Chapter 10 - Wrist and Hand Pain
Of all the soft tissue injuries the hand presents the greatest disability when impaired.

Pain is a frequent symptom, as is loss of function. Numerous tissues in the wrist and hand are subject to painful impairment and disability. Accurate diagnosis demands a knowledge of functional anatomy, a meaningful history, and a precise examination.

The hand is an organ of grasp as well as one of fine movement, with exquisite sensation and delicate discrimination. A large portion of the brain controls the function of the hand, indicating its ultimate development and training.

Restoration of function is the ultimate goal of treatment and is based on understanding the neuromusculoskeletal nature of the hand. Early care is paramount as prolonged immobilization is detrimental and reconstructive surgery still limited.

Loss of dexterity or impairment causes significant problems with activities of daily living and many occupations.

CARPAL TUNNEL SYNDROME
The carpal tunnel is a narrow fibro-osseous opening through which traverse six structures: the flexor pollicis longus tendon, four flexor digitorum profundus tendons, and the median nerve (fig. 10-1).

The distal volar skin crease of the wrist (fig. 10-2) is the proximal border of the canal.

The tunnel itself extends approximately 3 centimeters.

The roof of the carpal tunnel is the transverse carpal ligament, which comprises two bands: one from the hook of the hamate bone extending to the tubercle of the trapezium and another, more proximal, that extends from the tubercle of the navicular (scaphoid) bone to the pisiform bone (fig. 10-3).

The floor of the tunnel is occupied by the carpal bones of the hand.

Carpal tunnel syndrome (CTS) occurs when the median nerve is compressed within these anatomic structures.

The syndrome can be divided into three categories: (1) increased volume of the contents of the tunnel; (2) enlargement of the median nerve (rarely occurs); and (3) decreased cross-sectional area within the tunnel.

The increased contents are considered to cause tendinitis and tenosynovitis in most symptomatic patients.

Enlargement of the nerve is rare and decreased cross sectional area in the tunnel, without rheumatoid arthritis, is also infrequently seen.

Surgical intervention in patients with CTS has revealed thickened and edematous synovial sheaths of the enclosed tendons.
Most people have a minor tenosynovitis of the tendons within the tunnel but to a lesser degree than people with symptomatic CTS!

The biomechanics of the carpal tunnel tendons (flexor digitorum profundus, flexor digitorum superficialis, and the flexor pollicis longus) is that they move in the manner of a belt around a pulley with the transverse carpal ligaments acting as the fulcrum. Flexion and extension of the wrist causes these tendons to be displaced against or past the ligaments (Fig. 10-4).

The power grip or repeated finger flexion with wrist stabilization, which is so common in many workplace activities, involves simultaneous wrist extension and active finger flexor contraction. This action increases load on the flexor tendons; this leads to degenerative, and possibly inflammatory, changes in the tendons. Normally the coefficient of friction is minimal, but with increased muscular action and repetitive motions, friction undoubtedly increases.

The transverse carpal ligament is an important component of the digital flexor pulley system. Its action in the tunnel causes those problems that lead to the syndrome.

Its removal or modification in the treatment of the syndrome adds undesirable, as well as remedial, factors. It also plays a large causative role in the syndrome, which must be considered in prevention, and cure.

The flexor tendon pulley assures maximal flexion force of the joints with minimal excursion of the tendons.

Both definite flexion weakness and an inability to touch the fingers to the palm with the wrist fully flexed are present.

Flexion decreases with wrist flexion at 20° and is fully lost with the wrist flexed to 40°.

Maximum muscle strength occurs at resting length.

Passive stretching of 40% loses strength.

The closer to the center of rotation of a joint the tendon passes the less distance that tendon must traverse but with attendant loss of mechanical efficiency. This is the rule of the moment arm.

The greater the moment arm, the greater its mechanical efficiency, but also the longer the excursion is.

The flexor pulley system at the transverse carpal ligament minimizes the moment arm's decreasing the extent of tendon excursion, which in turn allows the muscle to remain closer to its resting stage.

**Pathophysiology of Nerve Compression**

Low-grade peripheral nerve compression reduces epineural blood flow (Fig. 10-6). Axonal transport is impaired (Fig. 10-7). With segmental axonal compression (see Figs. 1-9 and 1-10), the endoneural (Fig. 10-8) fluid pressure is increased with resultant paresthesia.
Clinically, patients with early CTS experience numbness and tingling in the distribution of the median nerve (fig. 10-9).

Early abnormalities are noted in vibration tests and in the Semmes-Weinstein monofilament testing (fig. 10-10).

Experimentally, pressure changes have been determined within the carpal tunnel depending on wrist positions (Figs. 10-11 and 10-12).

Applying pressure of 50 mm Hg for two hours causes endoneural edema. Clinically, median nerve compression of 60 mm Hg can cause complete sensory conduction block.18 Pressure changes resulting in median nerve ischemia have been postulated as the major cause of carpal tunnel syndrome, which has led to specific therapeutic approaches.

Of the three postulated causes of CTS, this increased volume of the contents of the carpal tunnel19 has the greatest number of proponents, but what increases the contents remains unclear.

Tenosynovitis brought about by repetitive trauma is most often implicated.

Venous congestion resulting from hyptonia during sleep was considered a likely precursor to anoxic capillary endothelial damage and edema in CTS.

Tenosynovitis in itself is not a major factor in CTS, but edema is.

Repetitive tendon excursions from sustained and possibly improper muscular tension are important factors.

Because the pulley mechanism of the transverse carpal ligament is a physiologic mechanism and so not a major cause in CTS, therefore the attempts to remove or elongate the tendon surgically to increase the volume of the carpal tunnel should be used only as a last resort.

Carpal tunnel release immediately reduces the pressure within the carpal tunnel.

Knowing that various positions of the wrist change the intracarpal tunnel pressures must be addressed in initiating a treatment protocol.

The long-range effects of high pressure on a nerve is well known, but the effects of lower pressure, as measured in CTS, suggest the disturbance of function is rapidly reversible and indicate that vascular insufficiency, rather that morphologic changes, occur in the nerve as a result of this amount of pressure.

Long-term pressure impairs microvascular flow with gradual leakage of proteins through the walls of the venules causing epineural edema: the primum mobile in CTS (fig. 10-13).

**Treatment**
Early intervention after diagnosis of CTS is preferred with gratifying results and avoidance of surgery.
CTS develops over a period of months; it is initiated by edema, which is reversible. This edema likely results from mechanical tendon friction from repetitive mechanical activities in which wrist motions coupled with certain finger flexor activities are the major type.

Friction over the transverse carpal ligament increases and thereby causes inflammation with resultant edema of the median nerve!

After symptoms occur (paresthesia, numbness, and weakness), significant edema can be assumed to be present. If early signs are noted or if the patient is occupationally susceptible early testing of median nerve conduction time is indicated.

The early signs are:
- A positive Tinel sign which means that gentle tapping of the median nerve at the wrist elicits tingling in the distribution of the median nerve. Three taps are considered positive if tingling results from each tap.
- A positive Phalen test, which means making a forceful flexion of the patient's wrist and holding it there for 1 minute can reproduce the paresthesias. Phalen felt no need for electrodiagnostic testing if results of both the Tinel and Phalen tests were positive. Careful studies have refuted the value of a Tinel sign but continue to accept the Phalen test.

Weakness of the abductor pollicis brevis and change in sensory testing by light palpation, vibration test or Semmes-Weinstein filament test are also indicators.

Nerve conduction is diagnostic and electromyographic (EMG) testing is necessary in the presence of significant motor weakness.

Occupational factors that need concern the examiner are:
- Repetitive activities
- Computer keyboard activities
- Grip force
- Pinch force
- Wrist flexion
- Palm pressure
- Vibration
- Glove use
- Exposure to cold
- Intensity of work conditions

After the occupational situation and early delayed conduction times are confirmed, the following measures appear to be indicated:
1. Night splinting and splinting during working hours (Fig. 10-14)
2. Prework exercises
   - Flexibility
   - Strengthening (Fig. 10-15)
3. Job technique modification
   - Dynamic studies on repetitive passive flexion-extension motions pumps up carpal tunnel pressure in patients with CTS and remains higher than normal even after rest. Active flexion-extension has additional effect. Flexion-extension isometric
or isotonic exercises of the forearm muscles increase carpal tunnel pressure, but because forearm muscles do not extend into the wrist, the reason for increased carpal tunnel pressure is not clear. Certain advocates suggest active flexion-extension exercises to provoke symptoms of CTS. These factors cast doubt on the advocacy of forearm exercises in the treatment of CTS even though they have proven effective. Exercises may mobilize the tendons within the tunnels, but this remains conjectural. Rest is apparently effective as is immobilization of the wrist during activities of daily living.

4. Local steroid injections. A provocative paper has asserted a relationship between cervical pressure and CTS. Hellenbrandt, Houtz, Partridge, and Waltos demonstrated that repetitive, fatiguing upper extremity movement exerted reciprocal reflex activities in the neck muscles. Changing neck and head postures affects the limb muscle activity and vice versa. Neck muscle fatigue significantly affects limb muscle activity. Increased load on the tendons from increased forearm muscle tonus, resulting from cervical muscle asymmetry, increases the inflammatory response that leads to CTS.

The protocol is to evaluate muscle imbalance and asymmetry and then to strengthen the weaker side. Asymmetric weakness has been found in the sternocleidomastoid and neck extensors. After strengthening exercises, frequent rotation to one side with sustained contraction is followed by a return to midline. This procedure should be implemented in daily working activities.

CTS is a cumulative trauma disorder (CTD) or a repetitive strain injury (RSI) or both.

The incidence of CTS has increased over the decades as more workers perform fine manual motor activities for increasingly longer periods.

Awkward hand and wrist positions and repetitive hand-finger activities are prominent in CTDs.

Robbins postulated that extreme flexion-extension of the wrist reduced the volume of the carpal tunnel by compressing the median nerve. Smith, Sontegard, and Armstrong replaced the median nerve with a water-filled cylindrical balloon and found that pressure on the nerve (balloon) was increased when the wrist was flexed to an extreme angle and when the flexor tendons were tensed at various wrist flexion angles.

Biologic changes occur in the flexor tendons as they pass under the carpal tunnel ligament with resultant hyperplasia and increased density from repeated exertions of the wrist, hand, and fingers. Repeated flexion-extension movements also increase the shear traction forces of the tendons from their pulley action at the transverse ligament.

Flexion-extension acceleration studies that did not require the use of hand tools were found to be prominent in CTDs and were supported by anatomic, physiologic, and biomechanical modeling studies.

Compressive forces are also exerted by forearm muscles that generate forces for repetitive contraction. These muscles also exert torque forces and co-contraction of the muscles to stabilize the wrist while the fingers are flexing and extending thus increasing the compressive forces within the carpal tunnel.
CTDs result, therefore, from repetitive flexion-extension activities with concurrent static wrist stabilizing forces (Fig. 10-19). Static muscle tension from inappropriate muscular activity and prolonged isometric contraction of the stabilizing muscles with co-contraction of the kinetic muscles performing the dexterity action has been asserted but, as yet, has not been fully confirmed.

Prolonged static contraction also probably impairs the agonist-antagonist reflex action with the kinetic muscles causing the agonists to function against the action of unreleasing antagonist muscles. This becomes cocontraction, rather than reciprocal agonist-antagonist action. Resultant improper tendon action is manifested at the carpal tunnel, producing CTS. This is being studied using intricate EMG testing, which may lead to precise pathonomonics of CTS. Other involved factors are evolving, such as posture, stress, and individual psychosocial considerations.

Some mechanisms trigger the pathophysiology of CTS in certain particularly susceptible individuals. One recent concept is the double-crush theory in which proximal nerve compressive syndromes cause or predispose to distal nerve entrapment. Such proximal factors include brachial plexus compression from scalene pressure (see Chap. 7) (Fig. 10-20).

The crush syndrome is one wherein neural compression allegedly alters the axoplasmic neural transport system. The axoplasmic transport theory is illustrated in Figure 10-21.

The crush occurs on a myelinated nerve with displacement of the nodes of Ranvier (Figs. 10-22 and 10-23). The resultant changes in the axonal transmission distally predispose to further impairment from a potential distal crush.

Why the proximal muscles cause the proximal aspect of the double-crush syndrome is being explored. These muscles fatigue and fail to recover in an appropriate time. Biomechanical stress of these muscles on contiguous nerves lead to entrapment. The fibers that fatigue first are the slow-twitch fibers which, if not periodically rested, shut off. Areas of local ischemia that develop become sites of fibromyalgia (see Chap. 3).

Stages of dysfunction follow with the proximal muscles initiating the process. The muscles involved are the trapezius muscles that support the scapula and normally maintain the supraclavicular space through which the plexus emerges.

With a postural forward head position necessary for the task, a scalene syndrome results with neurovascular compression as the scalene muscles assume the function of other scapular muscles. These muscles cause a proximal neurovascular compression that ultimately results in neurovascular distal compression within the carpal tunnel.

The initial symptoms are numbness and tingling with no discernible edema or delayed nerve conduction. The initial microscopic edema is in the forearm compartment. The intrinsic muscles of the hand undertake the function of the proximal forearm (flexor) muscles.

The slow-twitch muscles that are fatigued become unable to sustain prolonged activities. This is initially in the capsular, and then possibly the cervical, muscles.

Treatment protocols of incipient CTS treat the proximal component early. Work-rest cycles are mandatory and must be frequent.

Strengthening of scapular muscles found to be weak must be undertaken.
Treatment of the distal aspect of the syndrome must be initiated as well, but this therapy will fail if the proximal component is ignored.

**PRONATOR TERES - MEDIAN NERVE COMPRESSION**
The median nerve leaves the cubital fossa passing between the heads of the pronator teres muscle then passes under the tendinous edge of the flexor digitorum sublimis muscle (fig. 10-24).

The course of the median nerve through the pronator teres muscle varies between the heads (56%), behind the heads (11%), through the humeral head (3%), and through the ulnar head (2%).

In some patients, the ulnar head of the pronator teres is missing.

After leaving the pronator teres, the median nerve gives rise to the anterior interosseous nerve.

After traversing the teres muscle, the median nerve divides under the tendinous arch of the flexor digitorum sublimis and into the layer between the flexor digitorum sublimis and the flexor digitorum profundus.

The median nerve runs between the flexor digitorum sublimis and the flexor digitorum profundus, ultimately between the flexor carpi radialis and the palmaris longus to reach the carpal tunnel.

In the forearm, the median nerve supplies the pronator teres, flexor carpi radialis, palmaris longus, and digitorum superficialis.

Just distal to the pronator muscle, the median nerve sends branches to the ulnar half of the flexor digitorum profundus, flexor pollicis longus, and pronator quadratus.

The pronator teres pronates the forearm with the elbow preventing rotation by elimination of pronation from the brachioradialis and the long flexors of the forearm.

*The numerous causes of nerve compression vary from direct trauma, to static compression from a fibrous band and to prolonged external compression as seen in honeymoon paralysis.*

**Symptoms and Signs**
Subjective symptoms and objective signs of median nerve involvement found in that upper extremity are similar to CTS.

The pronator teres syndrome, however, involves not only the muscles of the thenar eminence but also the flexors of the wrist and the finger flexors.

The patient’s complaints are related to thumb, index finger, and middle finger flexion.

Sensory disturbances involve the volar and dorsal surfaces of the hand, palm, and several fingers.

A stress test that suggests pronator teres compression is the evoking of pain and paresthesia by resisted pronation of the forearm with the elbow extended.
Further aggravation of the symptoms occurs by resistance of flexion of the elbow and simultaneous supination of the forearm, implicating the lacertus fibrosus.

Resistance of flexion of the proximal interphalangeal joint of the middle finger implicates the FDS muscle belly.

A positive Tinel sign can be elicited by tapping or exerting direct pressure in the region of the two heads of the pronator teres muscle below the cubital space.

Use of EMG studies can assist by demonstrating abnormalities and delayed conduction velocity of the flexor carpi radialis, flexor digitorum sublimis, and flexor palmaris longus.

*In contrast to carpal tunnel median nerve compression, which usually involves the muscles of the thenar eminence, compression at the pronator teres site involves not only the thenar muscles but also those of the wrist and finger flexors.*

*Because the median nerve gives off a palmar branch before entering the carpal tunnel, a sensory deficit of the palm implies median nerve compression proximal to the carpal tunnel.*

**Treatment of the PTS**
Most pronator teres syndromes are mild and self-limited, thus merely reducing provocative movement of the forearm, wrist, and finger flexors for several weeks will be beneficial.

This process can be assisted by splinting the forearm in a neutral position between pronation and supination.

Local injections of steroids into the region of compression is also valuable.

Persistent symptoms and objective evidence of insidious paresis and anesthesia justify surgical decompression, which will allow identification of the involved structures, release of the origin of the humeral head of the pronator, and even neurolysis of the median nerve, if indicated.

**ANTERIOR INTEROSSEOUS SYNDROME**
The motor branch of the median nerve, the anterior interosseous nerve, in the cubital region can undergo compression, impairing function of the distal phalanx of the thumb and index finger.

The anterior interosseous nerve originates from the division of the median nerve progressing under the deep fascial layer of the flexor digitorum superficialis running along the interosseous membrane. It ultimately innervates the flexor pollicis longus and the flexor digitorum profundus to the second finger.

Many variations occur in the course of this nerve, which has prompted authors to consider this syndrome as being identical to the pronator teres syndrome.

*Clinically the patient develops an inability to pinch between the thumb and index finger because of paresis of the distal phalanges of both fingers.*

Pinching is then attempted with both joints in extended position.
In addition, the patient is unable to clench the fist or write (fig. 10-25).

Treatment consists of immobilization and avoidance of causative factors determined, but with persistent symptoms and EMG confirmation, surgical decompression is indicated.

**ULNAR NERVE COMPRESSION**

The ulnar nerve is subjected to compression at numerous sites along its course, causing characteristic sensory and motor symptoms (fig. 10-26).

At the wrist the ulnar nerve enters the hand in a shallow trough between the pisiform bone and the hook of the hamate bone (Guyon's canal) (fig.10-27).

The floor of this tunnel is a thin layer of ligament and muscle.

Its roof is composed of the volar carpal ligament and the palmaris longus muscle (fig. 10-28).

Proximal to its entry the nerve divides into dorsal and palmar branches that further divide into superficial and deep palmar branches that run into the tunnel.

Only these two terminal branches run into the tunnel, therefore *compression from tunnel entrapment spares the dorsal branch*.

The superficial branch of the palmar branch innervates the palmaris brevis muscle, the palmar skin of the fifth finger, and the ulnar skin of the fourth finger. The deep branch innervates the hypothenar muscles, the two lateral lumbricals, all the interosseus muscles, the adductor pollicis, and the deep head of the flexor pollicis brevis muscles.

Compression of the superficial branch causes motor and sensory symptoms (fig. 10-29) whereas compression of the deep branch causes only motor symptoms (figs. 10-30 and 10-31).

Numerous etiologies are proposed to explain ulnar nerve compression at Guyon's canal, but most are traumatic, such as bicycle or motorcycle riding, operating a pneumatic drill, among others.

Symptoms may describe difficulty in hand grasping activities and paresthesia of the dermatomal regions of the ulnar nerve.

Motor weakness may be described as clumsiness in performing fine movements; reduced pinch strength of the thumb is also apparent.

Atrophy of the interossei gradually becomes evident with deepening of the interosseus grooves on the dorsum of the hand.

A positive Tinel sign over the ulnar nerve may be elicited.

Confirmation using EMG is diagnostic.

Treatment should be conservative avoiding activities found to be responsible, and therapy with oral anti-inflammatory medication, steroid injections, and splinting.
Without significant relief, waiting longer than 6 months is not advocated.

If both Guyon's syndrome and CTS are present surgical release of the median nerve should also be contemplated.

**CUBITAL TUNNEL SYNDROME**
Compression of the ulnar nerve at the elbow is commonly called cubital tunnel syndrome, which is considered the second most common nerve entrapment of the upper extremity, second only to CTS.

It is becoming increasingly more common due to the increasing use of computers whose operators repetitively use the upper extremity held in an elbow-flexed position and put direct pressure on the nerve at the cubital tunnel (fig.10-32).

The ulnar nerve is very superficial at this site.

At the elbow the ulnar nerve enters the cubital groove on the posterior aspect of the medial epicondyle with its roof being formed from an aponeurotic band at an angle transverse to the nerve.

As the nerve leaves the tunnel, it passes between the two heads of the flexor carpi ulnaris muscle.

The aponeurotic band stabilizes the ulnar nerve behind the medial epicondyle, thus preventing subluxation of the nerve during elbow actions.

Hypermobility of the nerve exists, however, making compression outside the canal more possible.

The retinaculum is lax in elbow extension and taut only in full flexion.

With flexion the volume of the cubital tunnel decreases and causes greater pressure upon the nerve.

Cubital pressure also increases with the wrist extended and with the shoulder forward, flexed, and abducted, which replicates many postures used in the workplace.

Within the tunnel, the motor fibers supplying the intrinsics of the hand are superficial, whereas those supplying the flexor carpi ulnaris and the digitorum profundus are deeper. The superficial location of the sensory nerves also explains the impaired sensation found early in this process.

More proximally to the cubital tunnel, the ulnar nerve passes under the *arcade of Struthers*, which is a thick fascial band running from the medial head of the triceps muscle to the medial intermuscular septum.

This septum may also be a site of entrapment.

Because symptoms from entrapment at the site are often delayed, the term *tardy ulnar palsy* has evolved.

Surgical findings of this syndrome revealed a "markedly swollen and adherent ulnar nerve within the groove with no significant elbow deformity."
All patients who had operations to release the arch 'without nerve transposition' had relief of symptoms.

**Symptoms of Cubital Tunnel Ulnar Nerve Compression**
Aching pain on the medial site of the elbow near the medial epicondyle is frequently associated with shooting pains in the ulnar aspect of the hand and little finger.

This hypasthesia is activity related and is provoked by elbow flexion and occasionally relieved from elbow extension.

*Unlike the carpal tunnel syndrome, the paresthesiae are not nocturnal.*

Clumsiness of hand activities may be an associated complaint depending on the degree of motor involvement.

A positive Tinel sign may be elicited.

The sensory modality tests (fight palpation, vibration, Semmes-Weinstein monofilament, and two-point discrimination) are diagnostic of nerve involvement.

*A provocative test is made by flexing the elbow fully and extending the wrist for 3 minutes.*

Motor evaluation begins with weakness of the first dorsal interosseous (resisting index finger abduction) and abductor digiti minimi (testing little finger abduction).

Ultimately the presence of atrophy in the hypothenar region and the first web with clawing of the ring finger and little finger becomes apparent.

A positive Froment sign (pinching a piece of paper between thumb and side of the index finger) is apparent.

In this sign the distal thumb joint is flexed.

The major muscles to test are the flexor carpi ulnaris (testing wrist flexion in an ulnar direction) and the ulnar portion of the flexor digitorum profundus (testing flexion of the distal interphalangeal joint).

The normality of the bony aspect of the tunnel can be evaluated radiologically by special views.

**Treatment**
Prevention is obviously desirable, that is, avoidance of direct pressure on the flexed elbow.

Moderate flexion with a cushioning is desirable if possible.

Sleep position-induced compression must be identified and modified using a small pillow to maintain elbow extension and to avoid flexion.

Modification of vocational activities must also be addressed.
Frequent and forceful elbow flexion may also be incriminated and when identified, must be eliminated.

Oral anti-inflammatory nonsteroidal drugs (NSAIDs) have value; perineural steroid injection has also been proposed.

Disadvantages with the latter include subcutaneous atrophy and cosmetic skin depigmentation.

A physician attempting an injection in the cubital tunnel must warn the patient of these possibilities.

**Surgical Intervention**
Procedures vary from simple decompression to anterior transposition and medial epicondylectomy.

**TENDON PROBLEMS**

**Common Tendinitis**
Tendinitis implies inflammation that causes abnormal function of a tendon with impairment and often pain.

The gross and histopathologic changes in these tissues are nonspecific. They include fibrocytic proliferation, thickening, destruction of synovial tissue, and often adhesion to adjacent soft tissues. Tendinitis implies inflammation that causes abnormal function of a tendon with impairment and often pain.

Tendons normally connect muscle to bone for extremity articular function; muscle tonus generally determines the tension within the tendon fibers. The collagen fibers (Fig. 10-33) that form the tendons normally have a curled configuration.

Muscle tone is determined by intrinsic neurologic mechanisms in which the spindle system feedback, in conjunction with Golgi apparatus feedback within the tendon itself. The spindle system determines the rate of elongation, whereas the Golgi apparatus determines the strength (i.e., force) of the contraction.

The extrafusal muscles contract concentrically and eccentrically, thus causing the tendon fibers also to undergo commensurate tension and elongation. Deceleration of a musculotendinous unit occurs frequently in daily neuromuscular actions and places even greater Newtonian forces than concentric forces—a fact frequently overlooked in the clinical setting.

Relaxation after a muscular contraction allows the tendon fibrils to recoil. If the contraction is excessive or prolonged without an interim test period, the fibrils undergo excessive elongation with some structural internal disruption.

Tendons have a mechanical tendency to elongate until there is a specific degree of rupture.

Rupture is a break in the peptide chain after a nonphysiologic force of elongation.
These forces are plotted in a stress and strain curve in which stress is the amount of load per unit of cross-sectional area and strain is the resultant proportional elongation resulting.

It has been postulated that different regions of the tendon react to elongation forces within the stress-strain curve:

- **Toe region**—physiologic loading occurs for 1 hour
- **Linear region**—stress increases rapidly causing prolonged elongation. Microfailure results.
- **Progressive failure region**—the tendon remains intact to the naked eye but microscopically will demonstrate significant failure.
- **Major failure region**—the tendon still remains intact, but frank ruptures exist.
- **Complete rupture region**—a gross tendon disruption.

These regions are arbitrary, microscopic, and their true value lies in recovery, that is, the return to their original length.

The ideal spring elongation depends on the speed of stretch with the tension remaining constant at any given length.

Collagen does not perform as an ideal spring.

A tendon gradually lengthens from a constant or repeated load. This slow elongation is termed **creep**—it is transient if the force is within physiologic limits.

The viscoelastic properties of collagen vary with temperature. Temperature below 37°C can be considered physiologic with good chances for recovery, whereas temperatures between 37° and 40°C increase creep and temperatures above 40°C enhance permanent damage by melting the bonds between tropocollagen molecules.

These factors have clinical significance, because damage and recovery depend on stress and ambient temperature.

There are some non-traumatic factors that affect tendons. Immobilization cause significant loss of strength. The dense connective tissue of muscle loses 80% of its strength within 4 weeks, 50% in ligaments after 8 weeks. Length and flexibility diminish rather than strength.

In their daily functions, tendons are subjected to tensile forces, compressive stresses, and shear stresses.

Microfailure occurs if the tendon load is excessive or if the rest between loads is inadequate to allow the tendon fibrils to recover their resting length. Repetitive loading stress accelerates the micro-trauma by exceeding the reparative capacity of the tendon.

Pathologic changes within the tendon include swelling and thickening of the sheaths that cause vascular impairment within the mesotendon and failure of normal diffusion of nutritive elements.

Tendons glide upon their contiguous tissues.

This action is facilitated by the thin fibrous and cellular layers of the tendon termed **epitenon**, which adheres to tendon surfaces.
The tendons contained within sheaths contain synovial fluid for lubrication of the tendon within the sheath. The sheath itself has external lubricant against peritendinous tissues.

Systemic factors affect the viability and integrity of tendons such as rheumatoid arthritis, hypothyroid, diabetes mellitus, gout, calcium pyrophosphate, collagen vascular disease, and infections.

Occupational stress factors frequently cause tendinitis. These CTS include repetitious tasks requiring forceful motions, unusual postures, exposure to temperature extremes, and exposure to vibration. These factors were recognized as early as 1717.

Diagnosis reveals swelling, tenderness, warmth, crepitus, snapping, and occasional numbness.

Direct identification of the involved tendon is mandated; placing that specific tendon under stress (resisted movement) or passive elongation reveals the precise tendon.

Every tendon in the hand is a potential site of tendinitis.

Most such sites can be identified by evaluating the specific functional impairment of that precise tendon. Generalities of tendon injuries apply to the more specific sites.

**Tendon Rupture**
A tendon may be ruptured by an acute stretch injury when the fibrils are elongated beyond the limit of their physiologic recoil.

The tendon is normally strongest at its musculotendinous link where it seldom tears. Tearing occurs at its insertion site with and without avulsion of the bone or periosteum.

A tendon damaged by a systemic disease or a tendon which has sustained numerous microtraumata will tear more frequently from a lesser stress.

**Surgical Intervention**
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**Extensor Tendons**
Injury to extensor tendons of the fingers and thumb is common because they are superficially prominent and have little overlying skin and subcutaneous tissue to protect them.

They are also subject to direct injury when the hand and other fingers are extended.
Extensor tendons tend not to retract after being severed and thus can be sutured soon after injury, when they will require 3 to 4 weeks before they regain integrity.

Tendons contained within a sheath when severed show greater deterioration and heal more slowly than those not contained within a sheath.

The reason is that the swelling of the injury tendon within the sheath obstructs venous and lymphatic return, which impairs tendon nutrition and subsequent healing. Adhesions may form about the tendon which may also impair nutrition and ultimate function.

Sutured extensor tendons usually result in good function despite postoperative immobilization.

**Flexor Tendon Injury**

Suturing a flexor tendon in "no man's land" (fig. 10-37) has an unfavorable prognosis. The anatomic structure and relationship of the tendons in the area indicate the reason for this poor prognosis.

A sutured tendon usually swells—in this area of the fingers no room is available for expansion of the tendon. Ischemic necrosis can result.

Tendons severed distal to no man's land, for example, profundus tendons, may be primarily sutured.

Severed flexor tendons proximal to no man's land, especially at the wrist level, can be primarily sutured with good functional results.

**Specific Tendinitis Problems of the Hand**

**Extensor Tendon Tears**

**Mallet Finger**

Tear of the extensor tendon from its attachment on the distal phalanx (fig. 10-38) usually occurs during an acute flexion injury to the finger, that is, when the extensor tendon is taut.

Most patients have the tendon torn at its point of insertion on the phalanx, although 25% also sustain a bone avulsion from the injury.

Conservative treatment includes immobilization of the distal phalanx in hyperextension (fig. 10-39) for 5 weeks; this usually results in functional recovery.

If contracture of the flexors has occurred, passive stretch may be needed, as well as gentle active extensor exercises.

Surgical intervention is indicated when functional restoration has not been achieved or is not accepted by the patient.

**Extensor Tendon Rupture at the Middle Phalanx**

Rupture of the insertion of the extensor tendon into the middle phalanx, with or without bony avulsions, may be caused by a direct blow or by a crushing injury.
Rupture of the extensor tendon at that site causes the proximal phalanx to extend and the middle phalanx to flex (fig. 10-42).

This deformity is termed *boutonniere deformity*; it may result from rheumatoid disease or trauma.

Because of inadequate extensor slip function, the lateral bands dislocate to the flexor side of the joint fulcrum, thus causing a flexion deformity of the proximal interphalangeal joint and hyperextension of the distal joint.

Treatment of this deformity is difficult because a functional splint (fig. 10-43) is often ineffective and a rigid splint may maintain the manually corrected deformity (fig. 10-44).

**Extensor Pollicis Longus Rupture**
The extensor pollicis longus tendon curve around the dorsal radial tubercle of Lister (Fig. 10-45) passes over the radial wrist extensors and continues to the thumb.

At the point where the tendon angulates wear and tear can occur. Rupture of this tendon results in an inability to extend the distal joint of the thumb and weakness extending to the proximal joint.

Normally the tendon can be palpated when the wrist is actively extended and the thumb adducted.

After primary rupture, suturing the two fragmented ends of the tendon is not possible, because such repairs neither hold nor function.

Repair requires a graft from a site extending proximal to the dorsal retinaculum to the end located at the metacarpal.

Repair may necessitate the transfer of the tendon of the extensor indicis.

Once grafted the thumb must be splinted for at least 1 month before use.

**Tendinitis of the Extensor Compartments**

**De Quervain's Disease**
Tenosynovitis of the thumb abductors at their radiostyloid process and subsequent stenosis is very common.

The condition was named after the Swiss surgeon de Quervain.

The first dorsal compartment contains the abductor pollicis longus and the extensor pollicis brevis confined by the radial styloid and covered by a synovially lined ligament 1.5 inches long.

Symptoms include edema and pain over the radial styloid with the pain initiated and aggravated by forcing the wrist into an ulnar-deviated position and the thumb flexed and adducted: it is known as Finkelstein’s test.

Tenderness over the extensor sheath is often accompanied by swelling and thickness (fig. 10-46).
The pathology is increased vascularity of the outer sheath combined with edema that thickens the sheath and constricts the enclosed tendon.

The synovial fluid tends to increase and thicken with formation of fine hair-like fibers that adhere the adjacent tissues.

**Treatment**
For de Quervain's disease, splinting of the thumb and oral anti-inflammatory medication can be tried but local injection of steroids is preferable because one injection often suffices.

In refractory patients, surgical release may be required.

**Tendinitis in the Flexor Compartments**

**Trigger Fingers**

The parallel collagen fibers that form a tendon undergo attritional changes from overuse, trauma, and occasionally disease. As a result of trauma, the ligamentous sheath thickens impairing repeated gliding.

Thickening results from microscopic tearing of the collagen fibers and thickening of the sheath. After being torn, a collagen fiber has the tendency to retract and form a nodule (Fig. 10-47).

Snapping of the flexor tendons during active finger flexion is termed "trigger finger" and is palpable and audible during flexion and re-extension.

Flexion is restricted but if flexion is further attempted it occurs suddenly.

Once flexed the nodule within the finger has passed annular ligament remains locked in the flexed position, and cannot be reextended.

This trigger action occurs most frequently in the middle or ring finger and is related to direct, repetitive trauma to the flexor tendons of the fingers (Fig. 10-48).

Treatment consists of injecting a steroid into the sheath to lessen its constriction.

Although the nodule remains, it now may be able to pass the previous obstruction. Failure of this procedure necessitates surgical intervention in which a transverse incision of the sheath proximal to the nodule exposes the annular band, which has been slit. Excision of the nodule invariably causes formation of a new and often larger nodule.

**Flexor Carpi Ulnaris Tendinitis**

Tendinitis of this tendon (FCU) is common and results from repetitive trauma. It is especially common in tennis players and carpenters.

Clinically pain and swelling exist, proximal to the pisiform and exacerbated by wrist flexion in a radial direction. Because of its proximity, concurrent compression of the ulnar nerve within Guyon's canal may be present. Differentiation of pain located in this region from that of a pisiform fracture or arthritis of that bone is made by reproducing the pain with a side-to-side motion of the bone.
Treatment requires splinting in a mild wrist flexion and oral steroid or NSAID medication, or direct injection of steroids into the area.

In resistant patients, the ligament can be lengthened or the pisiform bone excised.

**FRACUTURES AND DISLOCATIONS**
The metacarpals inform three arches: the proximal, distal transverse, and the longitudinal.

The proximal carpal arch is formed by the carpal bones.

The distal transverse arch is composed of the distal portions of the metatarsal heads (fig. 10-49).

**Principles of Treatment**
The following principles in the care of injured phalanges and interphalangeal joints must not be violated:

1. Immobilization must be instituted to relieve pain and permit primary healing. Too early or inappropriate active or passive motion exercise may result in more pain and contracture. Usually immobilization must be maintained for at least 10 to 14 days.
2. Immobilization must be maintained in degrees of physiologic flexion (fig. 10-50). No fracture must be immobilized by using extension of all three joints.
3. All digits that do not require immobilization must be actively, not passively, mobilized.
4. The uninvolved digits of the hand, wrist, elbow, and shoulder must be actively placed through their normal range of motion with emphasis on frequent elevation of the hand above the level of the heart.

Persistent pain, loss of function, and work-related disability involving an upper extremity are affected by multiple factors.

These include physical capabilities as related to work demands, ergonomic risk factors in the workplace, psychological factors related to the worker in terms of work, and the ability to manage symptoms as required by the patient's desire to return to work.

Long-term vocational outcome involves multicomponent rehabilitative efforts:

1. Physical reconditioning
2. Modification of workplace ergonomics
3. Stress management
4. Vocational counseling and placement

With so many factors involved in upper extremity disability, a multidisciplinary program must be instituted to address all factors. Despite widespread growth of programs and services providing multidisciplinary rehabilitation, little or no outcomes assessment statistics have evolved. Because many, if not most, are soft tissue injuries, it is apparent that these injuries must be recognized, and then understood in a meaningful way, so that the components of disability can provide the basis of therapy and functional restoration.
Not all upper extremity disabilities need a multidisciplinary approach but when the condition is not recognized and does not respond to meaningful therapeutic approach before onset of chronic pain and persistent disability, the multidiscipline approach must be considered.

Simplistically, the patient’s return to work has become the standard for judging the benefit of therapy. Return to work before therapy is complete or before even pain is now significantly lessened mandated by employers and insurers. This edict implies reassuring the patient of performance of function "in spite of pain" is not only not harmful but may even be beneficial.

"Fear of reinjury" is, however, a prominent concern that only a meaningful, thorough, accurate examination and related treatment will be able to overcome.

Return to work must also address the workplace ergonomics in a meaningful and realistic manner. Prevention here, as in all other injuries, is as mandatory as recover from injury and its sequelae.