Disorders of the hand and fingers



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Pain and paraesthesia are two symptoms that are common in the hand. Pain is very often the result of either local trauma or overuse. Precise localization is possible in pain that is not referred from a lesion higher up in the limb. Paraesthesia may reflect a proximal lesion and the patient has difficulty in identifying the source of his symptoms.

Disorders of the inert structures

The capsular pattern

Any of the joints of the fingers may become affected by one or other form of arthritis, which results in limitation of movement with a capsular pattern.

The capsular pattern at a finger joint is an equal loss of movement at the beginning and end of the normal range in either direction. Some movement remains possible at the midpoint. Rotations are painful at the extremes of range. In very severe arthritis, rotations may also become limited.

The presence of a capsular pattern indicates that an arthritis has developed, the type of which can be defined from the history. The following questions are relevant:

- *How did it all start*? The possibilities are: no apparent cause, trauma or post-immobilization. A spontaneous onset indicates the possibility of rheumatoid arthritis or of a simple arthrosis. Trauma suggests traumatic arthritis.
- Are other joints affected as well? If they are, this suggests a rheumatoid condition.
- Which joints were affected first, the distal or the proximal joints? Arthrosis usually starts at the distal interphalangeal joints, whereas rheumatoid arthritis tends to start at the metacarpophalangeal joints.
- Is the joint capsule swollen? Swelling often occurs in a rheumatoid or traumatic arthritis.
- Does the joint change colour? The joint becomes red in gout.

Rheumatoid arthritis

Rheumatoid arthritis is undoubtedly the most deforming and most incapacitating disorder of the hand.¹

The symptoms and signs usually develop in the course of a few weeks or months and are often symmetrical. Frequently,

the first symptom is morning stiffness of the fingers. The basic functional examination is usually negative, but tenderness to touch can be elicited. The erythrocyte sedimentation rate is markedly raised.

Early in the course, a capsular pattern develops and one or more metacarpophalangeal joints or proximal interphalangeal joints of one or both hands show the familiar spindle-shaped swelling.

Later, when osseous destruction takes place, a palmar luxation of the fingers occurs and they deviate towards the ulnar side as the result of subluxation in the metacarpophalangeal joints. The fingers may develop the typical buttonhole and swan neck deformities: the former results in hyperextension of the metacarpophalangeal joint, flexion at the proximal interphalangeal joint and extension at the distal interphalangeal joint; the latter results in flexion at the metacarpophalangeal joint, hyperextension at the proximal and flexion at the distal interphalangeal joints. The thumb becomes Z-shaped (typical 'ninety-ninety' deformity): the metacarpophalangeal joint is fixed in 90° flexion, the interphalangeal joint in 90° extension. The joints are also warm to the touch.

Secondary rupture of tendons and ligaments may occur, with subsequent muscular contractures or muscular atrophy.

Treatment is systemic. Only in the initial stage, when few joints are affected, may intra-articular triamcinolone prove effective.

As in any other patient, symptoms may develop that have nothing to do with the patient's rheumatoid arthritis. Trigger finger, carpal tunnel syndrome and de Quervain's disease are common, and another possibility is a ganglion lying between the heads of the second and third metacarpal bones, which gives rise to vague local aching and responds well to aspiration.²

Traumatic arthritis

The typical history of direct contusion, indirect sprain or reduced dislocation of a finger joint indicates the presence of a traumatic arthritis – a very common condition. A chip fracture may also be responsible for the arthritis.

On inspection, a spindle-shaped swelling is often seen, which resembles the swelling of rheumatoid arthritis. Examination further reveals a capsular pattern, and on palpation warmth may be felt, especially after a severe injury. As the arthritis may be combined with a tendinous lesion, resisted movements of the fingers must also be tested.

Traumatic arthritis of the finger joints does not respond satisfactorily to any treatment. Intra-articular injection with a steroid, so useful in traumatic arthritis in the toe joints, affords no corresponding benefit in the fingers. Recovery is spontaneous over 6–18 months, depending on the severity of the original trauma and the age of the patient. Sometimes manual therapeutic techniques may favourably alter the natural course.³ Immobilization is strongly contraindicated.

Arthrosis

Occasionally arthrosis in one joint develops as the result of severe injury but more often the condition has a spontaneous

onset and affects several joints. Women between 40 and 60 years of age are often affected, and there is a strong familial predisposition.⁴

Arthrosis begins at the distal interphalangeal joints, and its knobbly appearance is quite different from rheumatoid arthritis. Both hands are usually affected more or less symmetrically. The index, middle and ring fingers are most usually affected. At the base of the distal phalanx, two small rounded bosses on the dorsum of the joint (Heberden's nodes)⁵ can be seen. A varus deformity may develop at a distal joint, usually at the index. Some years later, the arthrosis may spread to the proximal interphalangeal joints (with the formation of nodes at index and middle fingers - Bouchard's nodes); it seldom reaches the metacarpophalangeal joints. From time to time, a new node forms at an affected joint and the patient will mention some aching or slight pain over 1 or 2 months, during which time the fingertip may occasionally become pink. The colour is mottled and different from the shiny red of gout. After a month or two the discolouration passes off and the node ceases to be painful.

As the joints of the hand are not weight-bearing, arthrosis is not a very painful condition. The patient merely complains of stiffness and aching especially after exertion.

The radiograph clearly shows the usual arthrotic changes – osteophytes and erosion of cartilage.

Heberden's nodes and arthrosis cause hardly any symptoms. They are unsightly and may be associated with aching and clumsiness. Since the distal finger joints finally fix in 45° flexion, arthrodesis seldom brings much improvement unless an intractable painful traumatic arthritis supervenes after injury. Some patients are pleased to have the exostoses removed surgically for cosmetic reasons.

Gout

The hands become involved only very late in the evolution of gout. According to Dieppe and Calvent,⁶ the finger joints are affected in approximately 15% of cases.

The onset and the clinical appearance of chronic gout in elderly men may very closely mimic rheumatoid arthritis but diagnosis is not difficult when the patient is known to have gout and describes recurrent attacks. These typically start in the first metatarsophalangeal joint of the big toe, clear up completely and later spread to other joints. The affected joint usually has a shiny red appearance.

Tophi in the ears and a raised blood uric acid level finally appear but are of little diagnostic aid in the early, doubtful case.

Very characteristic, even diagnostic, is the rapid improvement of the arthritis within 48 hours of colchicine or phenylbutazone administration.

Non-capsular pattern

Unreduced dislocation

Dislocation is sometimes mistaken for traumatic arthritis at the interphalangeal joint of the thumb. The joint is so swollen that

it is not obvious that it is fixed in full extension – a clear non-capsular type of limitation. In late cases, reduction is impossible and surgery is required.

Disorders of the contractile structures

Strains of muscles and tendons in the hand are not infrequent. They have no tendency to spontaneous cure. Diagnosis is not difficult and conservative treatment leads to good results. All the intrinsic muscles of the hand and their short tendons respond immediately to adequate deep transverse friction but not to infiltrations with steroids. In contrast, friction has no effect on the long flexor tendons in the palm but triamcinolone infiltration is successful.⁷

Dorsal interosseous muscles

Pain

A lesion in an interosseous muscle is usually traumatic, either the result of a direct injury or of a fracture of a metacarpal bone. Less commonly it follows overuse (e.g. musicians and keyboard operators).

There are three possible localizations for the lesion: in the muscle belly, in the tendon where it crosses the metacarpophalangeal joint; and at the insertion into the base of the phalanx (Fig. 1).

In the muscle belly, the lesion is found between the metacarpal shafts, usually distally. The patient experiences pain at the dorsal aspect of the hand, elicited by resisted abduction of the extended finger. Passive movement at the metacarpophalangeal joint may cause pain at the extreme range of movement but, as the pain is not felt at the joint itself and resisted movements are also painful, attention is drawn to the interosseous muscle. Careful palpation reveals the exact site of the lesion. This lesion has no tendency to spontaneous recovery and it only responds – and very impressively – to deep transverse friction: two or three treatments suffice.

If the lesion is in the tendon or at the insertion, the pain is accurately felt at one side of one knuckle. The joint may be slightly swollen at the site of the lesion. Passive deviation of the finger away from the painful side is painful, as well as resisted abduction towards the painful side. Again, palpation must be performed very carefully to determine the exact painful spot.

Differentiation from traumatic arthritis is not always easy. Pain on resisted movement clarifies the diagnosis. Differential diagnosis is important because deep transverse friction will cure a tendon lesion but will have no effect on traumatic arthritis.

Untreated, the lesion may go on for years but it responds very well to deep transverse friction. The patient will be cured after four to ten treatments, however long the condition has lasted.

Technique: friction to the muscle belly

The patient sits at the couch with the hand resting on it. The therapist sits opposite the patient. The tender spot between the shafts of the metacarpal bones is palpated for, keeping the finger as parallel as possible to the metacarpals. Friction is imparted with the middle finger, reinforced by the index finger (Fig. 2a). The movement is pure pronation—supination, transverse to the muscular fibres.

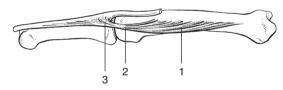


Fig 1 • Sites of lesions of a dorsal interosseous muscle: 1, in the muscle belly; 2, in the body of the tendon; 3, at the insertion into the dorsal aponeurosis.





(b)

0

Fig 2 • Friction to (a) the belly and (b) the tendon of a dorsal interosseous muscle.

Technique: friction to the tendon

It is sometimes difficult to get the finger to the exact spot because it lies more or less between the knuckles. Therefore the following procedure is used. With one hand the therapist takes the patient's hand, with the fingers in the palm of the patient's hand. The tips of the middle and ring fingers lie on the palmar aspect of the knuckle that contains the lesion and push the bone upwards. At the same time the thumb is laid on the dorsal aspect of the adjacent knuckle, which is pushed downwards. This manœuvre brings the affected tendon within reach of the thumb of the other hand. With the fingers of this hand counterpressure is exerted (Fig. 2b). The friction is given with the thumb, starting at the palmar aspect of the tendon and ending at the dorsal side of it. The movement is achieved by a supination movement of the forearm.

Weakness

Weakness of one or more interosseous muscles is the result of a neurological condition and is often one of the first signs of amyotrophic lateral sclerosis.

Weakness and wasting of one interosseous muscle only occurs in cases of localized pressure (usually occupational) on the deep palmar branch of the ulnar nerve in the palm of the hand (cyclist's palsy).8

A cervical rib can cause pressure on the lower trunk of the brachial plexus: apart from some of the interosseous muscles, the ulnar half of the flexor digitorum profundus and the flexor carpi ulnaris are also usually weak.

A first thoracic nerve root lesion may also be responsible for weakness of the small muscles of the hand.

Thenar muscles

A lesion of a thenar muscle may follow an abduction sprain of the thumb. The origin of the oblique portion of the adductor pollicis muscle at the palmar aspect of the base of the (second or) third metacarpal bone is most commonly affected. Resisted adduction of the thumb is painful, as is passive abduction.

Treatment consists of a few sessions of deep transverse friction.

Technique: friction

The patient sits at the couch, the supinated arm resting on it and the hand over the edge. The therapist sits opposite the patient. With the contralateral hand the wrist and the thumb are brought into extension in order to stretch the tendon and to bring it within reach of the fingers (Fig. 3).

Friction is imparted with the thumb of the other hand, starting at the radial aspect of the insertion and ending at the ulnar aspect.

Flexor tendons

A tenosynovitis may develop in the long flexor tendons in the palm of the hand. The rheumatoid type often presents with





Fig 3 • Friction to the adductor pollicis.

coarse crepitus on movements. This chronic tenosynovitis causes few or no symptoms. If necessary, the lesion responds well to infiltration with triamcinolone.

Trigger finger

Trigger finger is a common condition causing pain and disability in the hand. It can arise spontaneously or can be the result of repetitive minor trauma or a complication of rheumatoid arthritis. Primary trigger finger occurs most commonly in the middle fifth to sixth decades of life and up to six times more frequently in women than men.9 The third or fourth finger is most commonly involved. The disorder is caused by swelling of one of the digital flexor tendons just proximal to the metacarpophalangeal joint, in combination with narrowing of its tendon sheath.

The condition presents with discomfort in the palm during movement of the involved digits. Gradually the flexor tendon causes painful popping or snapping as the patient flexes and

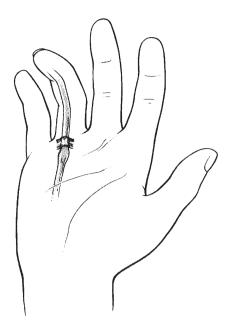


Fig 4 • Trigger finger.

extends the digit. As the condition progresses, the digit may begin to lock in a particular position, more often flexion, which may require gentle passive manipulation into full extension. A snap accompanies disengagement. Trigger finger arises through a discrepancy in the diameter of the flexor tendon and its sheath at the level of the metacarpal head known as the Al pulley. This thickening of the sheath can result in a narrowed tunnel for tendon excursion and ultimately result in a block to tendon excursion. However, the flexors are usually powerful enough to overcome this obstruction, whereas the weaker extensors are less able to counteract the block, resulting in the finger being locked in flexion. A painful nodule, the result of intratendinous swelling, is easily palpated in the palm, just proximal to the head of the metacarpal bone (Fig. 4).

If the condition gives rise to painful symptoms, infiltration with 10 mg of triamcinolone acetonide in and around the flexor sheath at the level of the A1 pulley should be given. Steroid injection is an effective method of treating patients with trigger finger and should be considered as the preferred treatment. If the result is not adequate, the tendon sheath can be slit up either at open surgery or percutaneously. Success rates have been reported as over 90%; however, its use is tempered by the risk of digital nerve or artery injury and tendon bowstringing. Is,19

Technique: infiltration of a trigger finger (Fig. 5)

The flexor tendon, the metacarpophalangeal joint and the painful nodule are identified. A 1 ml syringe, filled with 10 mg triamcinolone is fitted with a thin needle, 2 cm long. The needle, inserted at a point on the tendon, 1 cm proximal of the palpating finger, is directed at an angle of 30° to the horizontal until tough tendinous resistance is felt just underneath the palpating finger. Pressure is applied to the plunger and the needle gently manœuvred so that the tip is either deeper or more superficial to its initial position and the steroid will flow



Fig 5 • Infiltration for trigger finger.

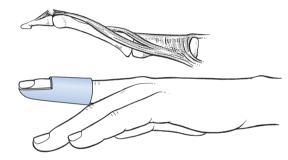


Fig 6 • Mallet finger. Splinting in extension by a stack splint.

freely. This procedure is repeated a few times medially and laterally.

Tendon rupture

Mallet finger

As the result of an injury that flexes the distal interphalangeal joint while it is actively held in extension, the long extensor tendon may rupture (Fig. 6) or may become detached (avulsion fracture) from the distal phalanx. Distinction between the two can be made by radiography.

On examination, the distal joint is held in flexion and the patient is not able to extend it actively. Passive testing is normal

Mallet injuries with and without a bony fragment may be effectively treated by splinting the distal interphalangeal joint in extension for 8 weeks, followed by 1 month of night splinting. Dorsal, volar, or pre-fabricated stack type splints all can be used but care must be taken to avoid dorsal skin ischemia. When a bone fragment has been retained with the extensor tendon, the opportunity to heal is enhanced because of the greater healing potential of bone compared to tendon. The proximal interphalangeal joint should be left free, as

immobilization of the proximal interphalangeal joint and its resultant stiffness may cause more morbidity than the original injury. Patients are counseled to expect a slight extensor lag $(5-10^\circ)$ under the best circumstances, with a mild loss of total motion. Internal fixation of mallet fingers is recommended in cases of volar subluxation of the distal phalanx or in cases where the dorsal component is greater than one-third of the joint surface. In the proximal property of the proximal phalanx or in cases where the dorsal component is greater than one-third of the joint surface.

Ruptured flexor tendon

Rarely, a tendon of the flexor digitorum longus may rupture at the level of the base of the distal phalanx. The whole tendon recoils into the palm of the hand. The distal interphalangeal joint cannot be actively flexed. Passive movement remains normal. Surgical repair is indicated.

Dupuytren's contracture

This disorder, named after G. Dupuytren, ²⁵ is a painless contracture of the palmar aponeurosis. The aetiology is still unknown but it seems to occur more often in combination with alcoholism, disorders of the liver, diabetes and epilepsy. ^{26–28} It is common in men after the age of 30, whereas in women it does not occur under the age of 45. ²⁹ A small node in the palm of the hand is the initial symptom. Further contraction of the palmar fascia leads to flexion contracture of the fingers, especially the ring and little fingers, which are affected in 85%. ³⁰

Once the contracture becomes disabling, because permanent flexion interferes with normal activities, the only treatment is surgery.

Südeck's atrophy

Reflex sympathetic dystrophy syndrome (RSDS), or Südeck's atrophy, is a curious disorder that is not uncommon. Different terminology has been used to label the same condition: Südeck's atrophy, causalgia, algodystrophy, algoneurodystrophy, reflex sympathetic dystrophy syndrome (RSDS).³¹ It is a controversial condition. The controversy concerns the manner in which the sympathetic nervous system is involved in RSDS. It was redefined in 1996 by an ad hoc International Association for the Study of Pain task force that suggested changing the name to 'complex regional pain syndrome' (CRPS).^{32–34} CRPS type 1 is reflex sympathetic dystrophy; type II is causalgia.³⁵

It occurs most commonly as a complication of major or minor trauma (up to 5% of patients with traumatic injuries) or in patients with myocardial ischaemia (5–20%) or hemiplegia (12–20%).³⁶ Kozin³⁷ stated that RSDS was found to occur most frequently after fracture (25%) or other trauma (27%). In 27% of cases, no specific precipitating event could be identified. Central nervous system or spinal disorders, myocardial ischaemia and peripheral nerve injury are responsible for, respectively, 12, 6 and 4% of cases.

The pathogenesis is unclear. Some authors regard it as an injury to the peripheral nerve mechanism; others propose the

theory of a central neurological dysfunction, causing abnormal activity in the sympathetic nervous system.

The main symptom is post-traumatic pain which is disproportionate to the injury. It spreads beyond the distribution of any single peripheral nerve, is usually felt distally in the arm and is often described as 'burning'. Tenderness is always present and is occasionally associated with allodynia and enhanced sensitivity to palpation. Swelling of the affected part is often present and pitting or non-pitting oedema may be found. Dystrophic skin changes are seen: nail and hair changes (hypertrichosis), shiny and taut skin and loss of wrinkling.

The radiographic changes (patchy osteoporosis) have been well described by Südeck,³⁹ Kienböck⁴⁰ and Hermann *et al.*⁴¹ A bone scan may be useful to arrive at a diagnosis.

Differential diagnosis has to be made with septic arthritis, rheumatoid arthritis, systemic lupus erythematosus, Reiter's syndrome and peripheral neuropathy.

The treatment of complex regional pain syndrome type I (CRPS-I) is subject to much debate. There is more or less concensus about the following. For pain treatment, the WHO analgesic ladder is advised with the exception of strong opioids. For neuropathic pain, anticonvulsants and tricyclic antidepressants may be considered. For inflammatory symptoms, free-radical scavengers (dimethylsulphoxide or acetylcysteine) are advised. To promote peripheral blood flow, vasodilatory medication may be considered. Percutaneous sympathetic blockades may be used to increase blood flow in case vasodilatory medication has insufficient effect. And the decrease functional limitations, standardised physiotherapy and occupational therapy are advised.

Disorders of the hand and fingers are summarized in Box 1.



Box 1

Summary of disorders of the hand and fingers

Disorders of the inert structures

The capsular pattern

Rheumatoid arthritis

Traumatic arthritis

Arthrosis

Gout

The non-capsular pattern

Dislocation

Disorders of the contractile structures

Dorsal interosseous muscle

Thenar muscle

Flexor tendons

Trigger finger

Rupture of a tendon

Mallet finger

Ruptured flexor tendon

Dupuytren's contracture

Südeck's atrophy (reflex sympathetic dystrophy syndrome)

References

- Tubiana R, Ghozlan R, Minkes CJ. Main rhumatoïde, Appareil Locomoteur. Encyclopédie Médicochirurgicale, Paris, 1978;10:14067.
- 2. Rhoades CE, Gelberman RH, Manjarris JF. Stenosing tenosynovitis of the fingers and thumb. Clin Orthop Rel Res 1984;190:236.
- Mink AJF, ter Veer HJ, Vorselaars JACTH. *Extremiteiten, Functie-onderzoek en Manuele Therapie*. 6th ed. Utrecht: Bohn, Scheltema & Holkema; 1990. p. 300.
- 4. Veys EM, Mielants H, Verbruggen G. Reumatologie. Ghent: Omega Editions; 1985. p. 621.
- Heberden W. Commentaries on the History and Cure of Diseases. London: Payne; 1802
- Dieppe PA, Calvent P. Crystals and Joint Disease. London: Chapman & Hall; 1982. p. 145.
- Cyriax JH. Textbook of Orthopaedic Medicine, vol I, Diagnosis of Soft Tissue Lesions. 8th ed. London: Baillière Tindall; 1982. p. 194.
- 8. Haloua JP, Collin JP, Coudeyre L. Paralysis of the ulnar nerve in cyclists. *Ann Chir Main* 1987;**6**:282.
- 9. Weilby A. Trigger finger. Incidence in children and adults and the possibility of a predisposition in certain age groups. *Acta Orthop Scand* 1970;41:419–27.
- 10. Sampson SP, Badalamente MA, Hurst LC, Seidman J. Pathobiology of the human Al pulley in trigger finger. *J Hand Surg (Am)* 1991;**16(4)**:714–21.
- 11. Taras JS, Raphael JS, Pan WT, et al. Corticosteroid injections for trigger digits: is intrasheath injection necessary? *J Hand Surg [Am]* 1998;**23**:717–22.
- Kazuki K, Egi T, Okada M, et al. Clinical outcome of extrasynovial steroid injection for trigger finger. Hand Surg 2006;11: 1–4.
- 13. 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* 1992;17B(1): 69–70. ⊗
- 14. Buch-Jaeger N, Foucher G, Ehrler S, Sammut D. The results of conservative management of trigger finger. A series of 169 patients. *Ann Chir Main Memb Super* 1992;**11(3)**:189–93.
- 15. 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.
- 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 1992;17A(1):114–7.
- 18. Pope DF, Wolfe SW. Safety and efficacy of percutaneous trigger finger release. *J Hand Surg [Am]* 1995;**20**:280–3.
- 20. Wehbé MA, Schneider LH. Mallet fractures. *J Bone Joint Surg* 1984;**66A**:658.
- 21. Kalainov DM, Hoepfner PE, Hartigan BJ, et al. Nonsurgical treatment of closed mallet finger fractures. *J Hand Surg [Am]* 2005;30:580–6.
- 22. Hooyboer PGA, Vuursteen PJ. De behandeling van de mallet finger; stackspalk of tenodermodese. *Ned Tijdschr Geneeskd* 1990;4:134.
- 23. Patel MR, Desai SS, Bassini-Lipson L. Conservative management of chronic mallet finger. *J Hand Surg [Am]* 1986;11:570–3.
- 24. Badia A, Riano F. A simple fixation method for unstable bony mallet finger. *J Hand Surg [Am]* 2004;29:1051–5. ⋄
- 25. Dupuytren G. Leçons Orales de Clinique Chirurgicale, vol 1. Paris: Baillière; 1832.
- Hueson JT. Dupuytren's contracture. In: Flynn JE, editor. *Hand Surgery*. 3rd ed. Baltimore: Williams & Wilkins; 1982. p. 797
- Lamb DW. Dupuytren's disease. In: Lamb DW, Kuczynski K, editors. *The Practice of Hand Surgery*. Oxford: Blackwell; 1981. p. 470.
- 28. James JIP. The genetic pattern of Dupuytren's disease and idiopathic epilepsy. In: Hueston JT, Tubiana R, editors. Dupuytren's Disease. 2nd English ed. Edinburgh: Churchill Livingstone; 1985. p. 94.
- Ewing J. Neoplastic diseases. In: Rothman R, Simeone F, editors. A Treatise on Tumors. 3rd ed. Philadelphia: Saunders; 1982. p. 368.
- 30. Lukes RJ, Collins RD. New approaches to the classification of lymphomata. *Br J Cancer* 1975;**31**:1.
- 31. Alvarez-Lario B, Aretxabala-Alcibar I, Alegre-Lopez J, Alonso-Valdivielso JL. Acceptance of the different denominations for reflex sympathetic dystrophy. *Ann Rheum Dis* 2001;**60(1)**:77–9.

- 32. Marchettini P, Lacerenza M, Formaglio F. Sympathetically maintained pain. Current Rev Pain 2000;4(2):99–104.
- 33. Stanton-Hicks M. Reflex sympathetic dystrophy: a sympathetically mediated pain syndrome or not? *Current Rev Pain* 2000;4(4):268–75. ♠
- 34. Stanton-Hicks M. Complex regional pain syndrome (type I, RSD; type II, causalgia): controversies. Clin J Pain 2000;16(2 Suppl):S33–40. ♠
- 35. Viel E, Ripart J, Pelissier J, Eledjam JJ. Management of reflex sympathetic dystrophy. *Ann Méd Interne (Paris)* 1999;150(3):205–10.
- Davis SW. Shoulder–hand syndrome in a hemiplegic population: 5 year retrospective study. Arch Phys Med Rehabil 1977;58:3553.
- 37. Kozin F. The painful shoulder and reflex sympathetic dystrophy syndrome. In: McCarty DJ, editor. *Arthritis and Allied Conditions*. 10th ed. Philadelphia: Lea & Febiger; 1985. p. 1322.
- 38. Goldstein DS, Tack C, Li ST. Sympathetic innervation and function in reflex sympathetic dystrophy. *Ann Neurol* 2000;48(1):49–59.
- Südeck P. Über die akute entzündliche Knochenatrophie. Arch Klin Chir 1900;62:147.
- 40. Kienböck R. Über akute Knochenatrophie bei Entzündungsprocessen an den Extremitäten (falschlich sogenannte Inaktivitätsatrophie der Knochen) und ihre Diagnose nach dem Röntgenbild. Wien Med Wochenschr 1901;5:1345.
- Hermann LG, Reinecke HG, Caldwell JA. Posttraumatic painful osteoporosis: a clinical and roentgenological entity. Am J Roentgenol 1942;47:353.
- 42. Perez RS, Zollinger PE, Dijkstra PU, et al. CRPS I task force. Evidence based guidelines for complex regional pain syndrome type 1. BMC Neurol 2010; 10:20.
- 43. Kozin F. Reflex sympathetic dystrophy syndrome. *Bull Rheum Dis* 1986;**36**(3):632–8.
- 44. Bushnell TG, Cobo-Castro T. Complex regional pain syndrome: becoming more or less complex? *Manual Therapy* 1999;4(4):221–228.
- 45. Oerlemans HM, Oostendorp RA, de Boo T, et al. Adjuvant physical therapy versus occupational therapy in patients with reflex sympathetic dystrophy/complex regional pain syndrome type I. Arch Phys Med Rehabil 2000;81(1):49–56. ♠