Hand Function in Common Hand Problems

14

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Introduction

Positioning the hand in space and placing it in functional positions is critical for us if we are to interact effectively with our environment. Our hands are our most important tools for survival and fun. Diseases with hand involvement affect its functional status and quality of life.

This chapter presents a discussion of several commonly seen hand impairments that are likely to influence function. The goal of this chapter is to provide a brief, practical guide to evaluation of some common, non-traumatic, functional hand problems.

It is important to proceed in a systematic way in evaluating these problems, using standardized assessments and considering possible contributions of posture and ergonomics of the work, home, and leisure activities. Included in this chapter are syndromes of various etiologies, but overuse is often a common component.

Included are the following:

Carpal tunnel syndrome is the most common compressive neuropathy of the hand. Its symptoms, often nonspecific, usually include dysesthesias along the median nerve distribution.

Trigger finger is characterized by a snapping or locking sensation and limitation of full flexion of the finger. Often it is the third, fourth, or fifth digit. Occasionally it remains in a fixed flexion position.

De Quervain's tenosynovitis is associated with pain on the radial aspect of the thumb. There is usually pain on palpation or on movement when one is using the thumb for pinching or gripping.

Dupuytren's contracture is the result of hypertrophy of palmar fascia affecting the fifth digit in about 70 % of people so affected. It is a clinical diagnosis made with the presence of palpable nodules and cords in the palmar fascia and associated with flexion contracture of the fourth and fifth digit.

Chronic regional pain syndrome is a chronic, neuropathic pain syndrome characterized by autonomic dysfunction and severe pain that may lead to crippling contractures of the limbs. The patient often presents with a cool extremity, color changes (ruddy or bluish), swelling, and allodynia.

Focal dystonia, also called writer's cramp/musician's cramp, is maladaptive response of the brain to repetitive performance of stereotyped hand movements. Usually, the individual presents with cramping and pain when they repeat the inciting task. When not used in that fashion, the hand appears normal.

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Measurement

A comprehensive hand evaluation, which includes descriptive and quantitative assessment, is essential to understand the impact of impairments on function. The use of standard imaging (x-ray, computed tomographic, magnetic resonant imaging, real time ultrasound, Doppler ultrasound blood flow) and standardized measurement are essential for proper treatment [1].

Carpal Tunnel Syndrome

Carpal tunnel syndrome (CTS) is one of the most frequently encountered problems and the most common compressive neuropathy in the upper extremity [2]. The median nerve and the flexor tendons pass through a tunnel at the wrist limited by carpal bones and the transverse carpal ligament (Fig. 14.1). Numbness and paresthesias are felt in the distribution of the median nerve (Fig. 14.2). In the United States, 15 % of the general population has symptoms consistent with CTS for which they seek medical attention. Symptoms are often non-diagnostic, because those associated with CTS are similar to radiculopathy, wrist arthritis, and tendonopathies. Therefore, electromyographic studies are usually considered necessary for confirmation. Using this as the diagnostic criterion, CTS has a 3 % prevalence in women and 2 % in men. Prevalence is greatest in women >55 years [3], in those who are obese, smoke, or have diabetes mellitus [4, 5]. A phenomenon called the "double-crush" syndrome has been reported, which has established the association between cervical spine radiculopathy, thoracic outlet abnormalities, and CTS [6].

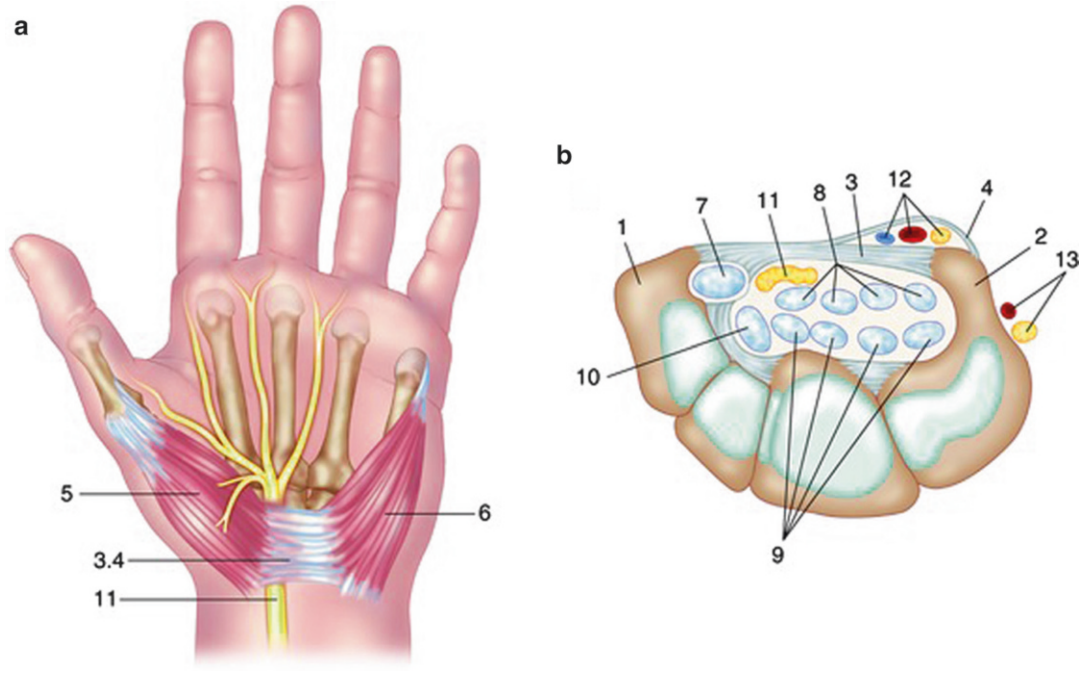

Fig. 14.1 Carpal tunnel syndrome: Anatomy of the carpal canal. (**a**) Typical median nerve and hand anatomy at the level of the transverse carpal ligament. (**b**) Carpal tunnel cross-sectional anatomy. (*1*) Trapezium tubercle, (*2*) hook of the hamate, (*3*) transverse carpal ligament, (*4*) palmar carpal ligament, (*5*) thenar muscles, (*6*) hypothenar muscles, (*7*) flexor carpi radialis tendon, (*8*) flexor digitorum superficialis tendon, (*9*) flexor digitorum profundus tendon, (*10*) flexor pollicis longus tendon, (*11*) median nerve, (*12*) ulnar artery, vein, and nerve superficial branches, (*13*) ulnar artery, vein, and nerve deep branches (With kind permission from Springer Science+Business Media: *Reoperative Hand Surgery*, Reoperative Options for Compressive Neuropathies of the Upper Extremity, 2012, Kang JR and Gupta R)

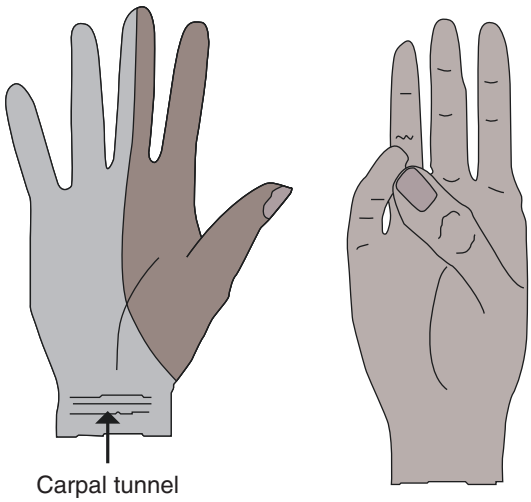

Fig. 14.2 Carpal tunnel syndrome: Area of sensation (*dark gray* in the *left picture*) and motor function (opposition of the thumb in the *right picture*) supplied by the median nerve

The diagnosis of carpal tunnel syndrome is based on history and clinical evaluation. Electromyogram (EMG) is often used for diagnostic confirmation of CTS. It can measure the extent of damage and demyelination of the median nerve [7]. In mild cases, there may be an absence of electromyographic and nerve conduction changes. As symptoms progress, sensory distal latency is usually the first abnormal EMG finding. Therefore, the diagnosis of CTS is first established on history and clinical findings and then may be confirmed by EMG evaluation. Recently there have been multiple reports about the usefulness of ultrasound evaluation of the median nerve to diagnose CTS. These studies have shown that there is a change in the cross-sectional area of the median nerve when CTS is present [8–10].

The carpal tunnel is located just distal to the palmar wrist crease. It is surrounded on three sides by the carpal bones, creating a fixed volume of space. The radial wall is bordered by the scaphoid and trapezium and the ulnar by the hamate and dorsally by the lunate and capitate. The boney arch is covered by a thick fibrocartilaginous band called the *flexor retinaculum* (or transverse carpal ligament). Tendons of the flexor superficialis (FDS) and flexor profundus (FDP) and pollicis longus (FPL) course through the carpal tunnel [11]. The median nerve travels with these innervating the thenar muscles and providing sensation to the radial three and one-half digits. CTS is therefore associated with motor and sensory findings.

Normal pressure within the carpal tunnel is 7–8 mm Hg with the wrist in neutral. Increased pressure of 30 mm Hg can result in symptoms of CTS and 90 mm Hg can be observed with wrist flexion and extension [12, 13]. This pressure increase causes relative ischemia and impaired nerve conduction of the median nerve [14, 15].

The prevalence of CTS increases with pregnancy, inflammatory arthritis, distal wrist fracture, amyloidosis, hypothyroidism, diabetes, and acromegaly and in individuals who use corticosteroids and estrogens [16]. One third of all cases of carpal tunnel are associated with these medical conditions [17]; diabetes is the most commonly associated diagnosis [16].

Cervical radiculopathy has been thought to potentiate CTS, causing the “double-crush” syndrome. The “double-crush syndrome” is a condition in which compression of an axon at one location makes it more sensitive to effects of compression at another [18]. For this to be true, one would need to show that there is compression of an axon at a primary location which causes sensitization at another location due to impaired axoplasmic flow [19]. There have been several review articles casting doubt on this, both from the theoretical physiological basis and from physical findings. Mechanical explanations, stemming from muscle imbalance due to positioning and/or postural changes, have been discussed as potential explanations [20, 21]. CTS is frequently associated with specific occupational activities. The repetitive use of tools that vibrate, such as drills and equipment used in food processing plants and mills, may cause CTS. Continuous compression of the median nerve with the wrist in flexion is also associated with CTS [22]. Debate remains as to the association of CTS and computer keyboard work [23, 24]. There remains considerable debate about whether CTS is a result of repetitive stress without other factors being present [24, 25].

The typical symptoms of CTS are numbness, tingling, pain, burning, or a combination of these [16]. These symptoms occur in the radial three and one half digits: the thumb, index, middle, and half of the ring finger. CTS often causes nocturnal awakening secondary to the hand paresthesias. These nocturnal symptoms are 51–77 % sensitive and 27–68 % specific for CTS [26]. Gripping, driving, holding vibrating objects, or prolonged pinching, such as holding a book, may result in increased paresthesias. Many patients describe relief of their symptoms with shaking of the hands, a phenomenon called the "flick sign" [27]. With progression, patients may describe an awkward feeling or weakness of the hand and begin dropping objects.

Physical examination usually begins with the exclusion of any cervical, shoulder, or elbow pathology, which may produce similar symptoms. C-6 radiculopathies are often confused with CTS because the sensory symptoms involve the radial aspect of the hand. Strength testing should include wrist flexion-extension, grip, and thumb opposition. Specific CTS provocative tests include Phalen's test, in which the wrist is held in full passive wrist flexion. This position increases pressure within the carpal tunnel and may reproduce paresthesias in individuals with CTS. This test has a wide reported range of sensitivity and specificity (40–80 %) [28]. The time to the development of paresthesias should be noted because it can be used to monitor change with treatment. Tinel's test involves tapping the median nerve just proximal to the transverse carpal ligament [29]. Reproduction of the paresthesias into the hand by the Tinel's test is 20–60 % sensitive and 67–87 % specific for CTS [28, 29].

Carpal tunnel compression involves pressure placed with the examiner thumbs or indexes or long fingers over the carpal tunnel. This pressure is maintained for 30 s to 1 min and if positive will reproduce paresthesias. Durkan [30] believes that this test is more sensitive and specific for CTS than Tinel's or Phalen's test.

The function in CTS is commonly assessed by Disabilities of the Arm, Shoulder, and Hand (DASH) scale, Boston Questionnaire (BQ), and Michigan Hand Outcome Questionnaire (MHQ). These questionnaires are valid, reliable, and responsive in CTS [31, 32] (Table 14.1).

Table 14.1 The carpal tunnel syndrome assessment

Maneuvers	Phalen's maneuver (Hold wrist in flexion 60 s); carpal tunnel compression, percussion along median nerve (Tinel's sign)
Neurological tests	2-point discrimination, Semmes-Weinstein filament test (threshold of >2.83 in radial digits)
Electromyography	Fibrillation potentials, sharp waves; sensory latency >3.4 ms; motor latency >4.5 ms compared with unaffected hand
Functional tests	Disabilities of the Arm, Shoulder, and Hand (DASH), Boston Questionnaire (BQ), Michigan Hand Outcome Questionnaire (MHQ)
Physical findings	Wasting of thenar muscles, decreased pinch (thumb/index finger), and/or grasp

A review of nonsurgical interventions is available for the reader. Their application is clinically accepted, and there is evidence of a moderate therapeutic effect [33]. Treatment of CTS begins with modification of repetitive or awkward activities that precipitate paresthesias. Splinting the wrist in a neutral position at night has been demonstrated to reduce symptoms in 80 % of patients [34]. Nonsteroidal anti-inflammatory drugs (NSAIDs), diuretics, vitamin B_6, and oral steroids have been tested, but no specific recommendations have been given for their prolonged usage [16]. Therapeutic interventions such as ultrasound, iontophoresis, gentle stretching and strengthening exercises, ice, and carpal tunnel protection principles may be employed. Protection principles stress avoidance of positions or activities that increase pressure within the carpal tunnel. Nerve and tendon gliding exercises have been described and are thought to be useful [35]. Acupuncture and yoga have also been demonstrated to decrease symptoms [36].

Corticosteroid injections into the carpal tunnel are recommended if splinting and other conservative measures fail to reduce the symptoms.

They have been shown to decrease symptoms in 75 % of patients and improve nerve conduction [37]. One study suggests that procaine is as effective as triamcinolone in controlling symptoms [38]. These injections are performed in a sterile fashion with needle placement ulnar to the palmaris longus. The needle is directed dorsally, distally, and radially at a 45° angle. In patients with severe CTS, 80 % have return of symptoms in 1 year despite appropriate conservative care.

If the patient has signs or symptoms of constant numbness, loss of sensation, or thenar muscle atrophy lasting longer than 1 year, serious consideration of surgery is recommended [11]. Surgery has been shown to be an effective intervention for CTS. The techniques, using open carpal tunnel release or endoscopic release, have been reviewed and compared [39]. Long-term surgical outcomes have some persistent symptoms, such as pain, inability to perform full wrist extension, and persistent numbness and tingling in some [40]. Postoperative rehabilitation versus home exercises seem to have the same outcomes, except that it has been shown that rehabilitation hastens the time to return to work [41].

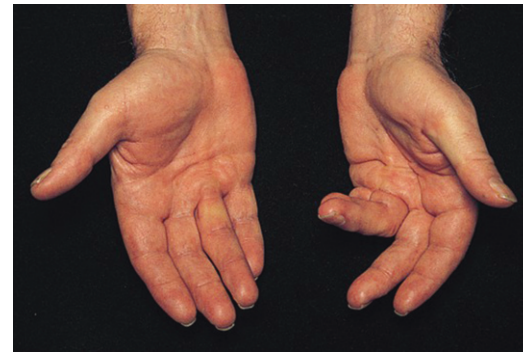

Fig. 14.3 Trigger finger: Stenosing tenosynovitis (trigger finger). (**a**) Synovitis of the tendon sheaths can lead to swelling, limitation of motion, and tendon rupture. Stenosing tenosynovitis can lead to "trigger finger," evident in the fourth finger of the left hand. Triggering occurs when the inflamed tenosynovial tissue cannot move through the tendon sheath. Stenosis of the A-1 pulley can be palpated in the palm just proximal to the affected metacarpophalangeal joint. (**b**) Stenosing tenosynovitis. Tenosynovitis of the flexor tendon can lead to the trigger finger syndrome. With tenosynovitis, the digit is blocked in the flexed position, making extension difficult or even impossible. If the affected tendon is able to pass through the fibrous tendon sheath, a palpable "pop" may be detected. The action may be painful. The tendon may also be blocked in the extended position. Swelling of the tenosynovium proximal to the stenosed annular ligaments may be palpable in the palm as swelling (Courtesy of Alan T. Bishop, MD.)

Trigger Finger or "Stenosing Tenosynovitis"

The sensation of a finger catching or locking in a fixed position is common. This so-called trigger finger or stenosing tenosynovitis is a disorder characterized by snapping of the flexor tendon of the digit (Fig. 14.3). This includes both the profundus and superficialis, acting as pulleys to maintain the position of the tendon [42]. The trigger finger is now thought to be a chronic rather than acute problem and has been described as a disproportion between the sheath and its contents [43]. The most commonly affected area is the distal metacarpal. Sometimes a small nodule can be palpated. On physical examination, one may find a mild flexion deformity of the proximal interphalangeal joint and limitation of full flexion, with the inability to reach the fingertip to the mid-palmar crease. When the condition is chronic, it may progress to a situation in which the finger (often the middle and/or ring finger) becomes fixed in flexion and extension is limited [44]. Pain is not the most frequent presenting symptom.

The pathomechanics include a thickening of the A-1 pulley or flexor tendon owing to sheer or compression forces with inflammatory changes occurring during the acute phase [44, 45]. In chronic conditions, no inflammatory changes are noted, but the tendon is often attenuated [46]. For this reason, the nomenclature of "stenosing tenosynovitis" has lost favor. Chronic conditions result in degenerative changes consistent with fibrocartilaginous proliferation of the A-1 pulley or tendon. The pathologic thickening results in a disparity of the tendon pulley configuration [42]. This size differentiation causes a mechanical locking of the tendon proximal to the A-1 pulley

with finger flexion. Once the tendon is locked in the flexed position, the weaker finger extensors have difficulty overcoming the resistance [47]. When the stuck tendon does release during extension, there is a painful snapping in the region of the MCP joint.

When children have trigger finger, they are usually younger than 6 years [48]. In adults, it is more common in people over 40 years, women, and those with diabetes mellitus and limited joint mobility [49, 50]. The thumb of the dominant hand is most commonly affected, followed by the middle and ring fingers [49]. The symptoms usually consist of a snapping or locking sensation with full flexion of the digit. This sensation is usually painful, but nonpainful conditions have been described. The onset is usually gradual, over several months, but in certain situations can be due to trauma or carpal tunnel release [42]. The symptoms of locking or clicking phenomena are usually worse in the morning and after repetitive gripping or pinching-type activities.

Examination of the finger is usually unremarkable unless reproduction of the locking phenomena can be observed. Most often, a tendon nodule or crepitus can be felt over the palmar aspect of the MCP joint in the region of the A-1 pulley [42]. Grip strength can be diminished secondary to pain. Ligament and neurovascular integrity is normal. No diagnostic tests are confirmatory for this condition. X-rays have not been found to show any abnormality correlated with trigger finger [51]. Serologic testing should be done to check for the presence of underlying conditions such as diabetes mellitus, hypertension, and inflammatory arthritis, which are risk factors for trigger finger.

A trigger finger can lead to disabling pain and may influence work. Symptom control has been reported and ultrasound, iontophoresis, and ice may relieve symptoms [52]. Evans and associates further reported 73 % success in using a flexion-blocking splint at the MCP for 3 weeks [52]. Their protocol also included limiting activities requiring grasp, active flexion or repetitive stress, and hooked-fish exercises. Colbourn et al. confirmed these findings but required 6 weeks of continuous splint usage [53].

Corticosteroid injections have been reported to be somewhat efficacious in the treatment of trigger finger [54, 55]. There have been two small, randomized studies. Newport and associates [56] reported that one to three injections of local anesthetic and cortisone were associated with resolution or improvement in 77 % of 338 fingers. Marks [57] reported that 84 % of trigger fingers and 92 % of trigger thumbs responded to a single injection. This increased to 91 % and 97 %, respectively, with a second injection. Beneficial effects with cortisone are superior to those of placebo and last up to 12 months [56].

Surgical intervention has been advocated if injection therapy does not offer benefit. There has been a plethora of surgical information regarding A-1 pulley releases for the treatment of trigger finger. Thorpe [58] reported that of 53 operations, 60.4 % were completely successful and 11.3 % had incomplete resolution with persistence of clicking and pain within the first year after surgery. Long-term outcomes from these procedures are not well documented.

De Quervain's Tenosynovitis

De Quervain's tenosynovitis is an inflammatory process involving the extensor pollicis brevis and abductor pollicis longus tendons on the radial aspect of the wrist. It is characterized by radial-sided wrist pain at the first dorsal compartment (Fig. 14.4). Presenting symptom is usually pain on palpation or on movement, typically pinching or gripping movement involving the thumb. This most commonly affects women between the ages of 35 and 55 years [59, 60], at a 10-fold increase compared with men. Repetitive, prolonged unaccustomed posturing of the thumb or non-neutral wrist movements usually provokes symptoms [61]. Waitresses, nurses, garment workers, maids, assembly line workers, and machine operators are at greater risk for development of this condition [61, 62]. Pathogenetically, the process starts as inflammation within the first dorsal compartment. Not uncommonly, it recurs or fails to fully heal/repair the tendon pathology, leading to thickening

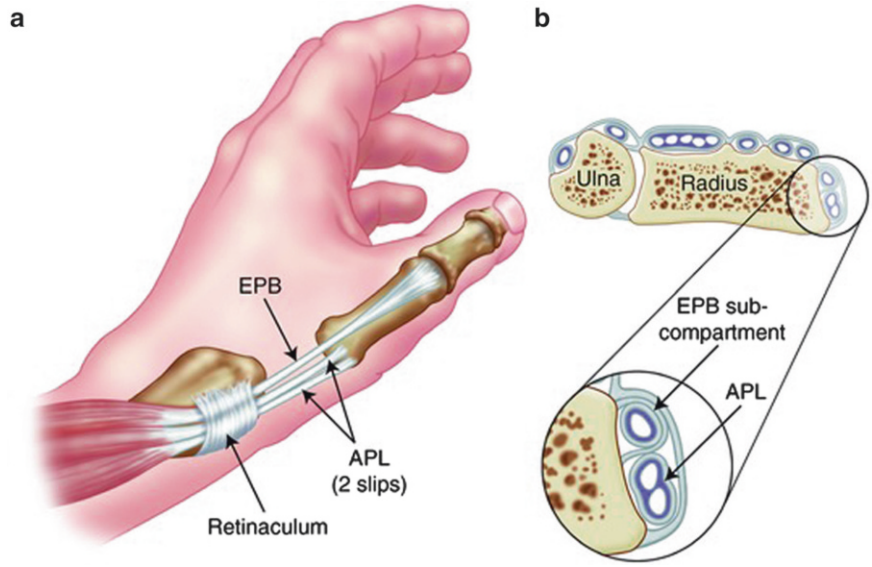

Fig. 14.4 De Quervain syndrome: (**a**) The first extensor compartment includes the abductor pollicis longus (APL) and extensor pollicis brevis (EPB) tendons. (**b**) The EPB tendon is often located within a separate subcompartment (With kind permission from Springer Science+Business Media: *Reoperative Hand Surgery*, Reoperative Tenosynovitis, 2012, Haase SC and Chung KC)

of the extensor retinaculum and synovial tendon sheath [63].

The extensor tendons to the fingers and wrist travel through six dorsal compartments of the wrist. The first (most radial) dorsal compartment contains the extensor pollicis brevis and the adductor pollicis longus. These tendons course through an osteofibrous canal to their insertion on the metacarpal and proximal phalanx of the thumb. A significant angulation is present as these tendons traverse over the radial styloid, placing the tendons at risk for repetitive injury [61, 63]. The function of these muscles is to position the thumb in extension and abduction in preparation for gripping and pinching. In these chronic states, inflammation is absent [59, 60]. The thickening results in a mechanical stenosis within the first dorsal compartment, causing impingement of the two tendons [63].

On physical examination, patients usually have tenderness with palpation over the fibro-osseous first dorsal compartment. Pain is commonly elicited with resisted thumb extension and abduction. A positive Finkelstein's test is pathognomonic for de Quervain's tenosynovitis [64]. This test is performed by flexing the thumb into the palm and making a fist around the thumb. The wrist is then *passively* deviated in the ulnar direction. Increased pain in the region of the radial styloid with this maneuver is considered positive. Pain increases with grasping, adduction of the thumb, or ulnar deviation of the wrist [65]. The symptom complex is usually gradual in onset, but traumatic etiologies have been described [59, 60].

De Quervain's tenosynovitis is a clinical diagnosis. Plain x-rays have not been found to be beneficial. Ultrasound, however, has been able to identify tendon pathology [66]. Other conditions with a similar presentation include peripheral neuritis, collagen vascular diseases, sprains of the CMC joint, arthritis of the CMC joint, fracture of the distal radius, ganglions of the wrist, acute calcific tendinosis, and aberrant CTS.

Non-pharmacological intervention, including education and environmental and ergonomic adaptation, is extremely important for treatment and prevention of de Quervain's and its recurrence. Interruption of highly repetitive activities that include pinching or gripping is

beneficial [61]. Immobilization of the thumb in a forearm-based thumb spica splint offers protection and rest. Heat modalities, stretching of the first dorsal compartment muscles, and ice may offer relief of symptoms during the acute stage. To date, there has not been an outcome study on the use of modalities and exercise for this condition.

Injection of local steroids has been shown to be of benefit [67]. Anderson and colleagues [68] reported that 81 % of individuals undergoing injections for this condition described symptom relief at 6 weeks. At 4-year follow-up, 58 % remained asymptomatic, and 33 % had complete reoccurrence. If conservative treatment is not effective, surgical release of the first dorsal compartment can be performed [69].

Dupuytren's Disease

Dupuytren's disease (DD) is a process of unknown etiology that leads to shortening and thickening of the palmar fascia and a flexion contracture of the digits (Fig. 14.5). Established risk factors include an autosomal dominant inheritance pattern [70, 71] and Caucasians of northern European origin, male, and older age [72, 73]. Smoking, high levels of alcohol intake, trauma, diabetes, epilepsy, and use of anticonvulsant drugs have all been implicated, with varying levels of evidence [74]. Theories of pathogenesis have included abnormal immune responses or tissue hypoxia secondary to the presence of oxygen-free radicals. The digital contracture is caused by myofibroblasts in the palmar fascia. The mainstay of treatment is surgical release or excision of the affected palmodigital tissue, but symptoms often recur. Nonsurgical correction of DD contractures can be achieved by anti-fibrotic substances and clostridium histolyticum collagenase injection, although the long-term safety and recurrence rate of this procedure requires further assessment [75, 76].

The contracture is a benign hypertrophy of the fascia. The first signs may be the palpation of almost imperceptible nodules in the area of the palmar crease, which progress to thick cords that form along the linear cord-like fascial lines of the palm [77]. The underlying tendons, synovial sheaths, and skin layers are not affected [78].

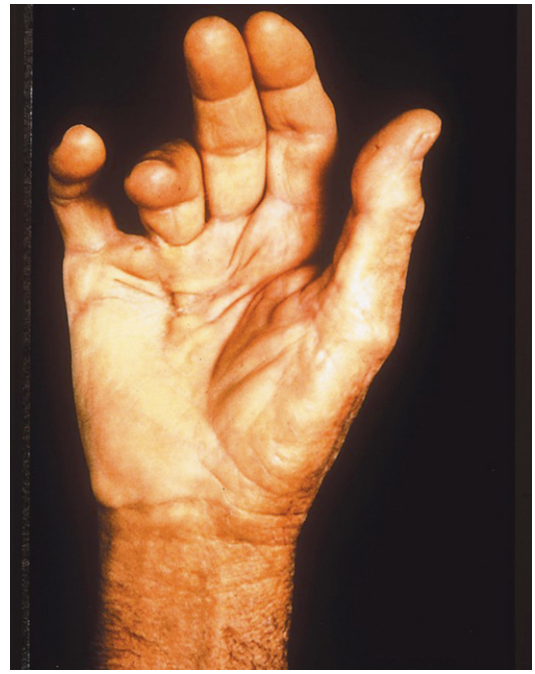

Fig. 14.5 Dupuytren's disease: Dupuytren's contracture involves the palmar fascia and can result in nodules in the hand and a fixed flexion contracture of any of the digits of the hand. As shown in this case involving the ring finger, the central cord proximal to the base of the metacarpophalangeal joint results in flexion contractures of both the metacarpophalangeal joint and the proximal interphalangeal joint

The pathophysiology of Dupuytren's is not fully understood. The palmar fascia thickening is caused by an abnormal proliferation of fibroblasts [74]. This proliferation is closely correlated with that observed in scar formation and healing. Three stages in the nodule and cord formation have been described. The first stage is proliferation. During this stage, the numbers of myofibroblasts within the palmar fascia spontaneously increase. The second stage is involution, when the myofibroblasts align along the tension lines of the palm and digits. The fascia enlarges owing to contraction of the myofibroblastic activity. In the third phase, the myofibroblasts resolve, leaving contracting collagen, which is perceived as nodules and matures into cords

[78, 79]. As the process progresses, these may become somewhat tender. The first finger to be affected, in 70 % of those with Dupuytren's, is usually the fifth digit. All digits, however, may be affected. Rheumatic diseases, synovitis, and Type 1 diabetes may be associated with similar symptoms [80].

Dupuytren's contracture is a clinical diagnosis made with the presence of palpable nodules and cords in the palmar fascia. It is often a diagnosis of exclusion. The anatomical distribution of the findings usually establishes the diagnosis. Joint deformity, including flexion contractures of the MCP, PIP, and DIP, is usually present in advanced conditions. Transverse or webspace contractures may also occur. These contractures can result in significant functional limitations necessitating treatment.

There has been minimal effectiveness of interventions, including splinting, radiation, vitamin E, anti-gout medications, physical therapy, and therapeutic ultrasound [75, 81]. Definitive treatment of advanced Dupuytren's is surgical fasciectomy. Advanced Dupuytren's is usually determined based on the performance of a "tabletop test" [82]. In this test, the individual places the palm on a flat surface and attempts to extend the involved finger actively. A positive test is noted if the MCP joint cannot be placed flat against the surface. This usually correlates with a greater than 30° fixed flexion contracture of the MCP joint. The goal of surgery is to restore function, not to cure the disease [83]. Despite surgical treatment, this condition can be quite recalcitrant, and reoccurrence rates range from 28 to 80 % [84].

Recently, there has been a great deal of interest in percutaneous or enzymatic fasciotomies as an alternative to surgical fasciectomy. Hurst [75] has demonstrated that by injecting collagenase into the fibrous cords, joint contractures can be improved. They report that 90 % enjoyed excellent results at an average of 9-month follow-up. Although no long-term studies have been completed, this procedure does offer promise. Additionally, an 8-year follow-up has recently been reported. While it consists of a relatively small sample size, a relatively high benefit and low risk over the long term was observed to prove long-term follow-up has been reported [85].

Postoperative surgical rehabilitation is extremely important following fascietcomy, with concentration on maintaining skin integrity, restoration of joint range of motion, and overall improvement of function [84].

Complex Regional Pain Syndrome

Reflex sympathetic dystrophy (RSD), causalgia (minor and major), algodystrophy, shoulder-hand syndrome, and Sudeck's atrophy are now considered complex region pain syndrome. The cause of CRPS is not fully understood. One theory, developed from an ischemia model in animals, suggests that symptoms are the result of microvascular injury leading to release of inflammatory cytokines [86]. Complex regional pain syndrome (CRPS) is a neuropathic pain syndrome characterized by autonomic dysfunction and severe pain that may lead to crippling contractures of the limbs. Mitchell first described CRPS during the American Civil War when he observed wounded veterans who had burning pain in an injured limb [87]. The term shoulder-hand syndrome described a variant of CRPS in which the entire upper limb was affected.

In 1993, at the meeting of the International Association for the Study of Pain (IASP), a task force proposed a unifying classification for these syndromes [88].

The task force of the IASP proposed two types of regional pain syndromes [89]:

Type 1, formerly known as reflex sympathetic dystrophy (RSD), Sudeck's atrophy, reflex neurovascular dystrophy (RND), or algoneurodystrophy, does not have demonstrable nerve lesions.

Type 2, formerly known as causalgia, has evidence of obvious nerve damage.

The two types share two features in common:

1. There is a history of edema, skin blood flow abnormality, or abnormal sweating in the region of the pain since the inciting event.
2. No other conditions can account for the degree of pain and dysfunction.

The diagnosis of Type 1 CRPS is based on four criteria:

1. The presence of an initiating noxious event or a cause of immobilization.
2. Continuing pain, allodynia (perception of pain from a nonpainful stimulus), or hyperalgesia (an exaggerated sense of pain) disproportionate to the inciting event.
3. Evidence at some time of edema, changes in skin blood flow, or abnormal sudomotor activity in the area of pain.
4. The diagnosis is excluded by the existence of any condition that would otherwise account for the degree of pain and dysfunction.

The diagnosis of Type 2 CRPS is based on three criteria:

1. The presence of continuing pain, allodynia, or hyperalgesia after a nerve injury, not necessarily limited to the distribution of the injured nerve.
2. Evidence at some time of edema, changes in skin blood flow, or abnormal sudomotor activity in the region of pain.
3. The diagnosis is excluded by the existence of any condition that would otherwise account for the degree of pain and dysfunction.

Patients who develop motor and/or trophic changes may complain of inability to initiate movement, weakness, tremor, or muscle spasms. Sometimes it is difficult to assess the function because of severe pain. Contractures can occur in late-stage disease.

The primary treatment for CRPS requires a combined approach using pharmacological and nonpharmacological agents. One approach has been to use an algorithm for guidance. Bisphosphonates have been studied in multiple controlled trials, based on theoretical benefit of relief of bone pain and bone resorption [90]. These have been only marginally successful. Many current rationales in treatment of CRPS (such as topical agents, antiepileptic drugs, tricyclic antidepressants, and opioids) are used because of their proven efficacy in other pain syndromes. Nerve blockade, sympathetic block, spinal cord and peripheral nerve stimulation, implantable spinal medication pumps, and chemical and surgical sympathectomy have also been reported, have been shown to provide some relief, but have not been demonstrated to be consistently therapeutic. The use of gabapentin and pregabalin has shown therapeutic benefit in controlling pain [91]. In treating CRPS, one follows the classic order of rehabilitation beginning with pain and edema control, followed by range of motion, and then strengthening followed by function. It is important to convey to the patient that immobilization is not an effective treatment for the pain and swelling; in fact, it may be instrumental in the pathogenesis and chronicity of the process [92].

Edema control entails elevation, decongestive massage, and various forms of compressive wrapping or garments. Pain control may be difficult using physical modalities alone. However, physical modalities should be the first line of defense. Contrast baths, Fluidotherapy, transcutaneous electrical nerve stimulation (TENS), and desensitization may be used before and after therapy session or exercise. If these are unsuccessful in adequately controlling the pain to the point at which therapy can be progressed, then one may consider further pain-relieving measures. Typical oral medications that may be used are tramadol, gabapentin, amitriptyline, and various α_1-blockers. In about half of all cases, further augmentation of analgesia may be attained by injections such as stellate ganglion blocks. One may also use injections such as intravenous regional blocks, axillary blocks, and cervical epidural injections. These blocks may provide temporary pain relief, enabling the patient to begin more aggressive hand therapy. Once pain is controlled to the level that patients can tolerate therapy, then one may begin exercises [92, 93].

The next goal of CRPS treatment is to restore normal range of motion. Often, the enduring disabilities resulting from CRPS are hand contractures. Gentle active or active-assisted range of motion should begin in a pain-free fashion. Any advancement in therapy should proceed slowly and carefully, keeping in mind that an overly aggressive approach may increase pain and swelling, which would be counterproductive.

When recognized early and treated carefully, CRPS generally runs its course in 6–12 months with complete or nearly complete recovery.

About 5 % of cases may turn into chronic CRPS with ongoing issues of pain, dysfunction, and disability. These patients may be on long-term pain medications or often are severely disabled by pain, contractures, or both. Reviews of current thinking about the pathophysiology and management or CRPS are available [92, 93].

Focal Hand Dystonia

Writer's cramp (Figs. 14.6 and 14.7) and musician's cramp (Fig. 14.8) are both focal dystonias that affect a discreet anatomical area of the hand. Focal hand dystonia is maladaptive response of the brain to repetitive performance of stereotyped hand movements. However, not all patients have a strict history of excessive hand use [94]. The focal hand dystonia is characterized by disabling cramps, contractions, or spasms during specific activities [95]. When not so engaged, the hand appears and functions normally. The flexors are more commonly involved than the extensors. Among the flexors, the flexor digitorum superficialis and profundus, the flexor pollicis longus, and the lumbricals may be involved. The extensor pollicis longus, extensor indicis, and digitorum communis may be involved among the extensors. Dystonia may occur sporadically in the population or may be genetically transmitted. The gene for early onset dystonia (DYT1) has been sequenced. Approximately 10 % of people with dystonia have a family history of tremor or dystonia [96]. Others report that a higher percentage of those affected have a family history of dystonia [97].

The pathophysiology of dystonia seems to be a loss of inhibitory function. The anatomical locus has been demonstrated at spinal, brainstem, and cortical levels. There seems to be some mild sensory and sensorimotor deficits. The abnormality leads to unwanted muscle spasms. Increasing inhibition may be therapeutic [98].

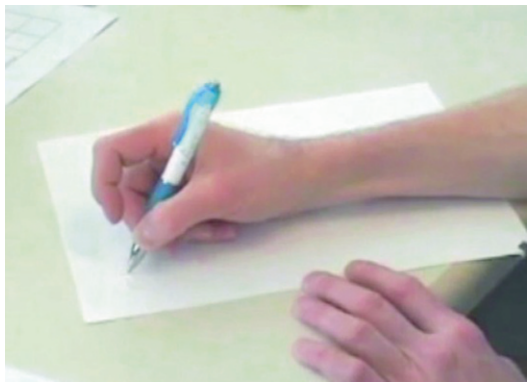

Fig. 14.6 Writer's cramp: The patient exhibits involuntary extension at the metacarpophalangeal joint of the index finger while writing (With kind permission from Springer Science+Business Media: *Movement Disorders*, Writer's Cramp, 2012, Bhidayasiri R and Tarsy D)

Fig. 14.7 Writer's cramp mirror movements (With kind permission from Springer Science+Business Media: *Movement Disorders*, Writer's Cramp, 2012, Bhidayasiri R and Tarsy D)

Fig. 14.8 Musician's cramp: Musician's cramp, analogous to writer's cramp, is a focal dystonia of the arm induced with the action of playing a musical instrument. This patient has a pianist's cramp that is manifested when she attempts to perform piano-playing movements on top of the desk (With kind permission from Springer Science+Business Media: *Atlas of Clinical Neurology*, Movement Disorders, 2009, Fahn S, Greene PE, Ford B, Bressman SB and Frucht SJ)

The incidence of writer's cramp is reported to be 2.7 per million in Rochester, MN [99]. It tends to affect male young adults. It is usually idiopathic and not a result of overt trauma, although it may follow a traumatic episode. Patients frequently have mirror dystonia, demonstrated by inducing the writer's cramp in the dominant hand even when attempting to write with the nondominant [96]. Focal dystonias tend to remain focal and do not become generalized dystonias over time.

The pathophysiology of dystonia is not entirely understood. However, there seems to be some evidence for abnormalities in the basal ganglia [95] or problems with cortical organization [100]. Electrodiagnostic studies show a co-contraction of muscle and a loss of alternation of agonist/antagonist muscle contractions. There are prolonged bursts of muscle contractions and overflow contraction seen in those muscles not activated by the motor task [101].

The use of botulinum toxin for focal dystonia has been demonstrated to be effective and safe even for chronic application [102].

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