration capacities, and cannot cooperate throughout the entire examination. Therefore it is not wise to decide on surgery after a single session, and assessment should be repeated before any decision making.

Video recording of each clinical session is most helpful, both for initial evaluation, for decisionmaking, and for evaluation of surgical outcome.

Resting Posture of the Upper Limb

Inspecting the limb at rest prior to examination provides much information on spasticity. It usually predominates in the adductor, flexor and pronator muscles, leading to a typical resting posture in shoulder adduction and internal rotation, elbow flexion, forearm pronation and wrist flexion and ulnar deviation (Figs. 13.1 and 13.2).

The fingers may assume varied positions. Most frequently they are clenched into a tight fist, as a result of spasticity of the finger flexor muscles. Less typically they assume a swan-neck deformity, resulting either from excessive traction on the extensor tendons due to excessive wrist flexion (extrinsic swan-neck), from spasticity of the interossei muscles (intrinsic swanneck), or a combination of both.

The fingers may also occasionally assume an "intrinsic plus" deformity with flexion of the metaphalangeal (MP) joints and hyperextension of the interphalangeal (IP) joints, which is due to



Fig. 13.1 Usual deformity of the spastic upper limb involving adduction and internal rotation of the shoulder, and flexion of the elbow, wrist an fingers



Fig. 13.3 Spasticity of the interossei muscles: the "intrinsic-plus" deformity

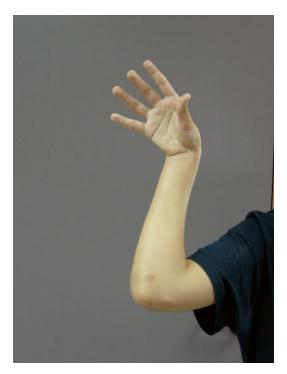


Fig. 13.2 Flexion and marked ulnar deviation of a spastic wrist

spasticity or contracture of the intrinsic muscles both (Fig. 13.3), or the opposite "intrinsic minus", or claw-type deformity, with the MP joints hyperextended and the proximal interphalangeal (PIP) joints flexed, due to a combination of excessive traction on the extensor tendons and paralysis of the intrinsic muscles. A boutonniere deformity is less common.



Fig. 13.4 Spastic adducted thumb

The thumb can assume either an adducted posture or an adducted and flexed posture. The adducted thumb is tightly clenched to the lateral aspect of the index or even the middle finger, with the MP and IP joint extended (Fig. 13.4). The 'flexus-adductus' thumb, often referred to as "thumb-in-palm", is embedded in the palm with full opposition and full flexion of both MP and IP joints. Often the clenched fingers are curled around the thumb.

Any factor that aggravates spasticity will increase these deformities.

Evaluation of Spasticity

Spasticity is a muscle hypertonia, characterised by five classic clinical features:

1. It is selective. Predominantly involving the flexor, adductor and pronator muscles and

responsible for the characteristic 'flexionpronation' deformity of the upper limb described above.

- 2. It is elastic. Attempts at reducing the deformity meet with a resistance, which increases with the strength applied. Unlike 'plastic' contractures, the limb returns to its initial position as soon as the attempt is stopped. However if the opposing force is maintained long enough, the deformity usually yields, sometimes abruptly.
- 3. It is present at rest, and exaggerated with voluntary movement, emotion, fatigue, and pain.
- 4. Osteotendinous reflexes are exaggerated, brisk, diffuse and polykinetic. Clonus is less frequent in the upper limb.
- 5. There may be an associated synkineses, described as 'the phenomenon whereby paralysed muscles incapable of a certain voluntary movement, execute this movement in a voluntary fashion by accompanying intact muscles' (Lhermitte sign). For example active shoulder abduction may be accompanied by synkineses of the fingers extensors and abductors (Souques synkinesis).

It is assessed for each muscle or muscle group and is infrequent around the shoulder. At the elbow, it usually involves predominantly the biceps and brachialis muscles, but also the brachioradialis to a lesser degree. The triceps, classically spared, can occasionally be spastic.

Wrist flexors and forearm pronators are most frequently involved, leading sometimes to an extremely hyperflexed (100°) and hyperpronated (150°) position.

Spasticity is not easy to assess in the fingers because of the wrist deformity. It usually involves the finger flexors, and to a various degree, the interossei muscles.

In the first web space and the thumb, spasticity involves not only the thumb adductor, but also frequently the first dorsal interosseous and the flexor pollicis brevis. Associated spasticity of the flexor pollicis longus leads to the classical "thumb in palm" deformity.

Spastic involvement is recorded on the standard chart for each muscle or muscle group. Its importance is assessed on a 0-5 scale [4] (Table 13.1).

 Table 13.1
 Modified Ashworth scale (from Bohannon and Smith D)

Grade	Description	
0	No increase in muscle tone.	
1	Slight increase in muscle tone, manifested by a catch and release, or by minimal resistance at the end of the range of motion (ROM) when the affected part(s) is moved in flexion or extension.	
2	Slight increase in muscle tone, manifested by a catch, followed by minimal resistance throughout the remainder (less than half) of the ROM.	
3	More marked increase in muscle tone through most of ROM, but affected part(s) easily moved.	
4	Considerable increase in muscle tone, passive movement difficult.	
5	Affected part(s) rigid in flexion or extension	

Muscle Contracture

Muscle contracture may involve spastic muscles. Unlike spasticity, it is permanent and cannot be overcome, although shortening the involved articular segment can alleviate it. For example posturing the wrist in flexion relieves contracture of the finger flexors. This is assessed by Volkmann's angle, which is the degree of wrist flexion required to obtain full passive finger extension. Contracture of the intrinsic muscles of the fingers is assessed by the Finochietto test (Fig. 13.5a, b).

Clinical distinction between contracture and spasticity may be difficult to establish. In such cases nerve blocks with lidocaine are useful [5]. The anaesthetic may be injected either in the nerve trunk or in the motor point of the involved muscle(s). Spasticity yields completely whereas contracture persists [6]. Botulinum toxin yields the same result, with a much longer lasting effect.

Joint Deformity

Passive motion of the involved joints may be difficult to assess, not so much because of spasticity but because of muscle contractures. It can only be tested with the involved muscles fully relaxed. Motor blocks are not very helpful here, as they do not alleviate muscle contracture. Sometimes assessment is so difficult that it is not until

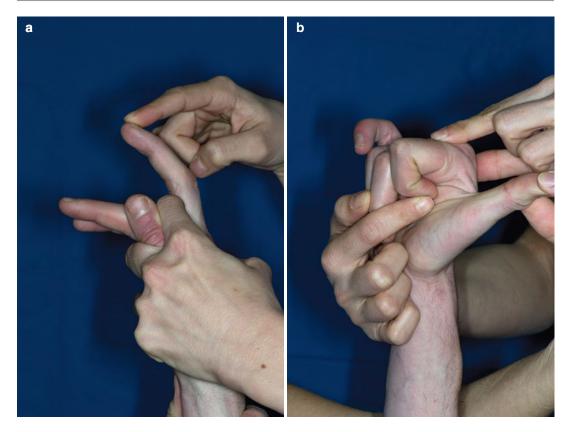


Fig. 13.5 Finochietto's test. (a) The PIP and DIP joints cannot be passively flexed when the MP joint is maintained in extension. (b) Flexion of the MP joint allows full passive flexion of the distal joints

immediate preoperative examination under anaesthesia that the actual range of passive motion can be evaluated.

Some joints of the fingers and thumb may have increased passive extension, resulting in joint instability. This occurs mainly at the thumb MP joint, and at the finger MP and PIP joints which can lead to a swan neck deformity (Fig. 13.6).

Motor Assessment

Motor examination of the upper limb is not easy in children, especially under 5 years of age. The child should be provided with toys of different forms and colours, and be observed at play. Video recording is extremely helpful at this stage, as it avoids lengthy repetitions of tasks. Rather than individual muscles, it is easier to evaluate muscle groups contributing to the same function.



Fig. 13.6 Spastic swan-neck deformity of the fingers

Paralysed Muscles

The palsy (or 'pseudo palsy') usually predominates in the distal part of the upper limb, and involves the extensor and supinator muscles, namely the wrist and finger extensors, abductor pollicis longus, extensor pollicis longus and brevis, and supinator muscle. Motor examination of these muscles may be difficult when the antagonist flexors and pronators are severely spastic.

Actually, rather than really paralysed, these muscles may be present, but made ineffective by the spastic antagonists.

Botulinum toxin has proved very helpful in assessing these muscles: when injected in the spastic antagonist muscles, it reduces dramatically their tone for several months, making it possible to evaluate and exercise the 'paralysed' muscles, which may end up demonstrating, in a number of cases, a satisfactory voluntary control.

This lack of control may also vary with limb position. For instance, voluntary movement of the thenar muscles is often facilitated by elbow extension.

Active Muscles

The flexor, adductor, and pronator muscles, mostly spastic, usually retain some voluntary control. However, their examination is made difficult when severe deformities are present. For example, extreme flexion deformity of the wrist prevents evaluation of the strength of the finger flexors, which are mechanically shortened. If the deformity is spastic, botulin toxin may again be helpful in these cases. Synkineses and cocontractures, when present, may also complicate the motor examination. We have not found electromyographic (EMG) studies to be very helpful in evaluating either the pseudo-paralytic or the spastic muscles (see below).

Finally a **general motor assessment** is performed, in order to evaluate the global motor control of the upper limb.

Spontaneous involuntary movements are recorded: they usually indicate athetosis, which is generally a contra-indication to surgery.

Dystonia is an unadapted muscular response to command in which intermittent muscle contractions cause repetitive movements and/or abnormal postures. Again it is also usually a contra-indication to surgery.

Standard tests are performed such as the "head-to-knee" test where the patient is asked to

place his hand on his head, then to move it to the controlateral knee. The speed and precision of the movement are recorded. These non-specific tests involve many of the elements susceptible to perturbation (hypertonia and muscle contracture, ataxia, apraxia, and extrapyramidal lesions).

Primitive reflexes are also sought. They are due to an abnormal sensory motor development, and may greatly impair the functional capacity of the limb. The classical "asymmetric neck reflex" is triggered when the head is turned actively or passively to one side, this produces abduction of the shoulder, and extension of the elbow, wrist and fingers of the ipsilateral upper limb while the controlateral limb all the joints flexed.

Once the motor examination has been completed, an attempt at classification can be made, using, for example, Zancolli's classification [7] where:

- Type I includes the spastic 'intrinsic-plus' hands, in which spasticity of the intrinsic muscles causes flexion of the MP joints and extension of the IP joints, sometimes associated with a swan-neck deformity. In this type a wrist flexion deformity is rare.
- Type II includes the spastic 'flexion-pronation' hands with (hyper) flexion of the wrist and pronation of the forearm. Within type II there are three sub-groups classified by the degree of active finger flexion.
 - In Group 1, there is full active extension of the fingers with the wrist in neutral or near neutral.
 - In Group 2 there is nearly complete active extension of the fingers, but with some degree of wrist flexion. This group is further subdivided based on the presence (subgroup A) or absence (subgroup B) of an active wrist extension.
 - In Group 3 there is no active finger extension, even with maximum wrist flexion.

Goldner [8] has produced another classification in group I, the wrist and MP joint can be extended at least to neutral. There is active grasp and release. The main deficiencies are delayed speed, slow coordination, and minimal dexterity; in group II, there is weakness of wrist and finger extension, with a mild contracture of the wrist, fingers, and thumb flexors. The thumb remains in the palm during hand extension. The hand is used only as an assist and a stabiliser; in group III, the wrist and finger flexors are severely contracted. The primary goal of surgery is to improve cosmesis; in group IV, the hands are both spastic and athetoid.

Tonkin [9] has described for the thumb deformities, a classification of three types, modified from House [10]:

- type I (intrinsic deformity) where spasticity of the medial thenar muscles (AP, FPB, and first DIO) associated with paresis of the thumb abductor and extensor muscles cause adduction of the first metacarpal, flexion of the MP joint and extension of the IP joint.
- type II (extrinsic deformity), where the dominant deforming force is the FPL, opposing a weak EPL. Metacarpal adduction is less marked, but there is hyperflexion of the IP joint.
- type III (combined deformity), where there is a combination of spasticity of the adductor muscles and of the FPL, with weakness of the abductor and extensor muscles. This results in the typical "thumb in palm" deformity, with adduction of the thumb metacarpal and flexion of the thumb MP and IP joints.

Aside from Tonkin's classification, we have not found any of the available classifications easy to utilise, since there is such a wide range of clinical pictures, depending on the amount and extent of the initial brain damage. There are no two identical cases and many of them do not fit accurately into any of the described categories. Moreover these classifications are not very helpful in the process of decision-making.

Sensory Examination

Sensory examination is practically impossible before the age of 4–5 (and two-point discrimination before age 6–7). Furthermore, besides the child's cooperation, it requires a certain level of intellectual capacities and language ability [11]. In cerebral palsy the basic sensory functions (light touch, pain, temperature) are essentially intact, while complex sensations (fine sensibility, proprioception, stereognosis) are more readily affected.

Light touch is explored using a smooth point or a finger, pain with a needle, and temperature with tubes of hot and cold water. Fine sensibility is explored with two-point discrimination.

Proprioception is tested by vibration (tuning fork) and by the sense of position of the limb: the patient is blindfolded, the unaffected limb is placed in one position, and he is asked to reproduce it with the affected limb. Proprioception is usually more disturbed in the distal part of the limb than in the proximal part.

Gnoses are the most affected. Stereognosis is tested by placing an object in the child's hand and asking him to identify it. Graphesthesia is tested by drawing figures or forms in the patient's palm.

On the whole, sensation is considered satisfactory when the child identifies at least three out of five objects, can recognise large figures drawn in the palm, and has a two-point discrimination of no greater than 5–10 mm [12].

It has been shown [13] that severely impaired sensation often goes along with upper limb discrepancy in hemiplegic CP children.

Pain may be present, but is difficult to evaluate, especially in children, who may not report it, and may not know how to describe it. It may be linked to severe contractures, or to a deformed joint, or, occasionally at the wrist to a Kienböck disease secondary to a severe flexion deformity [14].

Functional Assessment

A large variety of tools are utilised to assess the functional value of the spastic upper limb.

Whichever tools are used, the assessment should be video-recorded, as the videos can be viewed as many times as necessary, thus shortening the actual duration of the test.

The same tests will be repeated and recorded after surgery, and will then serve as a comparison for evaluation of the results of surgical procedures.

Functional Tests

A great diversity of functional tests are available. Some tests are analytic, assessing a single function, other are purely functional, assessing the use of the upper limb in ecologic situations (AHA).

• The pick-up and release test evaluates not only hand prehension, but also the contribution of the whole limb to that function. Objects of different sizes and volumes are placed in front of the child. Him/her are asked to pick them up, and then to move them to a different place. Computerised systems have been used more recently in an attempt to quantify hand grasp and release. They allow three dimensional analysis of the movement, and provide a repeatable protocol for objective evaluation of upper limb motor performance [15, 16].

- In the "box and block test", the patient is asked to move as many wooden blocks as possible from one compartment of a box to another in 1 min.
- Bimanual activities (such as carrying a container with two handles, cutting meat, holding one object into which another one should be placed, or holding a ruler while drawing a line with the unaffected hand) give accurate information on the child's actual functional ability.

Questionnaires

The child and family are also asked to describe precisely how the hand is used in activities of daily living such as dressing, self-care, and eating. Questionnaires are adapted to the child's age, and can be completed during the session, or at home (self-questionnaire).

In a number of cases the child neglects the upper extremity in spite of some potential functional capacity. In these cases, the child may persist in ignoring it even when functional ability can be improved through surgery.

There are several validated non-specific questionnaires evaluating hand function. To the best of our knowledge, currently there is no validated questionnaire adapted to the cerebral palsied child.

Grading Scales

Many scales have been designed to quantify the functional value of the upper limb, although few are specifically designed for the spastic upper limb [8, 11]. Among them :

- Hoffer [12] tests dressing, personal hygiene, feeding, bimanual activities, grasp and release, as well as the lateral pinch.
- House provides an eight grade classification, based both on the grasping capacities, and on

the contribution of the hand to bimanual activities.

- The Shuue [17] scoring system mixes analytic measurements, and functional measurements (grasp and release, spontaneous functional analysis, and dynamic positional analysis), adding up to a numerical scoring.
- The MACS score is designed for quadriplegic patients

General Preoperative Assessment

The aim of this general examination is to evaluate the real benefit the child could gain from surgery, taking into account other neurological impairments, the patient's age, intelligence, motivation and environment.

Other Neurological Impairments

As these children are usually hemiplegic, the lower limb deficit must also be assessed, and it is especially important to evaluate the child's walking ability, and the possible need for walking aids (wheelchair, crutch). If operations are necessary for improvement of the lower limbs, they are usually undertaken before any upper extremity surgery.

Associated extrapyramidal signs should also be detected. These include the following:

- Athethosis, which is characterised by unexpected, non-voluntary movements causing a slow oscillation of the limbs. It is reduced at rest, abolished at sleep, and increased by noise, fatigue, and emotions.
- Chorea is made of brisk rapid and anarchic non-voluntary movements, of variable amplitude, which can involve all territories. In the upper limb, these contortions of the forearm, hand, and fingers often make activities of daily living impossible.
- Parkinson syndrome is characterised by the classic triad: resting tremor, plastic hypertonia (predominant in the proximal muscles) with the cogwheel sign, and akinesia.

If these extrapyramidal signs are predominant, they preclude surgery, as the child is unable to use his hand because of these non voluntary movements.

The capacity of the child to communicate must be evaluated, seeking for visual, hearing

and language problems. Behavioural problems such as irritability, inability to concentrate, and emotional instability may also constitute contraindications to surgical treatment if they predominate.

Intelligence is evaluated through the intelligence quotient (IQ). It is usually stated that functional surgery is not indicated when the IQ is lower than 70, although this is not an absolute rule, as a number of surgical procedures aimed at improving comfort, cosmesis and personal hygiene are still indicated [18].

Age

Because the neurological deficit in cerebral palsy does not evolve, early surgery can be undertaken. Sometimes it is necessary to operate very early because of an increasing deformity. However, in most cases one prefers to wait until the child is old enough that their motor and sensory capacities can be evaluated accurately and he or she can cooperate with surgery and more importantly postoperative rehabilitation.

In the adult, the surgeon should be more cautious when recommending surgical intervention, as many of these patients have often adapted functionally and socially to the handicap and surgery may be a bit more detrimental than beneficial.

Motivation and Environment

Any assessment of motivation should take into account the patient's ability to understand the goal, the modalities and the expected benefit of the proposed treatment, and to participate actively in the postoperative regimen. Understanding and motivation from the child's parents are also mandatory. Environmental factors during the surgical period are also important, such as a rehabilitation centre with an integrated school system and access to physiotherapists experienced in treating children with cerebral palsy.

According to Tonkin [19], "the ideal candidate is a cooperative 6-year-old child, with stable family support, who has a predominantly spastic upper limb deformity, with satisfactory hand sensibility, hemiplegic or monoplegic and without significant neurological deficits".

X Rays and Electromyography

X-rays are part of the preoperative evaluation. They are aimed at assessing any growth disturbance and joint deformity linked to the spasticity, although satisfactory views may not always be obtained when there is a severe deformity such as wrist hyperflexion. Contralateral views in the same position may be helpful.

They may reveal growth disturbances of the distal radius, ulna, carpus and occasionally avascularity of the lunate [14], or dislocation of the radial head [20].

EMG studies provide information on the spastic muscles (voluntary control, phasic control), as well as on possible co-contractures of the antagonist muscles. In pseudo-paralytic muscles, they may identify voluntary control that is not clinically detectable (because of spastic and/or retracted antagonists, joint deformity and/or stiffness). However, they do not provide quantitative information on the power of the tested muscle.

Dynamic EMG studies, although difficult to perform in young children, may be particularly useful in determining the most appropriate donor muscles when planning a tendon transfer. Most of the potential donors are spastic to some degree. They can be utilised only if they are capable of relaxation at rest or during the antagonist movement (phasic control). A muscle that fires continuously is not a good candidate for use as a transfer [21, 22].

Clinical Pearl

- Surgical intervention should only be undertaken after a thorough and if necessary sequential evaluation by all clinicians, including the surgeons, occupational and physiotherapists, neurologists, etc. The use of video recording is often helpful.
- The use of dynamic EMG's and particularly botox as a diagnostic tool can be extremely useful.
- The ideal candidate is a cooperative 6 year old child, who has a predominantly spastic upper limb deformity, with satisfactory hand sensibility and with little other significant neurological deficit.

Stroke

In adult hemiplegia related to a vascular stroke, the clinical picture is quite different.

Patients are usually relatively old. Spasticity occurs after a few weeks and follows a flaccid phase. There are usually few active muscles, and the wrist is often paralytic, both in flexion and in extension. Sensation is often severely impaired in the hand, involving mainly deep sensation and stereognosis. It can persist despite dramatic motor improvement, thus improving functional recovery. Trophic changes, such as reflex sympathetic dystrophy and vaso-motor changes, are frequently associated. These features, are, usually a contraindication to surgery. However, procedures aiming at reducing spastic contractures, and improving hygiene and nursing may be helpful in some cases.

Head Injury

The initial trauma may have involved various portions of the brain and cerebral trunk and the clinical features will vary accordingly. Thus the clinical picture is extremely varied in head injury patients. Motor impairment depends upon the extent of brain damage. It may regress rapidly in some patients, or remain in others.

Other neurologic disorders are often predominant, e.g., cerebellar syndrome or frontal impairment. Many of which contra-indicate surgical attempts at improving function to the upper limb.

Tetraplegia

Spasticity in tetraplegic patients usually occurs in the lower limbs. According to Zancolli [7], it affects the upper limbs in 15 % of patients only, mainly those with an incomplete tetraplegia. In such patients, it involves mostly the wrist and finger flexors. It can be extremely useful to the patient, who by triggering the stretch reflex can initiate a pinch or a grasp [23]

Spasticity, when moderate, does not interfere with surgical rehabilitation of the tetraplegic

upper limb. When it is significant, it causes deformities which must be corrected prior surgery. When it is predominant, tendon transfers may be impossible [24].

Treatment

Surgery has a limited place in the treatment of spasticity of the upper limb. It is only one element of the rehabilitative care, which consists primarily of physiotherapy and splinting, occupational therapy, and pharmacological treatment as needed.

Any decision-making should include the patient and his/her family, as well as all the physicians and care-givers involved in the treatment, typically after several assessment sessions and video-recording of the patient's functional achievements.

As mentioned earlier, surgery seems more effective if performed earlier in the patient's life, preferably during childhood [19]. Later on, the patient can develop 'actions or tricks' allowing them to undertake various activities. Any surgery whilst undertaken with the best intention can result in deterioration in function. Another advantage of performing surgery in children is that there is some evidence that improved use of an extremity can improve cortical representation of the extremity and hopefully decrease the development of neglect. Beach et al. evaluated 40 patients with CP who underwent a tendon transfer to improve wrist extension (FCU to ECRB) with an average follow-up of 5.2 years, and found that the best results were seen in patients who had the transfer between the ages of 7 and 12 years [25]. In addition, early surgery may decrease the formation of contractures.

Rebalancing the Forces

The goals of surgical treatment can vary greatly, depending on the extent of the initial cerebral trauma. Whenever possible, it is to improve function. In some cases, however, it will be limited to improving nursing and comfort, or to correct a severe deformity.

In any case, the surgery will have to address all the deforming causes, in order to rebalance the forces exerted around the involved joint. Three types of procedures may thus be indicated, in isolation or together:

- 1. Those which aim at reducing spasticity
- Those which aim at reducing muscle and/or joint contracture
- 3. Those which aim at reinforcing paralysed muscles

Reducing Spasticity

Local Pharmacological Agents

Besides systemic medications such as Baclofen, which are used in severe and generalised spasticity, some agents are effective locally.

Before the era of botulinum toxin, nerve blocks were often used.

Lidocaine blocks have a temporary effect; they are mostly used as a diagnostic tool in difficult cases to differentiate between spasticity and contracture [6].

Alcohol blocks have also been widely used in the past [26, 27]. Injected either into the nerve trunk or the motor point of the involved muscle(s), they would produce a reduction of spasticity lasting up to several months and even longer if the antagonist muscles were active [5].

Lastly, phenol, which is more effective, may be toxic for the surrounding tissues, and must be applied surgically within the epineurium [28].

Botulinum toxin A is a neurotoxin produced by the bacterium Clostridium botulinum. When injected into a muscle, it blocks the release of acetylcholine at the neuro-muscular junction, resulting in the denervation of the involved muscle. This denervation is dose-dependant, and reversible. Its effect starts 10–15 days after the injection, is maximal at 2–3 months, and usually lasts for 4–6 months.

Used initially for blepharospasm and strabismus, it is now routinely used in spastic limbs [29, 30] with measurable and reproducible effects in cerebral palsied children [31, 32]. Charts are available which indicate the effective dose for each age group and for each individual muscle [33].

While yielding the same result as the previously mentioned agents, it is much easier to use, because it is injected into the muscle body instead of the motor point, which often proves difficult to locate. Canulated stimulation needles or ultra-sound localisation have rendered the injection even easier and more effective.

Indications

Besides its diagnostic use mentioned earlier, botulinum toxin (BT) may be used in isolation to reduce spasticity of a muscle or a group of muscles (usually wrist flexors and/or extrinsic finger flexors). During its temporary effect the antagonist muscles should be exercised. The stronger they get, the longer lasting the effect of BT will be. Muscle contractures and stiffness of the involved joint(s) may also be efficiently reduced during that period, enhanced by passive and/or dynamic splints. Cosmesis, and pain if present, are also improved.

BT may be repeated as required, possibly yielding a permanent improvement if the antagonist muscles improve their strength accordingly, thus balancing the spasticity more effectively. For instance, a patient with moderate spasticity of the wrist flexors associated with active extensors (even if weak prior to therapy) is a good candidate for injections of BT. If spasticity recurs after each injection, a more definitive procedure can subsequently be performed. BT also plays, in such cases, an educational role in simulating the effect of surgery. This is usually very much appreciated by the patient, who understands exactly what to expect from the surgical procedure.

BT is also indicated in spastic upper limbs secondary to head injury, where spasticity may be temporary. It is particularly useful in spasticity of the elbow flexors which may be severe, resistant to other types of conservative treatment, and yield severe and permanent muscle and joint flexion contractures of the elbow if left untreated. In such cases, BT should be used early, in association with rehabilitation and splinting as needed, and repeated until spasticity decreases.

Finally BT may be used pre- or immediately postoperatively to attenuate the spastic muscles when performing a tendon transfer to the antagonists, in order to protect and facilitate education of the transferred muscle.

Tenotomy

Tenotomy of a spastic muscle will obviously permanently relieve spasticity. However, it will also permanently suppress muscle function. As a consequence, one must be certain that the muscle to be tenotomised would not be better employed as a transfer in order to rebalance the deforming forces.

In the upper limb, it is mostly used for the wrist flexors (FCU), and the pronator teres (PT). For the FCU, tenectomy with dissection of the fascial connections of the muscle is advocated by de Bruin et al. [34], who have demonstrated that this is much more effective than simple tenotomy in reducing the wrist flexion torque.

Selective Neurectomy

("Hyponeurotisation")

Selective neurectomy, like tenotomy, suppresses both spasticity and function.

It may be indicated in non functional upper limbs with severe spasticity, in order to facilitate hygiene and nursing, and to improve cosmesis. A specific indication is spasticity of the intrinsic muscles where a neurotomy of the motor branch of the ulnar nerve will improve cosmesis and self care, and sometimes function.

Selective neurectomy involving only part of the nerve fascicles, in an attempt to retain some function, had been suggested as early as in 1913 by Stoffel [35] This technique has gained some popularity after Brunelli and Brunelli [36] published a series of clinical cases in 1983, coining the term "hyponeurotisation". The procedure is performed at the entry point of the nerve into the muscle, where it usually divides into three or four fine fascicles. Under magnification loops, part of the fascicles are resected (Fig. 13.7).

Brunelli initially advocated removing 50 % of the fascicles, but because he experienced some recurrence of the spasticity ("adoption" phenomenon), he then recommended resection of two thirds of the fibres.

In order to simplify the procedure, some have chosen to perform a "partial" neurectomy at the level of the nerve trunk, without approaching the target muscle(s). The motor fascicles are identified using a stimulator and partially resected.

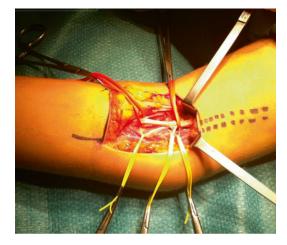


Fig. 13.7 Selective neurectomy of the motor branches of the median nerve to the wrist flexors and pronator teres: each motor branch is dissected down to its entry into the muscle, then partially resected

While faster and more limited in exposure, this technique is quantitatively less accurate, with potential injury to sensory fibers.

Indications

Selective neurectomy is indicated when one wishes to reduce spasticity permanently. Resection of the motor fibres must include at least 2/3 of the fascicles, knowing that it will reduce the strength of the muscle in the same proportion as spasticity.

In our hands, this technique has proved more effective on large muscles with a single or mostly predominant motor pedicle. It has been less satisfactory in small intrinsic muscles such as the thumb adductor and first dorsal interosseous muscles.

Hyponeurotisation and partial neurotomy have no effect on muscle or joint contractures; if either are present, these must be addressed by another procedure.

Neurosurgical Procedures

Treatment of spasticity by posterior rhizotomy was initially recorded in the late nineteenth century literature. It is now frequently used for spasticity of the lower limbs. In the upper limb, however, results have been very variable, with a number of respiratory complications [37]. Bertelli et al. [38] has recently reported a significant reduction in upper limb spasticity with a new technique of brachial plexus dorsal rhizotomy in 61 children or adolescents with spastic hemiplegia.

Muscle Contracture

Several types of procedures can be employed to overcome muscle contracture.

Tenotomy

Besides FCU and PT (see above), this technique may be useful in severe contractures occurring in a non-functional upper limb, particularly for hygiene or nursing purposes (fingers permanently flexed in a tight fist, for example).

Muscle Release

The classical flexor-pronator release procedure, described by Page in 1923 [39], consists of releasing the proximal insertion of the wrist flexors and pronator teres muscle from their medial epicondylar origin. The muscles are allowed to slide 4–5 cm distally. It has also been recommended that reinserting them distally to the ulnar periosteum will avoid a secondary supination deformity [40].

This procedure can be extended to the finger flexor muscles, as described by Scaglietti. It is then referred to as the "Scaglietti-Page" procedure. The skin incision is extended distally, and all the finger flexor insertions are freed from the anterior aspect of the ulna and radius. Care must be taken to protect the anterior interosseous artery during the procedure. This procedure necessitates a wide dissection of the anterior forearm compartment, which makes careful haemostasis and postoperative suction drainage mandatory.

Finally, Zancolli and Zancolli [41] has described an ingenious and more limited release of the medial epicondylar muscles consisting in a transverse resection of the inter- and peri-muscular fascia of all the involved muscles, performed 6 cm distal to the medial epicondyle ("flexor aponeurotic release") (Fig. 13.8).

Tendon Lengthening

Goldner [8] advocated performing a Z lengthening of each individual tendon. This procedure, simple when there is only one or a few tendons involved, becomes more complicated and time



Fig. 13.8 Zancolli's flexor aponeurotic release: a transverse band of the muscular fascia is resected

consuming if it is to include all the flexors of the wrist and fingers.

Fractional Lengthening

When multiple lengthening are required, this is preferably performed at the muscle-tendon junction. This technique consists in performing multiple transverse incisions of the tendon in the area where muscle and tendon overlap (Fig. 13.8). Careful passive extension then allows the muscle fibers to lengthen, while retaining muscle-tendon continuity.

Post-operatively, no immobilisation is required, and early active motion is initiated.

STP

For severe finger flexor contractures, Braun et al. [42] has described an ingenious procedure of transfer of the flexor digitorum superficialis to the profundi (STP). It consists in sectioning all the finger superficialis (FDS) tendons distally at the wrist, then sectioning all the flexor profundi (FDP) 5–7 cm more proximally, extending the fingers, and suturing as a whole the proximal stump of the FDS to the distal stump of the FDP. This predictable procedure reduces finger flexors contracture and pain, while improving hygiene [43], but suppresses independent finger flexion.

Bone Shortening

Shortening the skeleton of both bones of the forearm does reduce muscle contracture, and has been advocated in non-cooperative adults.

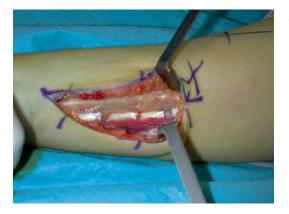


Fig. 13.9 Fractional lengthening of the flexor carpi ulnaris muscle

Omer and Capen [44] performed proximal row carpectomy together with muscle transfers. This procedure reduces slightly muscle contracture, while improving wrist mobility in cases of joint contracture. It may also be indicated in those spastic patients who develop a symptomatic Kienbock's disease [14].

Indications of Muscle Release Procedures

Mild contracture of the wrist flexor and pronator muscles will be relieved by Zancolli's flexor aponeurotic release. This procedure has been reported as less effective in adults, where there is a component of myostatic contracture, than in children [45].

Muscle contracture involving only one tendon, or a limited number of tendons, may be treated by Z lengthening of the individual tendons. Multiple contractures of the wrist and finger flexors are best treated by fractional lengthening. This procedure has progressively replaced the classical Scaglietti-Page release, which is much more invasive, with potentially more severe complications (Fig. 13.9).

In non or poorly functioning hands where contracture interferes with whatever function remains and/or with hygiene and nursing care, the simple STP procedure is best indicated.

Indications for bone shortening are uncommon.

None of these procedures are effective in the presence of joint contracture. If, after all muscle contractures have been eliminated, there is still a limitation of passive motion, a joint procedure must be added.

Joint Contracture

Conventional arthrolysis procedures may be required to treat the spastic upper limb, taking into account that there is often combined muscle and joint contractures, both of which need to be addressed, and that the contracture is likely to recur if either the spasticity is not relieved or if the antagonist muscles are not active (or activated).

A fixed pronation deformity may require a release of the interosseous membrane. Severe longstanding deformities in adults may only respond to osteotomy of the forearm, or arthrodesis in a more favourable position.

Tendon Transfers

Tendon transfers are required when the paretic or paralysed muscles require augmentation. They are usually performed to improve forearm supination, wrist extension, thumb extensionabduction and finger extension.

They differ from classical tendon transfers in several ways:

- Muscles available for transfer vary with each patient. They are often difficult to select because spasticity, co-contracture, muscle weakness and lack of coordination may render individual muscle assessment extremely difficult. Careful and repeated muscle evaluation, EMG study and botulinum toxin may all be helpful with this decision.
- The transferred muscle is often spastic to some degree. It has been stressed that a spastic muscle that does not have a phasic control should not be used as a transfer [21, 22]. Dynamic EMG studies are very helpful in selecting adequate muscles.
- Tendon transfers will be successful in activating a paralysed muscle, only if the spastic and/or contracted antagonist muscles are attenuated and/or released (prior to or) at the time of tendon transfer.

Most Frequent Procedures

The extreme diversity of clinical pictures makes it impossible to describe all the surgical combinations of procedures performed in the spastic upper limb. Any surgical planning adapts to each specific situation, bearing in mind that all deforming elements should be treated, and preferably at the same time in order to rebalance local forces, for an optimal result.

We will describe here our most frequent procedures.

At the Shoulder Level

Shoulder retraction in adduction and internal rotation, which impairs function of the hand should be treated. If necessary a prior injection of botulinum toxin will differentiate muscular contracture from spasticity. Muscle contracture is released by releasing subscapularis and lengthening pectoralis major (and minor if required). Postoperative physiotherapy is initiated immediately, and continued for 8 weeks at least.

At the Elbow Level

The deforming forces at this level are spasticity and/or contracture, whereas paralysis is rare.

In head injury patients, spasticity of the biceps may be isolated and temporary. In these patients, severe muscle hypertonia may lead to early and irretractable flexion of the elbow [46]. If conservative therapy fails to improve it rapidly, botulinum toxin is indicated, followed by intensive physiotherapy combining active and passive extension of the elbow. If spasticity recurs, selective neurectomy ("hyponeurotisation") of the musculo-cutaneous nerve is indicated. It is particularly effective in this large muscle which has a single and easily accessible motor branch [47].

In a number of cases, however, the other elbow flexors may also be spastic. Each spastic muscle must be treated, whether either botulinum or hyponeurotisation is used.

In CP patients the flexion deformity may be complex, combining spasticity and muscle (and joint) contracture of the elbow flexors, and not infrequently spasticity of the triceps muscle.

Botulinum toxin is a very effective diagnostic tool at this level: it allows assessment of the degree of spasticity of each individual elbow muscle. EMG studies by Keenan et al. [48] have shown that even brachioradialis may be severely affected.

Spasticity leads to impaired elbow flexion not only during voluntary movements, but also while standing, walking or running. It is also frequently perceived as cosmetically unacceptable by children and their families. If targeted botulinum toxin is effective, selective neurectomy usually leads to a satisfactory permanent result.

If the contracture is mild (less than 40° of extension lag), it does not usually require surgical correction. More severe cases are treated by lengthening of each contracted muscle:

- the biceps may be lengthened by multiple transverse myo-fasciotomies at the muscletendon junction if the contracture is mild [49]. If it is severe, a long Z plasty of the biceps tendon is more effective, requiring postoperative immobilisation for 4–6 weeks.
- the brachialis is lengthened by multiple transverse fasciotomies, after dissection and protection of the vascular bundle [50].
- the brachioradialis may require release from its proximal insertion, after dissection and protection of the radial nerve and its motor branches.
- release of the flexor-pronator muscles, which are accessory elbow flexors, may also improve elbow extension.

Contracture of the elbow joint can be demonstrated only after muscle contractures have been released. If it is severe it requires a conventional anterior arthrolysis. Results of this surgery, however, may be disappointing in the long term, especially if the triceps muscle is weak, or has a poor voluntary control.

Tendon transfers are very seldom indicated at the elbow, and only for the triceps muscle. The choice of donor muscles is large, as many proximal muscles are usually active.

In non-functioning limbs with a severe elbow deformity, a simple procedure, such as a neurectomy of the musculo-cutaneous nerve [51], or a biceps tenotomy, extended as required to the other elbow flexors, usually improves elbow extension immediately by 40° [46]. Successive postoperative plaster casts usually further improve the result.

At the Forearm Level

A symptomatic pronation deformity can be improved surgically. Available procedures include:

- selective neurectomy or tenotomy of the pronator teres
- lengthening of the pronator teres if the muscle is contracted, with release of the interosseous membrane when required
- tendon transfer, usually by pronator rerouting [52, 53] if the supinators are paretic or paralysed
- rotational osteotomy of the forearm bones if the deformity is fixed.

Gschwind and Tonkin [54] have established a classification of pronation deformities and a proposed surgical plan for each group:

- Group I, where there is active supination beyond neutral, does not require surgery
- Group II, where active supination is limited to neutral or less, may be treated by pronator quadratus release, associated with a flexorpronator release if the flexor-pronator group of muscles is contracted. The quality of the result in this group will depend on the ultimate strength of active supination
- Group III patients display no active supination, but have full passive supination. This group is treated by muscle transfer of either the pronator teres (rerouting), or the FCU
- In group IV, there is no active supination, with a fixed pronation deformity. A release of the spastic pronator and pronatory-effect muscles is indicated to allow possible active supination to be unmasked. If this does not occur, a pronator teres transfer is indicated [45]

At the Wrist Level

The wrist is the most frequent site of surgical treatment in the spastic upper limb. Here again, the deformity occurs as a result of a combination of spasticity and muscle contracture of the wrist and finger flexors, and muscle imbalance due to paresis or paralysis of the antagonist wrist extensors.

If there is no muscle contracture (i.e. full passive extension of the wrist and hand), botulinum toxin is injected in the spastic muscles, followed by a regimen of strengthening of the extensor muscles. This protocol may be sufficient in mild spasticity, provided rehabilitation and splinting are pursued for many months. If the extensor muscles do not respond to strengthening, then selective neurectomy of the spastic muscle together with tendon transfer to the paralysed extensors (usually to the ECR muscles) is an option. Relieving spasticity in the wrist flexors may unmask spasticity of the finger flexors, which will then tend to perpetuate the wrist flexion deformity. They must be treated accordingly.

Contracture in the flexor-pronator muscles group should be treated as described earlier. It has been our experience that this release usually attenuates spasticity of the involved muscles and as a consequence there is usually no need for any complementary procedures aimed at reducing spasticity.

Paralysis or paresis of the wrist extensors is treated by tendon transfers, usually involving the ECR muscles. The FCU ("Green transfer") is the most appropriate muscle when it demonstrates adequate relaxation at rest (phasic control), provided the FCR is active. Other motors may include brachioradialis [55], pronator teres or a finger flexor superficialis.

The ECU is often paretic, although when the flexion deformity of the wrist is severe, it may sublux volarly, and become a wrist flexor. Rerouting it dorsally and radially will help rebalance the wrist, by decreasing ulnar deviation, and enhancing wrist extension.

In cases of a non- or poorly functioning hand with a severe wrist flexion deformity, fusing the wrist may be a reasonable option for improving cosmesis and nursing care [56, 57]. The procedure involves bone shortening, usually through a proximal row carpectomy, or a dorsal wedge osteotomy, and wrist and finger flexors lengthening as required, in order to avoid a permanently clenched fist. Van Heest and Strothman [58] recommended to use plate and screws, although Hoffer and Zeitzew [59] obtained the same rate of fusion with K wires.

Mid-carpal fusion is an interesting option when the deformity is not too severe and one wishes to retain some mobility in the wrist joint.

At the Fingers Level

Flexion contracture of the fingers should be treated in conjunction with the wrist deformity.

Isolated spasticity of the finger flexors theoretically responds to either botulinum toxin or selective neurectomy. However the results of these two procedures are less predictable in the fingers than in the wrist, given the number of muscles and motor branches involved. Muscle contracture responds to fractional lengthening, which has supplanted the muscle slide procedure (Scaglietti). By means of its weakening effect, it is also indirectly effective on spasticity of the finger flexors, and we now tend to use it more frequently in this indication. STP is effective in poorly or non-functioning hands for cosmetic or nursing purposes. Carlson's experience [60] is that fractional lengthening of the digital flexors will be sufficient if the fingers can be extended fully with the wrist in the flexed position. Otherwise, if the fingers cannot be extended fully, then a superficialis to profundus transfer is indicated. Release of the finger flexion deformity may unmask an intrinsic spasticity, which will require additional treatment.

Tendon transfers to augment active finger extension are not frequently indicated, although there is no consensus in the literature [61, 62]. It was formerly recommended [63] not to perform them until a minimum 6 months after the release procedures at the wrist and fingers, in order to allow spontaneous recovery of the tone of the stretched extensors. The generalised use of botulinum toxin has rendered this precaution unnecessary in most cases.

Swan Neck Deformity

As swan-neck deformity may be secondary to wrist hyperflexion, the latter must be corrected first. If the swan–neck persists and interferes with function, it can be corrected surgically.

If the swan neck is due to muscle imbalance, a combination of intrinsic muscle spasticity and over activity of the finger extensors, this may be corrected by one of the following procedures: tenodesis of the flexor superficialis [64], tenotomy of the central band of the extensor tendon [63], lateral band tenodesis as performed by Littler and Cooley [65] or by Zancolli, spiral oblique ligament reconstruction [66].

In spastic patients, one must be extremely careful not to overcorrect the deformity, as this may lead to a flexion contracture.

Intrinsic Contracture

Intrinsic contracture may be isolated, or associated with a swan-neck deformity.

If the deformity is mild, the contracture may be released by resection of the triangular laminae [7].

- If the swan-neck deformity is severe, IP joint hyperextension must be treated as described above at the same time as the intrinsic release.
- Severe spasticity of the intrinsic muscles can be addressed by neurectomy of the motor branch of the ulnar nerve in Guyon's canal [67].

At the Thumb Level

Available procedures aimed at rebalancing the thumb, by means of reducing spasticity, include releasing the contracted muscles, stabilising thumb joints, and augmenting paretic or paralytic extensor muscles by tendon transfers.

Reduction of Spasticity and Muscle Contracture

As mentioned earlier, nerve procedures are not reliable enough when addressing intrinsic muscle spasticity.

An isolated contracture of the adductor muscle may be corrected by distal tenotomy at the sesamoid level, associated with a stabilisation of the MP joint to avoid the development of an hyperextension deformity. Most frequently, however, there is a combined contracture involving other thenar muscles (APB, FPB), the first dorsal interosseous together with the adductor muscle. In such cases, Matev [68] described an extended palmar release including adductor pollicis, flexor pollicis brevis, and the distal two-thirds of abductor pollicis brevis. The first dorsal interosseous muscle may also be released as required through the same incision. This procedure is technically demanding as one must release these muscles completely while protecting their motor branches [69].

When the flexor pollicis longus is contracted, it can be released by a fractional lengthening in the forearm.

Muscle contracture, especially in cerebral palsy, may be associated with a skin contracture of the first web space. This is treated by a Z plasty.

Joint Stabilisation

MCP joint hyperextension deformities can be treated by either a volar tenodesis or a capsulodesis, although both of these have a tendency to slacken with time. As a consequence, a sesamoidmetacarpal fusion, as described by Zancolli, or a simple MCP joint fusion may be more appropriate, particularly in severe cases.

Tendons Transfers

Tendons transfers are necessary when thumb extension-abduction is paralysed or weak. Suggested motors include the extensor carpi radialis if the wrist extensors are spared [68], brachioradialis [64, 41], flexor carpi radialis [70] or a flexor superficialis [71].

Inglis suggested rerouting the APL through the APB in order to increase thumb abduction.

Rerouting the EPL so as to change its adduction component into one of abduction, was performed by Manske through the first dorsal compartment, whereas Carlson used a pulley harvested from the most volar slip of the APL

Tonkin et al. [9] advocated a specific treatment for each group (see above for classification):

- type I (intrinsic deformity): treated using a combination of adductor/FPB release, and first dorsal interosseous release if necessary, rerouting EPL to EPB, BR transfer to APL, and stabilisation of the MP joint by either capsulodesis, sesamoido-metacarpal, or MP fusion as required.
- type II (extrinsic deformity): treated by a FPL tendon slide, together with a release of the other contractures as required.
- type III (combined deformity): treatment includes the same procedures as in Type I, associated with a FLP tendon slide, and IP joint fusion in recalcitrant flexion deformities.

Clinical Pearl

- The fundamental of any surgical treatment for spasticity is in the first instance to reduce the spasticity, then address either muscle or joint contracture, followed by rehabilitation and strengthening, particularly of the antagonist muscles.
- The fundamental of any surgical treatment for spasticity is to reduce the spasticity, and address at the same time all deforming forces, including muscle or joint contracture and muscle weakness, followed by rehabilitation and strengthening, particularly of the antagonist muscles.

Conclusion

Not many spastic patients are candidates for surgery of their upper limb, because of the many other neurological problems frequently associated.

Surgery should be decided upon only after several examinations of the upper limb. One should also take into account the other neurological impairments, the patient's functional achievements, and his (or his parents') wishes.

Surgery of the spastic upper limb is complex, involving reduction of spasticity, release of contracted muscles and joints, and augmentation of weak or paralysed muscles, together with the stabilisation of unstable joints. It is best performed early in CP children. Finally, there is no such thing as "standard procedures" in this group of patients, because each case is different.

References

- Rosenbaum P, Paneth N, Leviton A. A report: the definition and classification of cerebral palsy. Dev Med Child Neurol Suppl. 2007;109:8.
- 2. Little WJ. Course of lectures on the deformities of the human frame. Lancet. 1843;41:350–4.
- Lawson RD, Badawi N. Etiology of cerebral palsy. Hand Clin. 2003;19:547–56.
- Bohannon RW, Smith MB. Interrater reliability of a modified Ashworth scale of muscle spasticity. Phys Ther. 1987;67:206–7.
- 5. Roper B. Evaluation of spasticity. Hand. 1975;7:11-4.
- Braun RM, Mooney V, Nickel VL. Flexor-origin release for pronation-flexion deformity of the forearm and hand in the stroke patient. J Bone Joint Surg. 1970;52A:907.
- Zancolli E. Surgery of the hand in infantile spastic hemiplegia. In: Zancolli EA, editor. Structural and dynamic bases of hand surgery. 2nd ed. Philadelphia: JB Lippincott; 1979. p. 263–83.
- Goldner JL. The upper extremity in cerebral palsy. Orthop Clin North Am. 1974;5:389–414.
- Tonkin MA, Hatrick NC, Eckersley JRT, Couzens G. Surgery for cerebral palsy: part 3: classification and operative procedures for thumb deformities. J Hand Surg Br. 2003;26B:465–70.
- House JH, Swathmey FW, Fidler MO. A dynamic approach to the thumb-in-palm deformity in cerebral palsy. J Bone Joint Surg Am. 1981;63A:216.
- Tardieu G. Le dossier clinique de l'IMC: méthodes d'évaluation et applications thérapeutiques. 3rd ed. Paris: Massòn; 1984.

- Hoffer MM. The upper extremity in cerebral palsy. AAOS Instruct Course Lecture: 133. 1979a.
- Van Heest AE, House J, Putnam M. Sensibility deficiencies in the hands of children with spastic hemiplegia. J Hand Surg Am. 1993;18A:278–81.
- Leclercq C, Xarchas C. Kienböck's disease in cerebral palsy. J Hand Surg Br. 1998;23(B):746–8.
- Fitoussi F, Diop A, Maurel N, el Laassel M, Pennecot G. Kinematic analysis of the upper limb: a useful tool in children with cerebral palsy. J Pediatr Orthop B. 2006;15:247–56.
- Butler EE, Ladd AL, Louie SA, LaMont LE, Wong W, Rose J. Three-dimensional kinematics of the upper limb during a Reach & Grasp Cycle for children. Gait Posture. 2010;32:72–7.
- Davids JR, Peace LC, Wagner LV, Gidewall MA, Blackhurst DW, Roberson WM. Validation of the Shriners Hospital for Children Upper Extremity Evaluation (SHUEE) for children with hemiplegic cerebral palsy. J Bone Joint Surg Am. 2006;88A(2): 326–33.
- Mital MA, Sakellarides HT. Surgery of the upper extremity in the retarded individual with spastic cerebral palsy. Orthop Clin North Am. 1981;12:127.
- Tonkin MA. The upper limb in cerebral palsy. Curr Orthop. 1995;9:149–55.
- Sneineh AKA, Gabos PG, Miller FMD. Radial head dislocation in children with cerebral palsy. J Pediatr Orthop. 2003;23:155–8.
- Hoffer MM. Dynamic electromyography and decision-making for surgery in the upper extremity of patients with cerebral palsy. J Hand Surg Am. 1979; 4:424.
- Van Heest AE. Functional assessment aided by motion laboratory studies. Hand Clin. 2003;19:565–71.
- Maury M, Audic B, Guillaumat M, Francois N. L'évolution du traitement de la spasticité dans les lésions médullaires. VIème Congrès International de Médecine Physique, vol. II. Barcelone: 1972. p. 543–49.
- Hentz VR, Leclercq C. Surgical rehabilitation for the unusual, incompletely injured tetraplegic patient. In: Hentz VR, Leclercq C, editors. Surgical rehabilitation of the tetraplegic upper limb. London: W.B.Saunders; 2002. p. 211–4.
- Beach WR, Strecker WB, Coe J, Manske PR, Schoenecker RL, Dailey L. Useofthe green transfer in treatment of patients with spastic cerebral palsy: 17 year experience. J Pediatr Orthop. 1991;11:731–6.
- Tardieu G, Tardieu C, Hariga J, Gagnard L. Treatment of spasticity by injection of dilute alcohol at the motor point or by epidural route. Dev Med Child Neurol. 1968;10:555–68.
- Carpenter EB, Seitz DG. Intramuscular alcohol as an aid in management of spastic cerebral palsy. Dev Med Child Neurol. 1980;22:497–501.
- Braun RM, Hoffer MM, Mooney V, et al. Phenol nerve block in the treatment of acquired spastic hemiplegia in the upper limb. J Bone Joint Surg Am. 1973; 55(A):580–5.

- Das TK, Park DM. Botulinum toxin in treating spasticity. Br J Clin Pract. 1989;43:401–2.
- Memin B, Pollak P, Hommel M, Perret J. Traitement de la spasticité par toxine butulique. Rev Neurol. 1992;148:212–4.
- Koman LA, Mooney JF, Smith BP, et al. Management of spasticity in cerebral palsy with botulinum A toxin: report of a preliminary randomized double blind trial. J Pediatr Orthop. 1994;14:299.
- 32. Fehlings D, Rang M, Glazier J, Steele C. An evaluation of botulinum-A toxin injections to improve upper extremity function in children with hemiplegic cerebral palsy. J Pediatr. 2000;137:331–7.
- 33. Graham HK, Aoki KR, Autti-Ramo I, et al. Recommendations for the use of Botulinum toxin type A in the management of cerebral palsy. Gait Posture. 2000;11:67–79.
- deBruin M, Smeulders MJC, Kreulen M. Flexor carpi ulnaris tenotomy alone does not eliminate its contribution to wrist torque. Clin Biomech (Bristol, Avon). 2011;26:725–8.
- Stoffel A. Treatment of spastic contractures. Am J Orthop Surg. 1913;10:611.
- Brunelli G, Brunelli F. Partial selective denervation in spastic palsies (hyponeurotization). Microsurgery. 1983;4:221–4.
- Fraioli B, Nucci F, Baldassarre L. Bilateral cervical posterior rhizotomy for severe spastic syndromes with dyskinesias. Appl Neurophysiol. 1977;78:26–40.
- Bertelli JA, Ghizoni MF, Frasson TR, Borges KSF. Brachial plexus dorsal rhizotomy in hemiplegic cerebral palsy. Hand Clin. 2003;19:687–99.
- Page CM. An operation for the relief of flexion contracture in the forearm. J Bone Joint Surg. 1923;5:233–4.
- Braun RM. Stroke rehabilitation. In: Green DP, editor. Operative hand surgery. New York: Churchill Livingstone; 1982. p. 195–211.
- Zancolli EA, Zancolli Jr ER. Indications opératoires et traitement de la main spastique infantile. Ann Chir Main. 1984;3:66–75.
- Braun RM, Vise GT, Roper B. Preliminary experience with superficialis to profundus tendon transfers in the hemiplegic upper extremity. J Bone Joint Surg Am. 1974;56(A):466–72.
- 43. Keenan MA, Todderud EP, Henderson R, et al. Management of intrinsic spasticity in the hand with phenol injection or neurotomy of the motor branch of the ulnar nerve. J Hand Surg Am. 1987;12A:734–9.
- Omer GE, Capen DA. Proximal bone carpectomy with muscle transfers for spastic paralysis. J Hand Surg Am. 1976;1:197–204.
- Gschwind CR. Surgical management of forearm pronation. Hand Clin. 2003;19:649–55.
- 46. Hoffer MM, Waters RL, Garland DE. Spastic dysfunction of the elbow. In: Morrey BF, editor. The elbow and its disorders. Philadelphia: Saunders; 1985. p. 616–26.
- 47. Cambon Binder A, Leclercq. Anatomical study of the musculocutaneous nerve branching pattern: application for selective neurectomy in the treatment of

elbow flexors spasticity. Surg Radiol Anat, (in press 2014).

- Keenan MAE, Haider TT, Stone LR. Dynamic electromyography to assess elbow spasticity. J Hand Surg Am. 1990;15:607.
- Waters PM, Van Heest A. Spastic hemiplegia of the upper extremity in children. Hand Clin. 1998;14: 119–34.
- Mital MA. Lengthening of the elbow flexors in cerebral palsy. J Bone Joint Surg Am. 1979;61A:515–522.
- Garland DE, Thompson R, Waters RL. Musculo cutaneous neurectomy for spastic elbow flexion in non functional upper extremities in adults. J Bone Joint Surg Am. 1980;62A:108–12.
- Colton CL, Ransford AO, Lloyd-Roberts GC. Transposition of the tendon of the pronator teres in cerebral palsy. J Bone Joint Surg Br. 1976;58(B):220.
- Sakellarides HT, Mital MA, Lenzi WD. The treatment of pronation contractures of the forearm in cerebral palsy. J Hand Surg. 1976;1:79–80.
- Gschwind CR, Tonkin M. Surgery forcerebral palsy: part 1. Classification and operative procedures for pronation deformity. J Hand Surg Br. 1992;17B:391–5.
- McCue FC, Honner R, Chapman WC. Transfer of the brachioradialis for hands deformed by cerebral palsy. J Bone Joint Surg. 1970;52A:1171–80.
- Pinzur MS. Carpectomy and fusion in adult acquired hand spasticity. Orthopedics. 1996;19:675–7.
- 57. Rayan GM, Young BT. Arthrodesis of the spastic wrist. J Hand Surg Am. 1999;24A:944–52.
- Van Heest AE, Strothman D. Wrist arthrodesis in cerebral palsy. J Hand Surg Am. 2009;34A:1216–24.
- Hoffer MM, Zeitzew S. Wrist fusion in cerebral palsy. J Hand Surg Am. 1988;I3A:667–70.
- Carlson MG, Athwal GS, Bueno RA. Treatment of the wrist and hand in cerebral palsy. J Hand Surg Am. 2006;31A:483–90.

- Hoffer MM, Lehman M, Mitani M. Long term followup on tendon transfers to the extensors of the wrist and fingers in patients with cerebral palsy. J Hand Surg Am. 1986;11A:836–40.
- Tonkin M, Gschwind C. Surgery for cerebral palsy. Part 2. Flexion deformity of the wrist and fingers. J Hand Surg Br. 1992;17B:396–400.
- Smith RJ. Surgery of the hand in cerebral palsy. In: Pulvertaft RG, editor. Operative surgery – the hand. London: Butterworth; 1977. p. 215–30.
- Swanson AB. Surgery of the hand in cerebral palsy and muscle origin release procedures. Surg Clin North Am. 1968;48:1129–38.
- Littler JW, Cooley SGE. Restauration of the retinacular system in hyperextension deformities of the proximal interphalangeal joint. J Bone Joint Surg. 1965; 47(A):637.
- Thompson JS, Littler JW, Upton J. The spiral oblique retinacular ligament (SORL). J Hand Surg Am. 1978; 3A:482–7.
- 67. Keenan MA, Korchek JI, Botte MJ, et al. Results of transfer of the flexor digitorum superficialis tendons to the flexor digitorum profundus tendons in adults with acquired spasticity of the hand. J Bone Joint Surg Am. 1987;69:1127–32.
- Matev I. Surgical treatment of spastic "thumb in palm" deformity. J Bone Joint Surg. 1963;45(B):703–8.
- Witthaut J, Leclercq C. Anatomy of the adductor pollicis muscle. A basis for release procedures for adduction contractures of the thumb. J Hand Surg Br. 1998;23(B):380–3.
- Inglis AE, Cooper W. Release of the flexor-pronator origin for flexion deformities of the hand and wrist in spastic paralysis. J Bone Joint Surg. 1966;48(A): 847–57.
- Smith RJ. Tendon transfers of the hand and forearm. Boston: Little, Brown and Co.; 1987.

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